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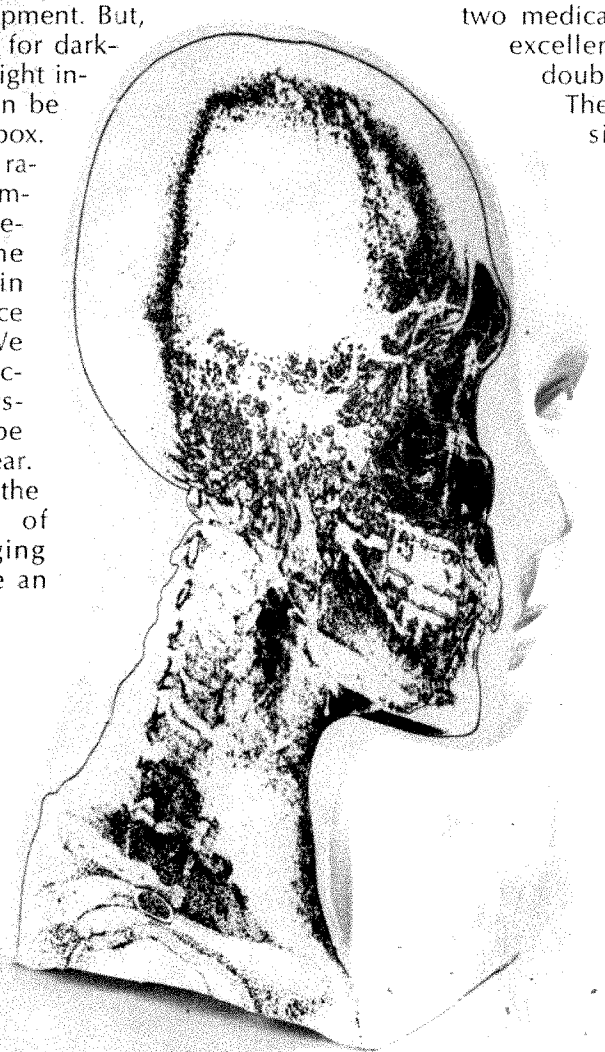
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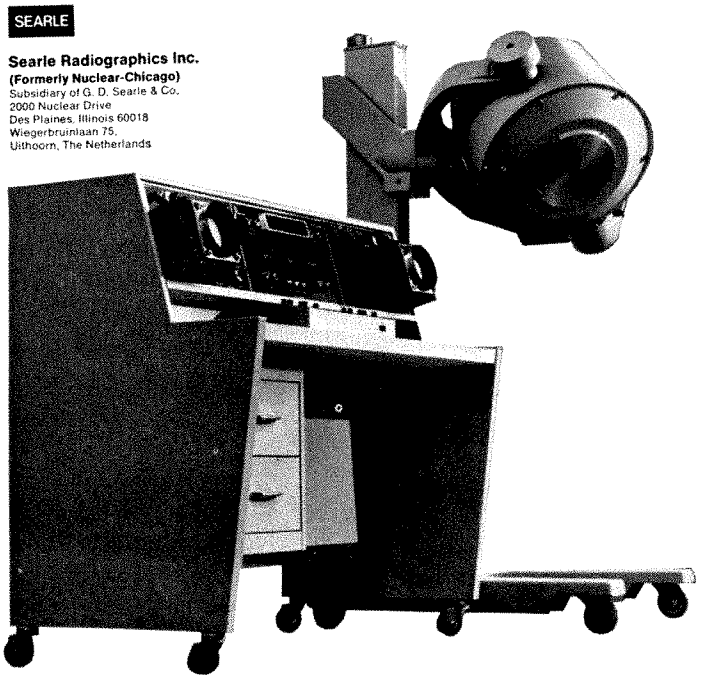
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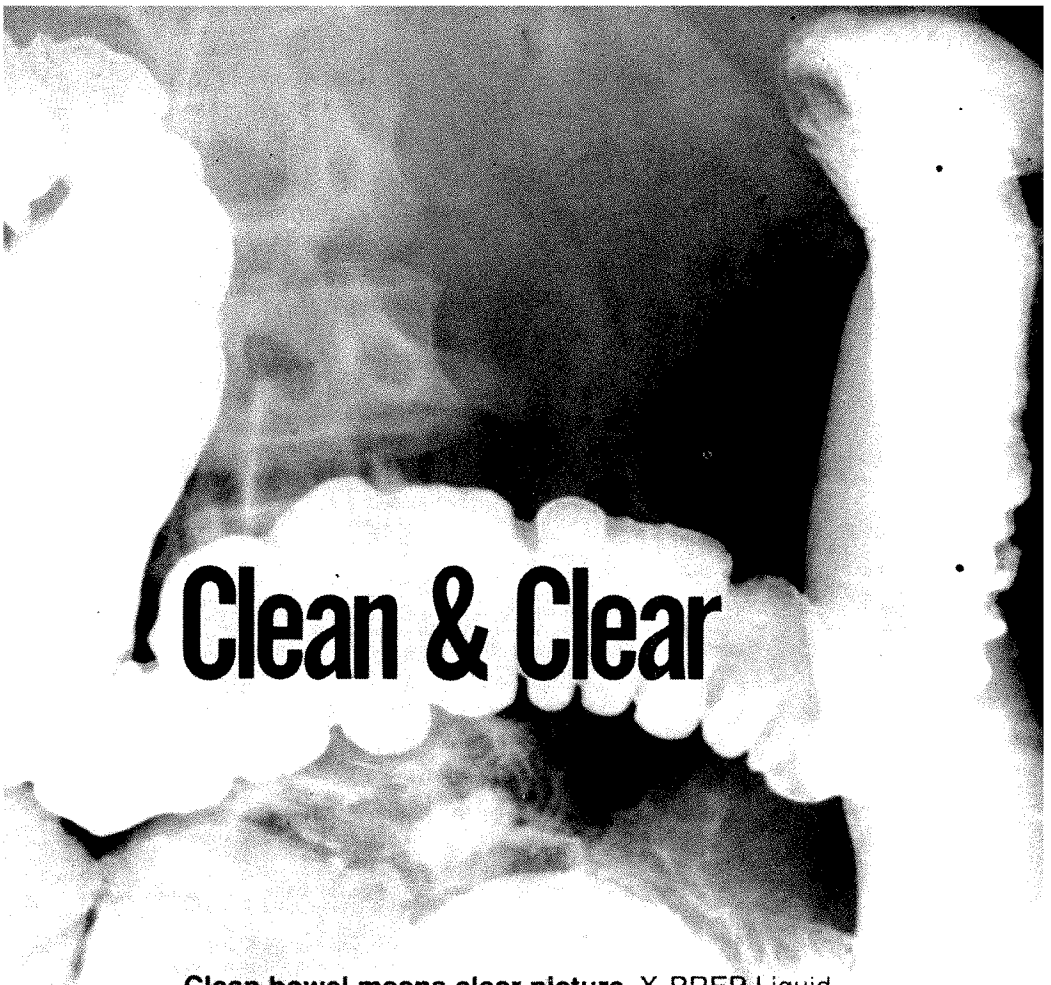
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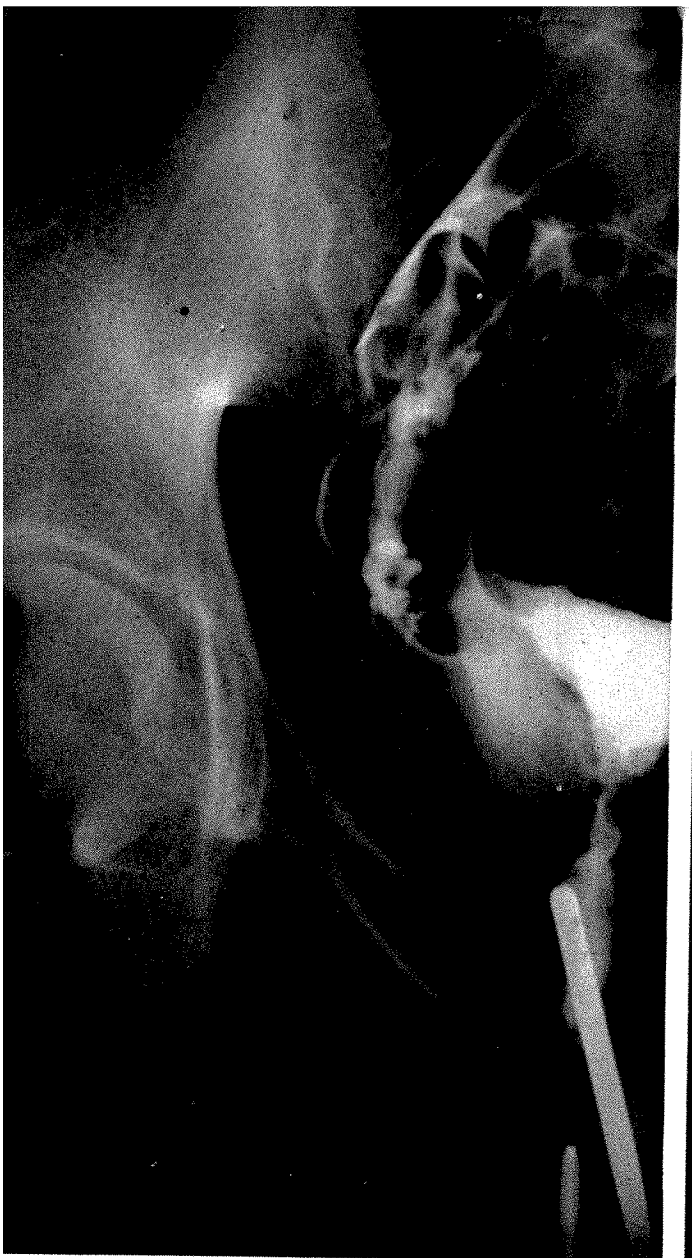
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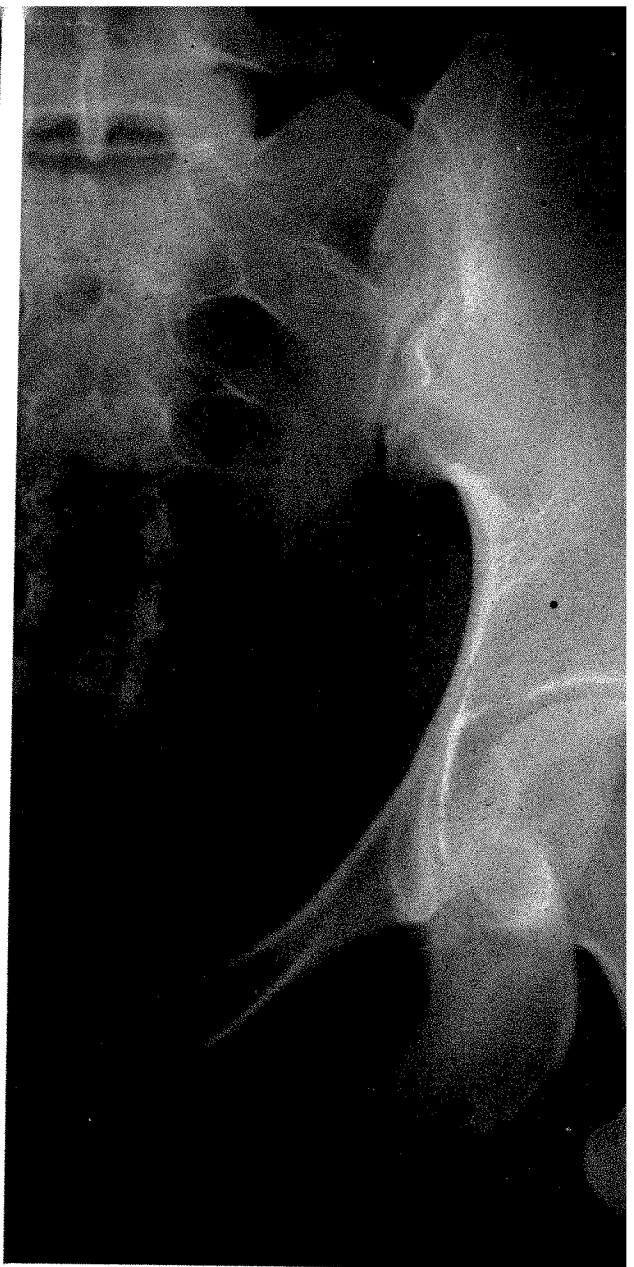
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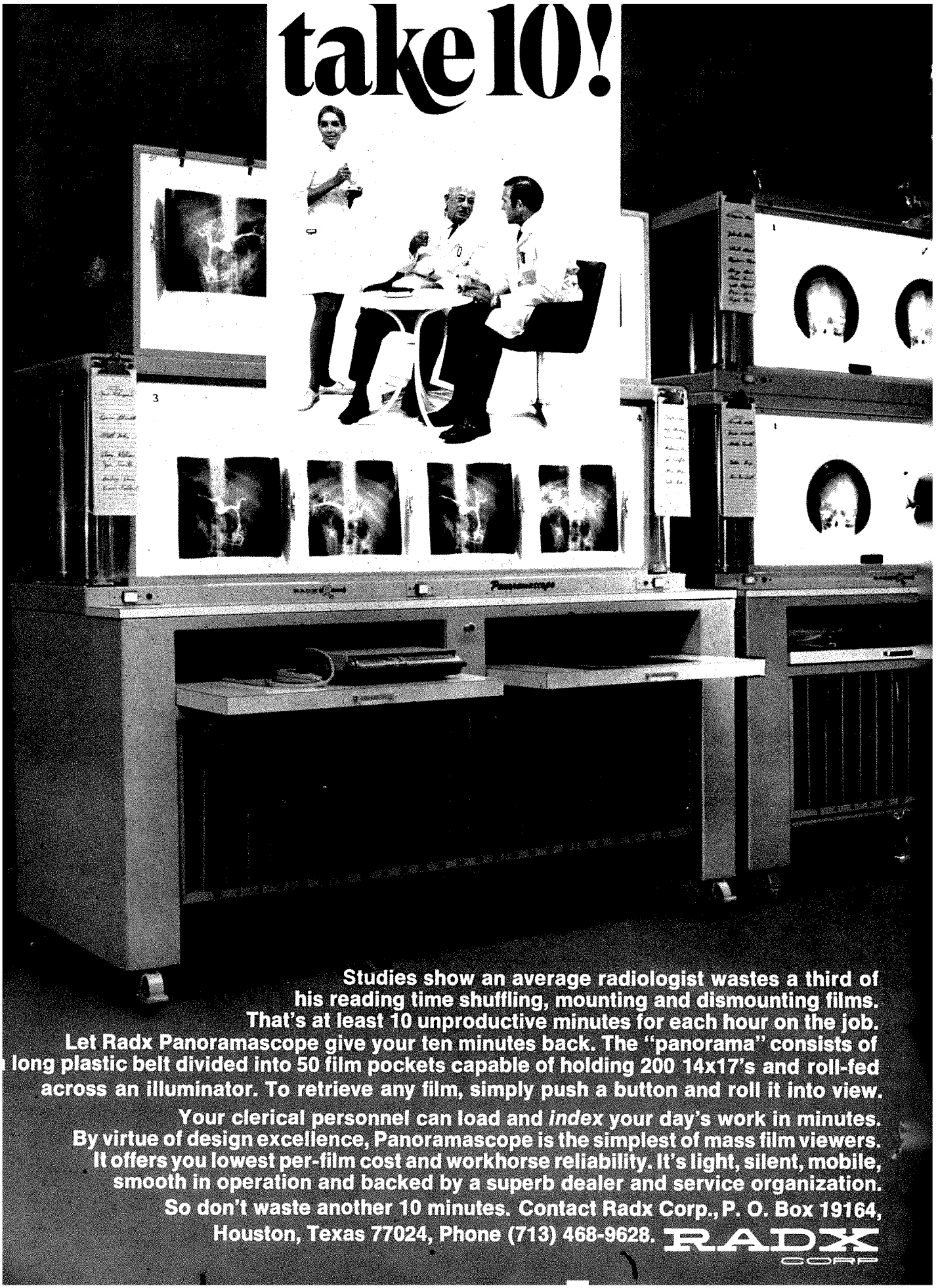
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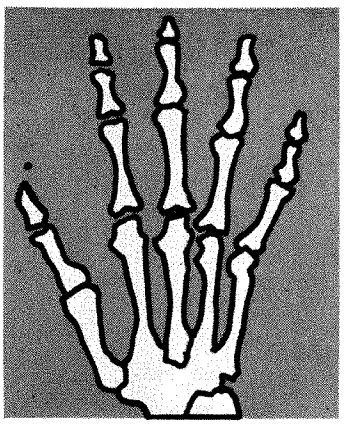
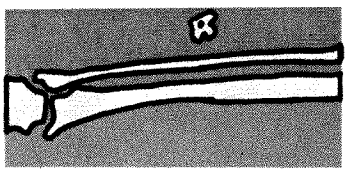
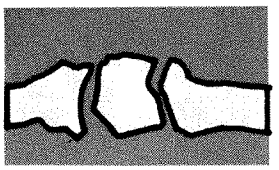
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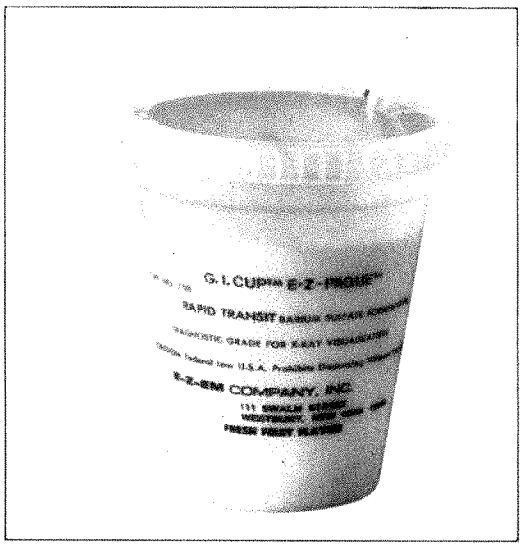
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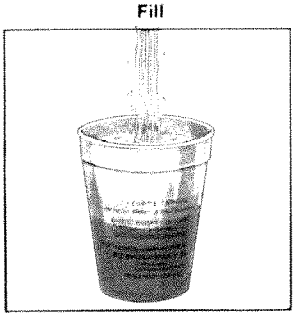
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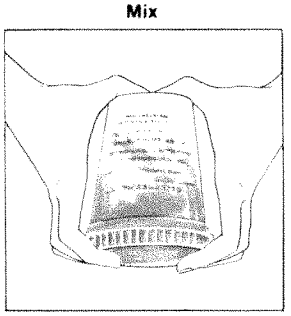
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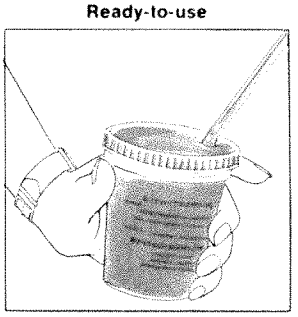
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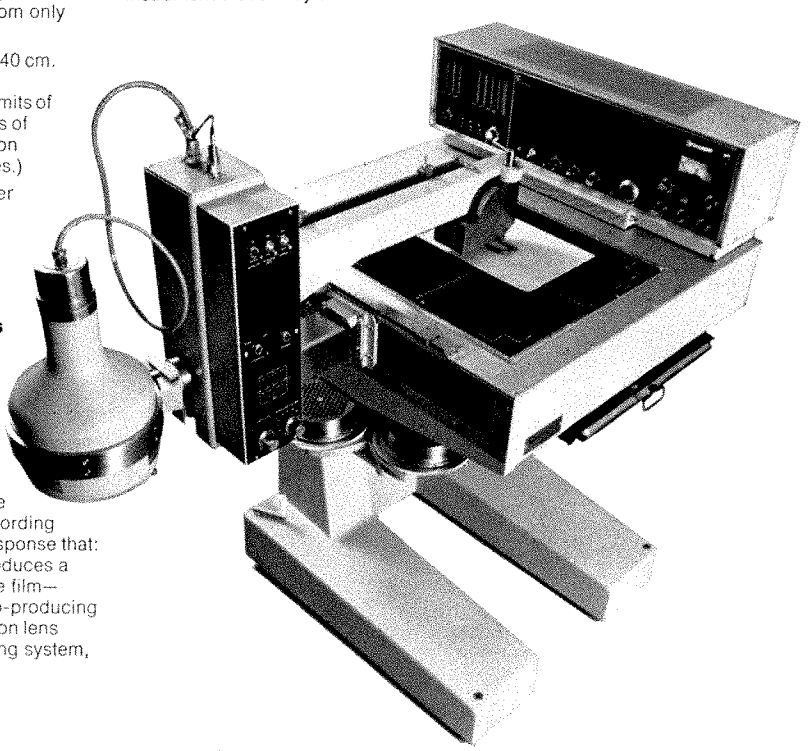
Quick-Change Collimators—Collimators are stored in a lazy susan tray below the scanning head—the 4-collimator capacity tray easily swings into position for collimator changing.

Digital Response—Both the photorecording and dot recording systems feature a digital response that: 1) with no suppression, produces a sharp-isotope image on the film—thanks to the digitized photo-producing light source and the precision lens system in the photorecording system, and that, 2) allows you to operate on a one-dot per one-count basis over a count-rate range of 0-4,000 counts per minute! Thanks to the exclusive Rapi/Dot™ tapper. (With this system you can obtain a tap scan that provides a sharp, continuous-tone reproduction of the isotope pattern!)

Enough to whet your interest? If you'd like to learn more about all the features of this truly unusual instrument that's 'way ahead of its time . . . more like 2002 A.D. than 1973 . . . contact your Searle Radiographics (formerly Nuclear-Chicago) sales engineer or write to us for our free Pho/Dot brochure.

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New Hypaque-DIU 30%

brand of **meglumine diatrizoate, USP**

- Extremely low sodium content (less than 0.02 mg./ml. in stabilizing agent)—of special advantage in patients with congestive heart failure.
- Highly diagnostic urographic contrast—reduces the need for retrograde pyelography.
- Good systemic tolerance.*

*Consult important product information on next page for patient selection, prescribing, and precautionary recommendations.



A logical addition to the expanding Hypaque® product line

New Hypaque-DIU® 30%

brand of

meglumine diatrizoate, USP

Sterile Aqueous Solution
For Infusion Urography

Description: Hypaque-DIU 30 per cent is a solution of meglumine 3, 5-diacetamido-2, 4, 6-triiodobenzoate ($C_{18}H_{26}I_3N_3O_9$). The 30 per cent solution (weight/volume) contains approximately 141 mg. of organically-bound iodine per ml. It has a viscosity of 1.94 cps at 25°C. and 1.42 cps at 37°C. The pH is adjusted with hydrochloric acid or meglumine solution. The sterile, aqueous solution is clear and colorless to pale yellow. Hypaque-DIU 30 per cent is relatively thermostable and may be autoclaved. It should be protected from strong light. The solution contains calcium disodium edetate 1:10,000 as a sequestering stabilizing agent.

Action: Hypaque-DIU 30 per cent is a diagnostic radiopaque medium which provides visualization of internal structures.

Indication: Hypaque-DIU 30 per cent is indicated for infusion urography.

Warnings: *Usage in Pregnancy.* No teratogenic effects attributable to a 60 per cent solution of Hypaque meglumine have been observed in reproduction studies in animals. However, before administration of Hypaque-DIU 30 per cent to women of child-bearing potential, the benefit to the patient should be carefully weighed against the possible risk to the fetus. In addition, most authorities consider elective contrast radiography of the abdomen contraindicated during pregnancy.

Serious or fatal reactions have been associated with the administration of radiopaque media. It is of utmost importance that a course of action be carefully planned in advance for the immediate treatment of serious reactions, and that adequate and appropriate facilities be readily available in case a severe reaction occurs.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravascularly.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake and direct thyroxine assays, are not affected.

Precautions: Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered.

Diphenhydramine hydrochloride (i.e., Benadryl®), however, should not be mixed with Hypaque-DIU 30 per cent since it may cause precipitation. The intravenous injection of a test dose of 0.5 to 1 ml. of the contrast agent before infusion of the full dose has been employed in an attempt to predict severe or fatal adverse reactions. The preponderance of recent scientific literature, however, now demonstrates that this provocative test procedure is not reliably predictive of serious or fatal reactions.

Severe reactions and fatalities have occurred with the test dose alone, with the full dose after a nonreactive test dose, and with or without a history of allergy. No conclusive relationship between severe or fatal reactions and antigen-antibody reactions or other manifestations of allergy has been established. A history of allergy may be useful in predicting reactions of a mild or intermediate nature.

Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations, or of sensitivity to iodine.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure, it is essential to omit preparatory dehydration, to administer fluids, and attempt to alkalinize the urine.

Immediate adverse reactions to infusion urography have not been reported to occur at a higher frequency or with greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Infusion urography imposes a sudden osmotic load. This should be considered in patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, there is a possibility of the development of congestive heart failure hours after the procedure.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating infusion urography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion urography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

Adverse Reactions: Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

The following have been reported after administration of diatrizoates and other iodinated contrast media. *Cardiovascular reactions:* vein cramp and thrombophlebitis following intravenous injection, rare cases of cardiac arrhythmias, hypertension, hypotension and shock, and cardiac arrest. *Allergic reactions:* asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria, and rarely, anaphylactic reactions. Signs and symptoms related to the *respiratory system:* pulmonary or laryngeal edema, bronchospasm; or to the *nervous system:* restlessness, convulsions. *Other reactions:* flushing, pain, warmth, nausea, vomiting, anxiety, headache, dizziness, hematoma, and ecchymosis. Occasionally, transient proteinuria, and rarely, oliguria or anuria. Rarely, immediate or delayed rigors can occur, sometimes accompanied by hyperpyrexia. Infrequently, "iodism" (salivary gland swelling) from organic iodinated compounds appears two days after exposure and subsides by the sixth day.

Dosage and Administration: The recommended dose for infusion urography is calculated on the basis of 2 ml. of Hypaque-DIU 30 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimal results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

The solution is infused over a period of three to ten minutes. Radiographs are usually taken at 10, 20, and 30 minutes from the beginning of the infusion. Earlier films may be obtained for the evaluation of hypertension. Nephrograms are usually obtained at the end of the infusion, and voiding cystourethrograms are usually obtained 30 minutes after the beginning of the infusion.

How Supplied: Calibrated bottles of 300 ml., rubber stoppered, packed with and without sterile disposable intravenous infusion sets.

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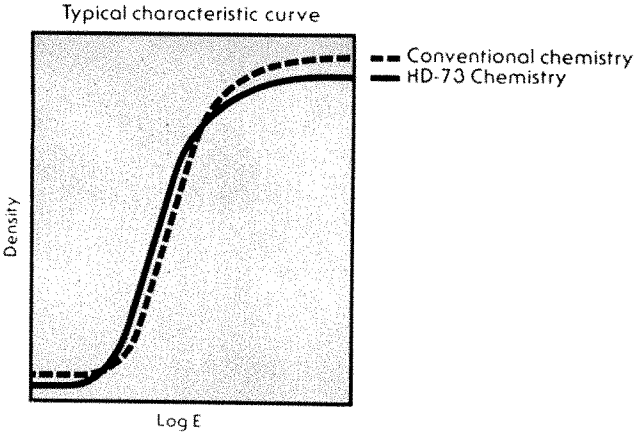
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Typical characteristic curve



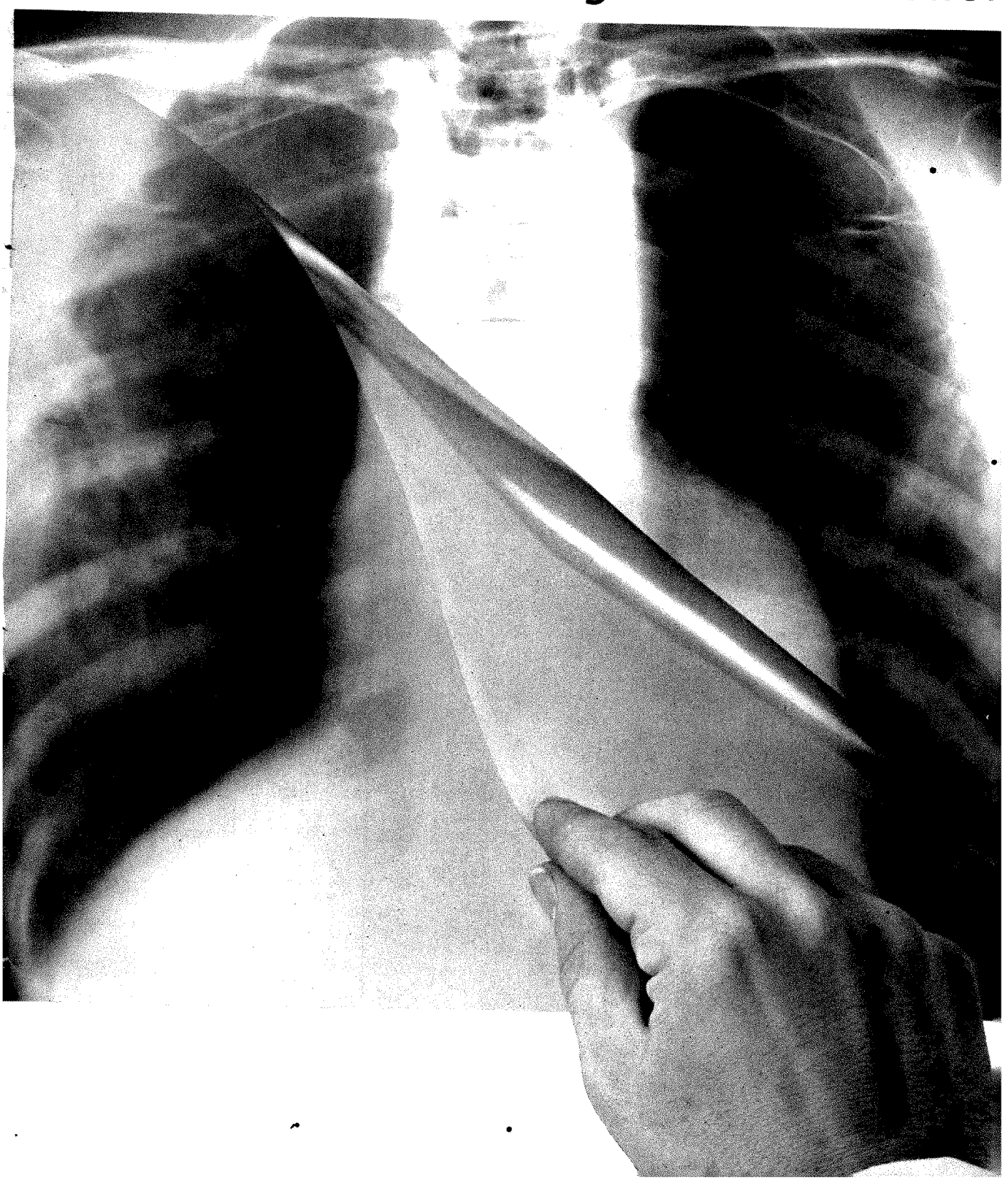
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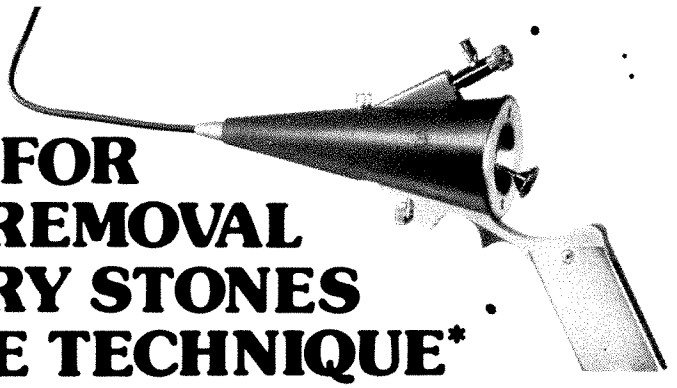
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THE SOFT-STEERABLE CATHETER SYSTEM FOR NON-OPERATIVE REMOVAL OF RETAINED BILIARY STONES VIA THE BURHENNE TECHNIQUE*



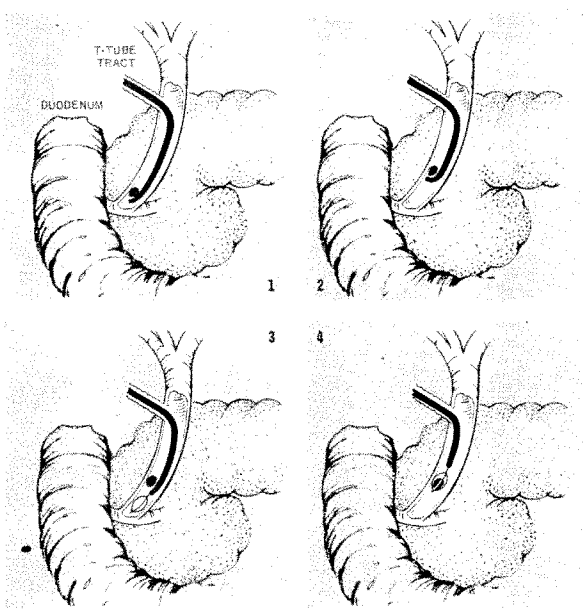
NCEPT

ng the Burhenne technique and the MEDI-TECH steerable catheter, a nificant number of non-operative removals of retained biliary stones have been ieved with consistent success.*

The safe, simple procedure, developed by Dr. H. Joachim Burhenne, is formed as a follow-up to cholecystectomy, after it has been determined, by a stoperative cholangiogram, that there are retained stones. The procedure is formed, at least four weeks after surgery, by removing the T-tube and advancing MEDI-TECH Soft Steerable Catheter through the remaining T-tube tract.

Unique maneuverability of the soft-steerable catheter tip permits the physician dislodge, manipulate and reposition the stone(s). Contrast media is injected ough the Luer-Lock and the Open Lumen of the MEDI-TECH catheter to hance visualization. Then, with the stone in desired position, a wire basket or are is passed through the Tuohy-Borst fitting and the open lumen to the site. ien the stone is secured, the entire assembly is withdrawn. Although the soft-tip ovides sufficient torque for this procedure, patient discomfort is minimal and anesthesia or pre-medication is used.





*REFERENCE

Burhenne, H. J., Non-operative Retained Biliary Tract Stone Extraction — A New Roentgenologic Technique, AMER. J. ROENTGENOLOGY, Vol. CXVII, No. 2, pp. 388-399, February 1973.

AN X-RAY CINE FILM of the Burhenne procedure is available. Please contact MEDI-TECH to arrange a viewing of the film and to obtain copies of reference articles plus complete information on the MEDI-TECH/BURHENNE Biliary Catheter System.

THE COMPLETE BILIARY CATHETER SYSTEM includes the MEDI-TECH Remote Control Handle, Special Biliary Catheters and Wire Stone Removal Baskets.

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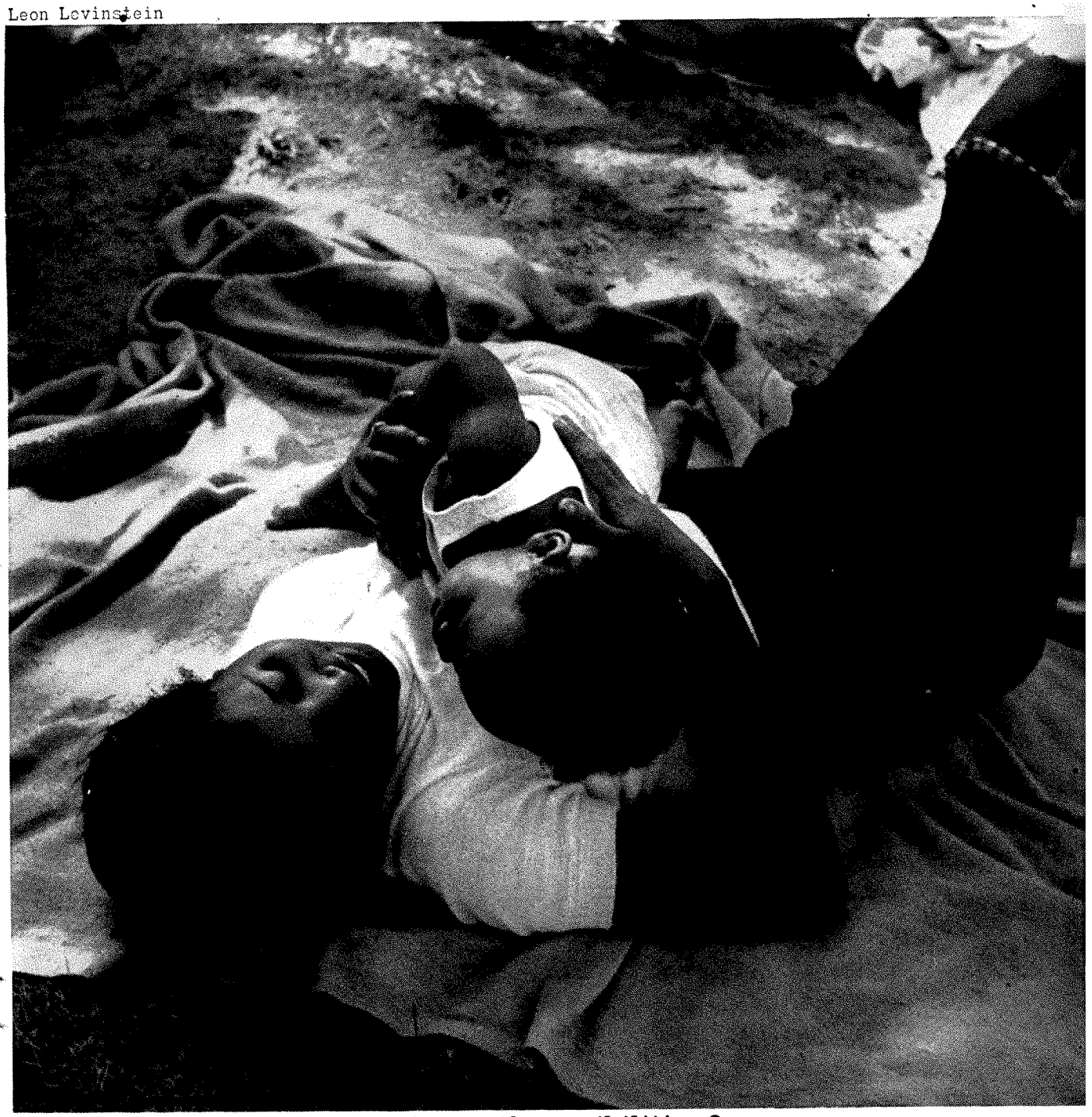
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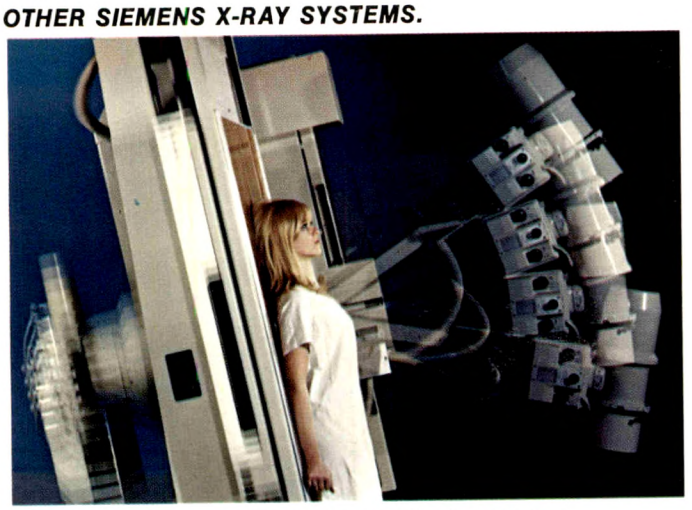


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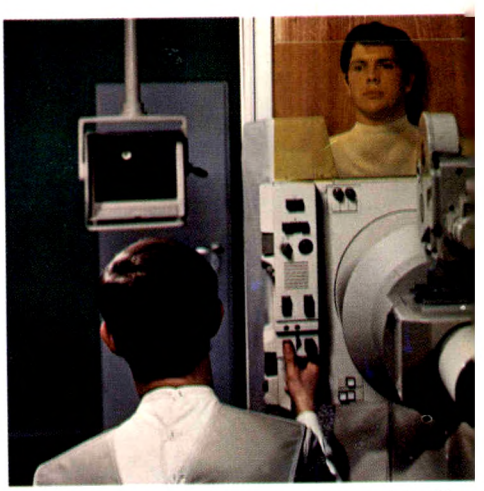


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BRIEF SUMMARY

DESCRIPTION: Ethiodol® (Ethiodized Oil) contains 37% iodine in organic combination with poppyseed oil.

INDICATIONS: For radiographic exploration in hysterosalpingography.

CONTRAINDICATIONS: Individual hypersensitivity; where hysterosalpingography is contraindicated; myelography.

WARNINGS: Not for use in bronchography. Observe extreme caution where sensitivity to iodine or other contrast materials is present. Iodine may alter results of thyroid function tests. Safety in pregnancy not established.

PRECAUTIONS: Usual precautions for hysterosalpingography, particularly intravasation of the contrast material in the presence of uterine bleeding, recent curettage or excessive injection pressure.

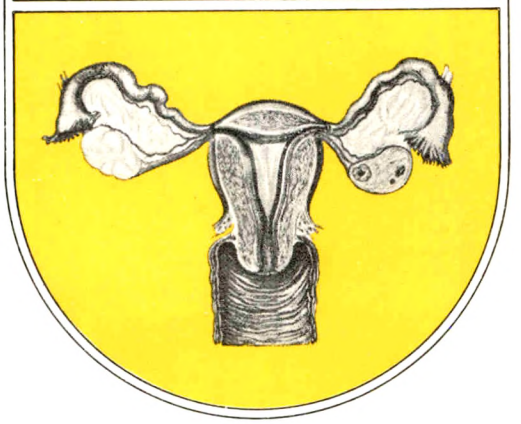
ADVERSE REACTIONS: Hypersensitivity reactions, foreign body reactions and exacerbation of pelvic inflammatory disease. Transient abdominal pain may occur.

DOSAGE and ADMINISTRATION: Introduce under pressure and preferably with fluoroscopic control. A preliminary film is exposed and a skiagram is made after the injection of 5 ml. Ethiodol®. The pressure is raised to 80-90 mm. Hg and in cases of normal bilateral tubal patency, the pressure falls immediately to below 60 mm. Hg. The wet film may be viewed immediately. Use a 24-hour follow-up to check for "spill" into the peritoneal cavity. Before using, see package insert for full details.

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The emergence of medical oncology as an area of specialization has been recognized by the American Board of Internal Medicine, which has created a sub-specialty examination for certification. This book is ideally suited not only for preparation for this examination, but as a definitive reference for every clinician who is in any way involved with the diagnosis, treatment or research of this disease.

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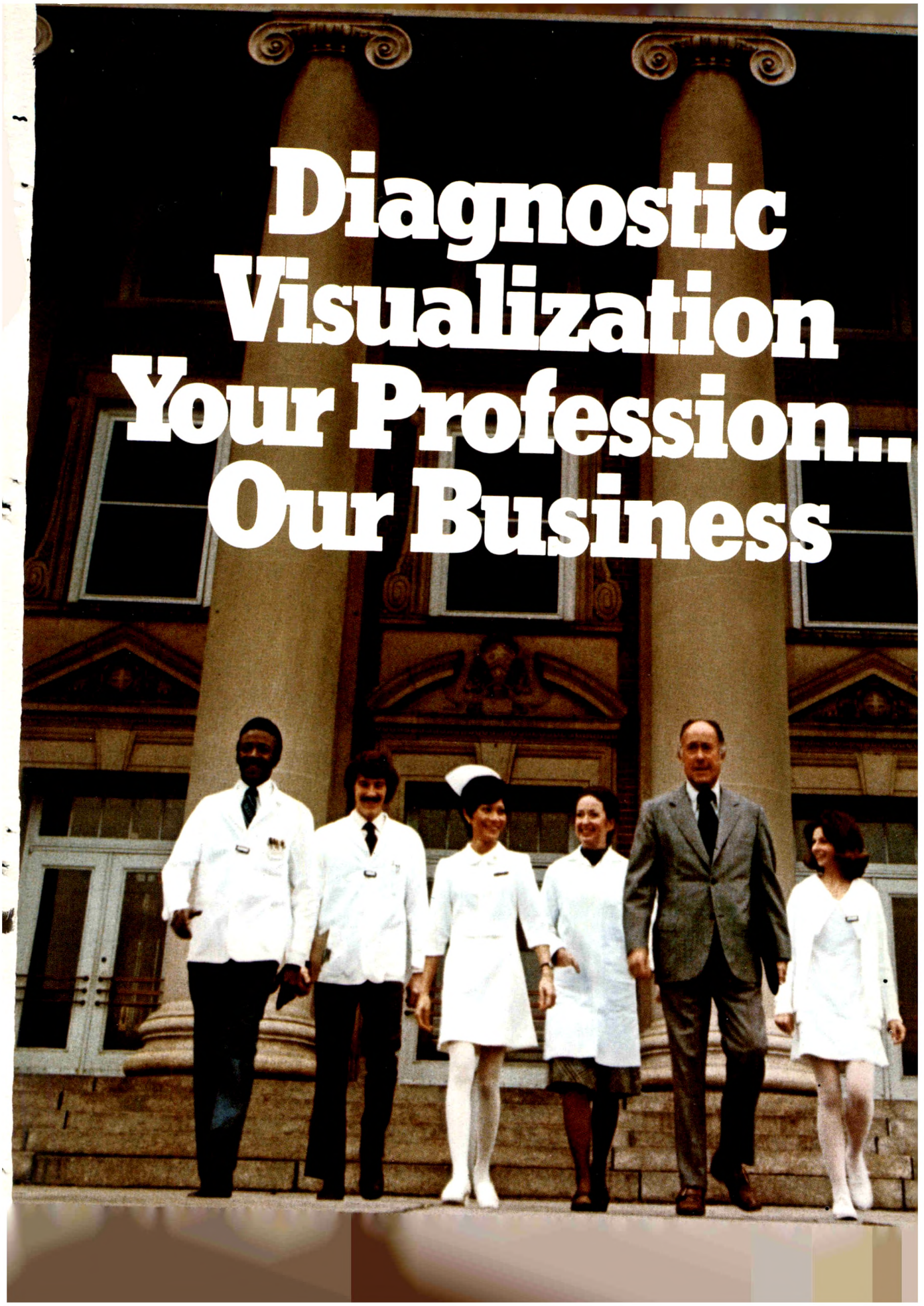
Etiology of Cancer; Pathogenesis of Cancer; Comparative Neoplasia; Epidemiology of Cancer; Cancer Detection and Prevention; Diagnostic Radiology of Cancer; Principles of Management; Experimental Design of Clinical Trials; Principles of Cancer Surgery; Principles of Radiation Therapy; Principles of Nuclear Medicine; Principles of Chemotherapy; The Chemotherapeutic Agents; Principles of Endocrine Therapy; Tumor Immunology; Host Effects of Cancer; Hematologic Complications of Cancer; Infections in Patients with Cancer; Hematologic Neoplasms; Brain and Peripheral Nervous System Tumors; Tumors of the Eye; Cancer of the Head and Neck; Lung Cancer; Alimentary Tract Cancer; Genitourinary Tract Cancer; Gynecologic Cancer; Breast Cancer; Neoplasms of the Skin; Melanoma; Soft Tissue Sarcomas; Bone and Cartilaginous Tumors; Childhood Solid Tumors; Principles of Rehabilitation; Selected New Developments.

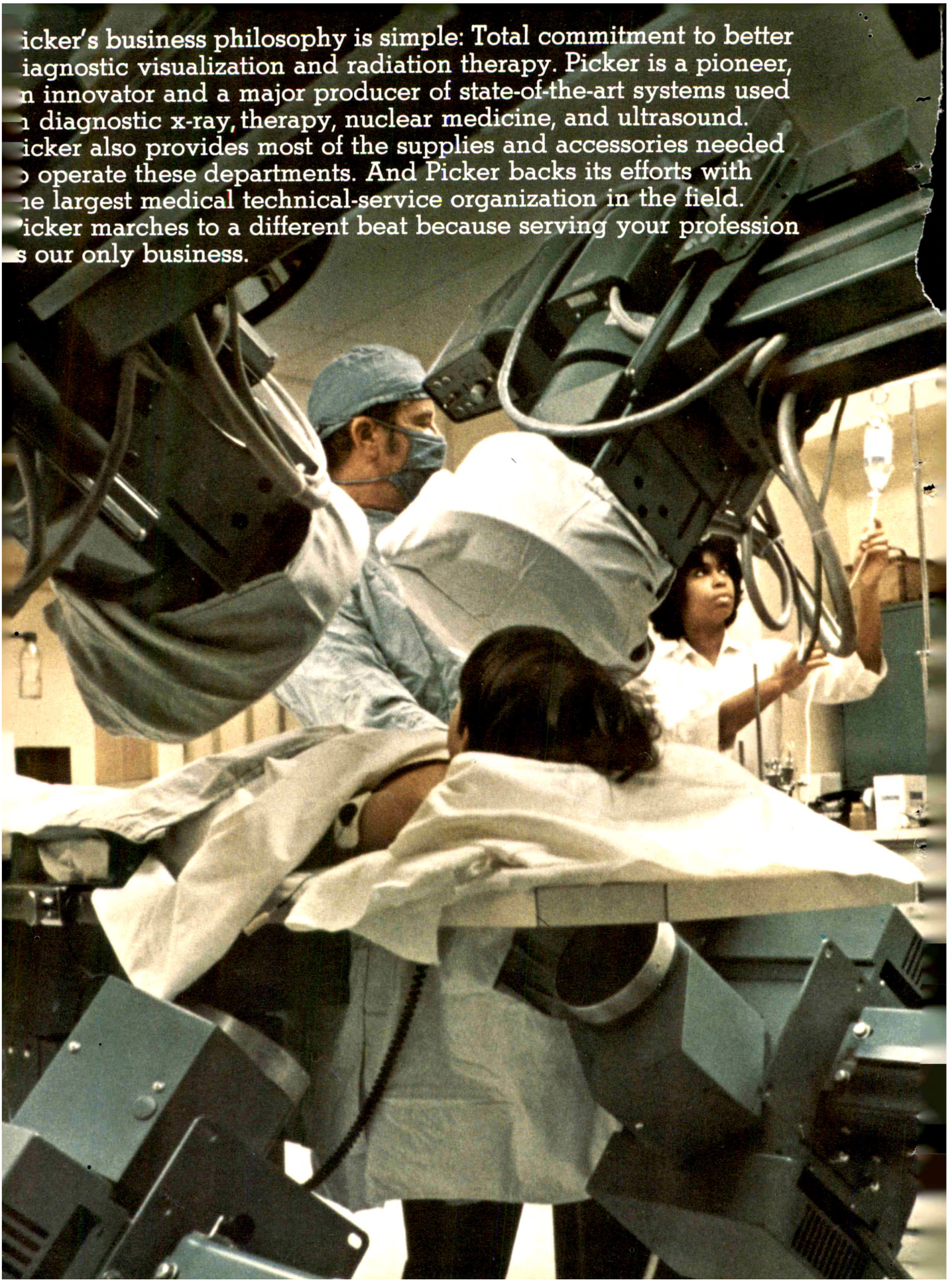
Edited and with contributions by JAMES F. HOLLAND, M.D., Professor and Chairman, Department of Neoplastic Diseases, Mt. Sinai School of Medicine of the City University of New York, formerly of the Roswell Park Memorial Institute, Buffalo; and EMIL FREI, III, M.D., Professor of Medicine, Harvard Medical School, Physician-in-Chief, Children's Cancer Research Foundation, Inc., Boston, formerly of the M. D. Anderson Hospital and Tumor Institute, Houston. 158 CONTRIBUTORS. About 2000 pages, 7" x 10". Illustrated. *New. Ready Soon.* (ISBN: 0-8121-0372-6).

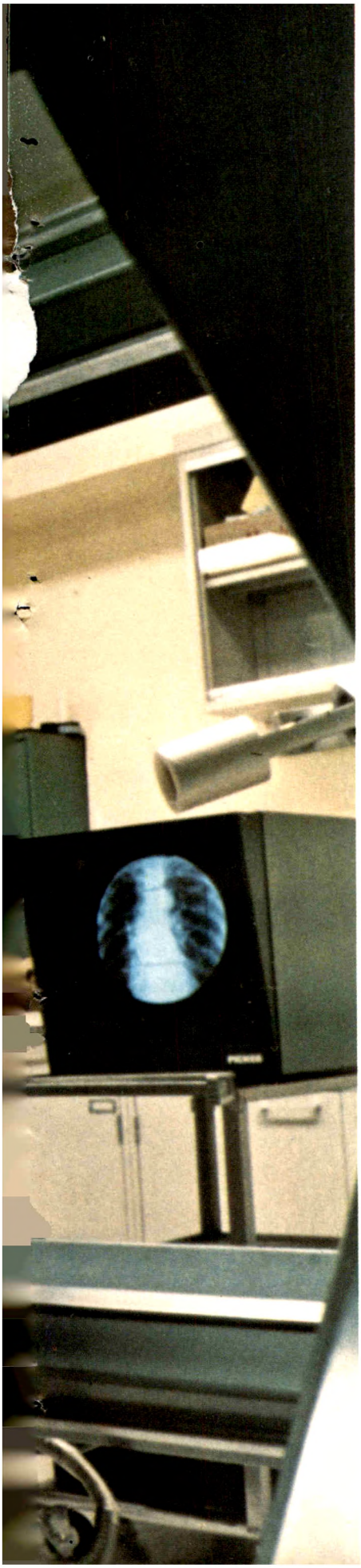
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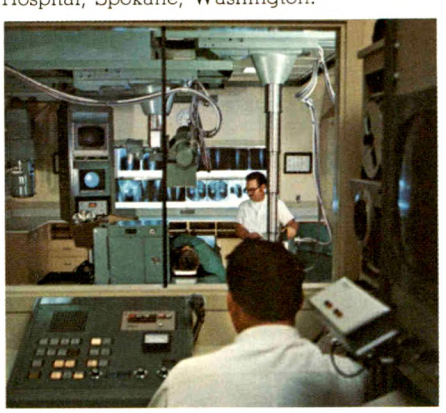

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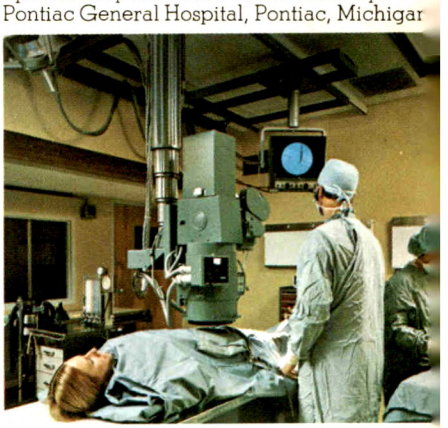
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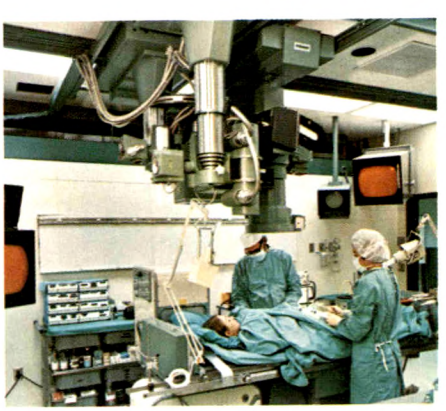
Vast experience in virtually all phases of the x-ray discipline has given Picker a synergistic know-how that enables us to serve the most demanding requirements in diagnostic special procedures. Picker's sophisticated designs result in greatest flexibility in adapting to caseload profiles . . . maximum efficien for patient throughput . . . boost utilization of both space and facilities.

Special-Purpose Room. Photo courtesy Radiology Department, Holy Family Hospital, Spokane, Washington.

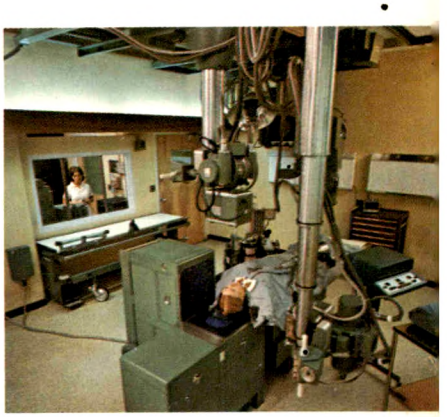


Special-Purpose Room. Photo courtesy Pontiac General Hospital, Pontiac, Michigan





Cardiology Laboratory. Photo courtesy Radiology Department, University Hospital of Jacksonville, Jacksonville, Florida.



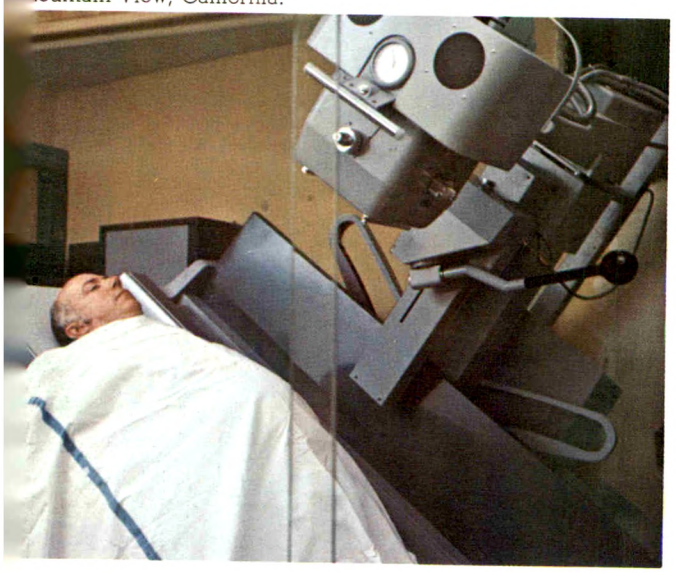
Special-Purpose Room. Photo courtesy Pontiac General Hospital, Pontiac, Michigan.

Photo courtesy Rainbow Babies and Childrens Hospital of University Hospitals, Cleveland, Ohio.

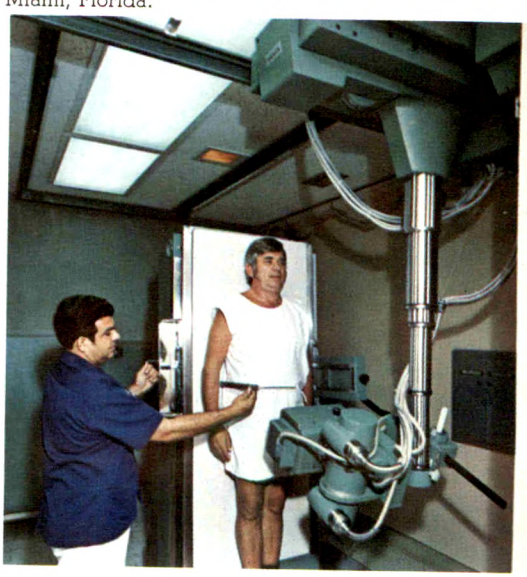
Radiographic & Fluoroscopic Rooms

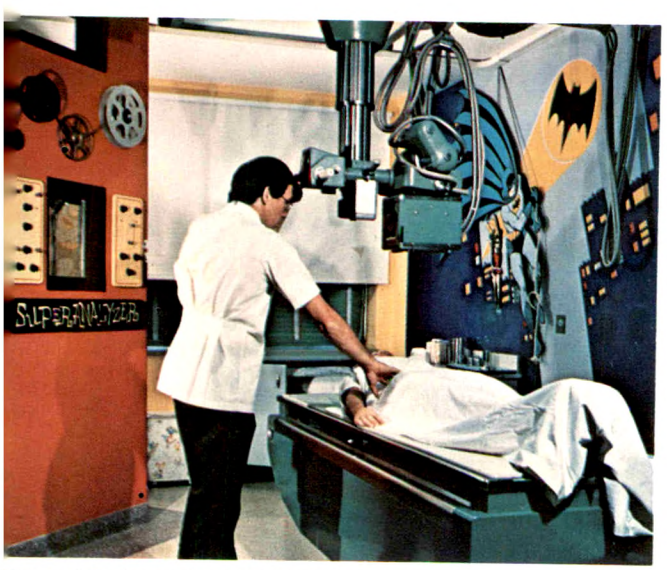
.cker carries the philosophy of the fully integrated diagnostic system most to its ultimate degree in Radiographic & Fluoroscopic Rooms. TV fluoroscopy, the entire process—including patient positioning, rogramming, phototiming, etc.—is precisely controlled and monitored. his enables the radiologist to obtain desired diagnostic data on film e first time so as to minimize unnecessary retakes.

emotely-Controlled R&F Room contains Picker Epic emotely operated diagnostic x-ray system. Photo ourtesy Radiology Dept., El Camino Hospital, lountain View, California.

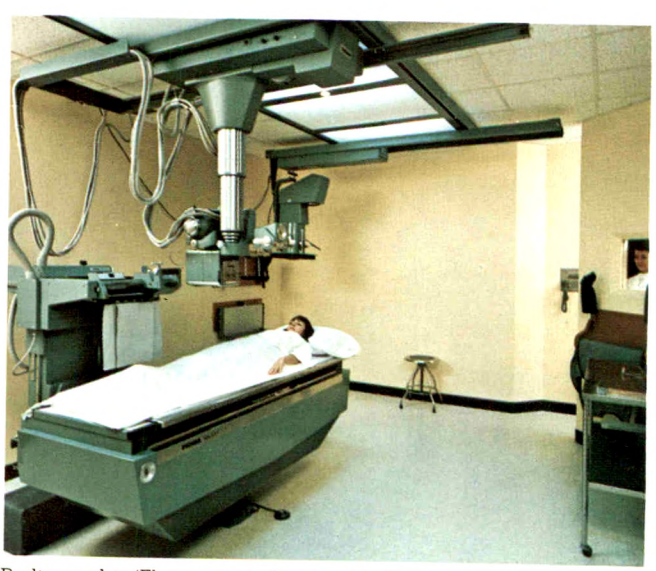


General Radiographic Room contains Galaxy table, Horizontal/Vertical Tomographic attachment, 800 mA generator, ceiling tube mount, Collimatic collimator. Photo courtesy Jackson Memorial Hospital, Miami, Florida.





General Radiographic (Bat Man) Room contains ilting radiographic table with manually adjustable collimator, illuminated ceiling tube mount, 300 mA generator. Photo courtesy Cardinal Glennon Memorial Hospital for Children, St. Louis, Missouri

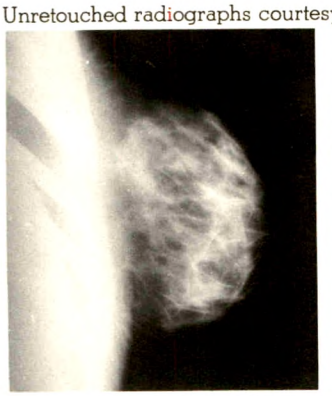


Radiographic/Fluoroscopic Room with Galaxy x-ray table, spot film device, image intensifier, ceiling tube mount, 500 mA generator. Photo courtesy Port Huron Hospital, General Hospital, Port Huron, Michigan.

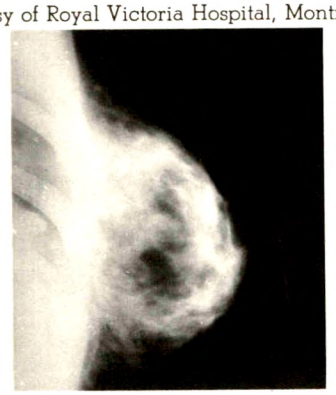
Mammography

The Picker Mammorex® is a completely coordinated breast radiographic system – the first specialized mammography unit that consistently shows the entire breast, from nipple back to the rib cage. Because Mammorex shows the complete breast in every desired position, it significantly increases the possibility of seeing even the smallest microcalcifications. Superb image quality of Mammorex is a function of the latest state-of-the-art x-ray tube, especially designed for mammography, combined with the unique Mammorex breast compressor. Since Mammorex is an isocentric system that completely moves around the patient, examination time is significantly decreased.

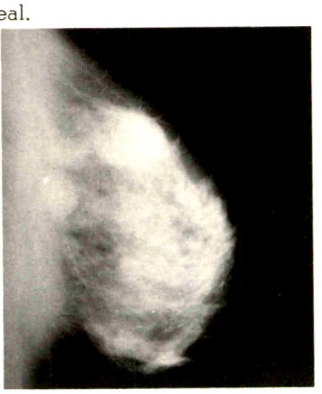
Unretouched radiographs courtesy of Royal Victoria Hospital, Montreal.



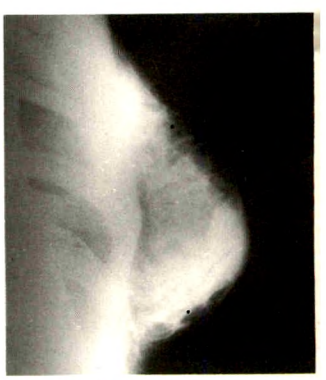
Mammorex is the first such specialized system that shows the entire breast all the way back to the rib cage.



Only when the chest wall is visible can one be sure that all of the breast is on the radiograph.



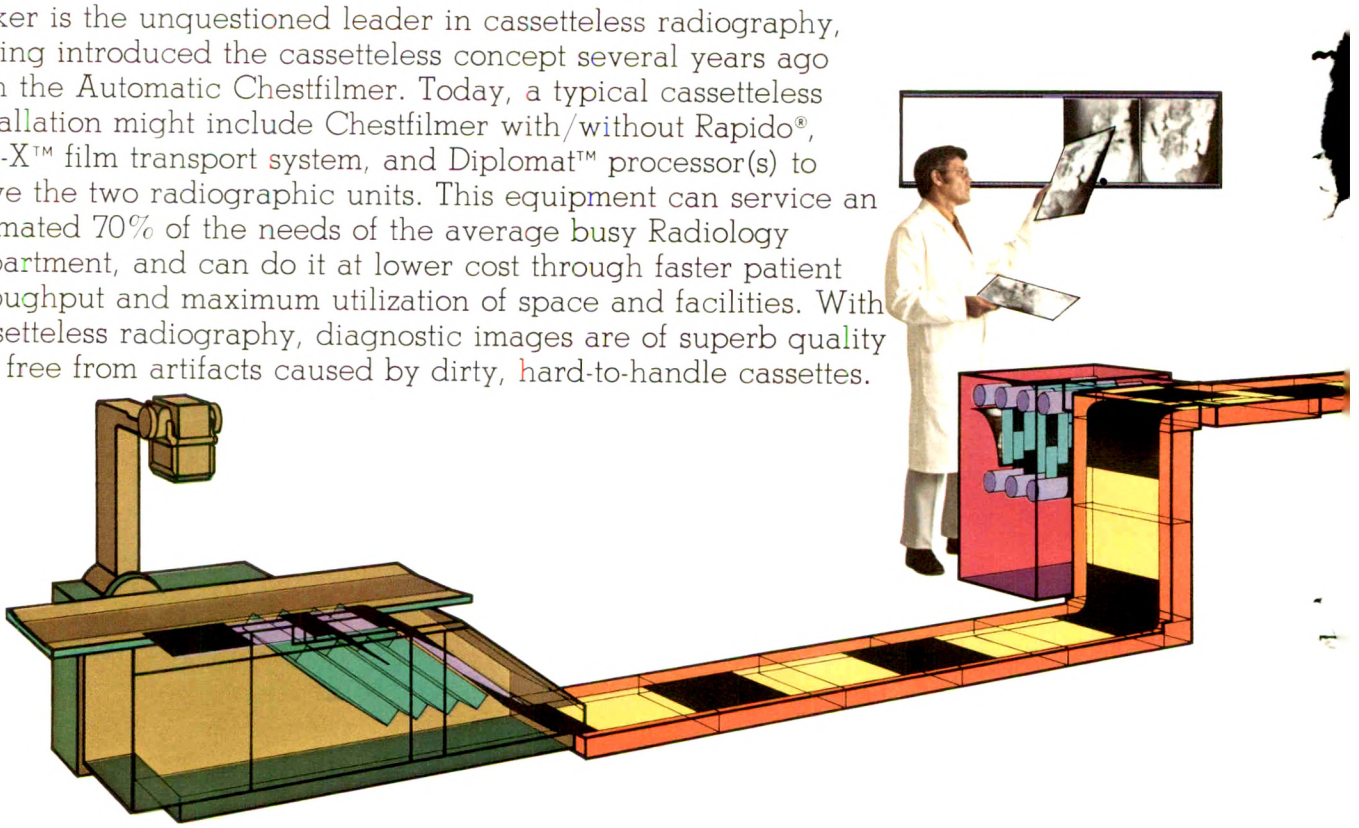
Malignancy shows no geographic preference; lesions proximal to the chest wall are visible with Mammorex



Mammorex yields high contrast even in the case of the dense young breast.

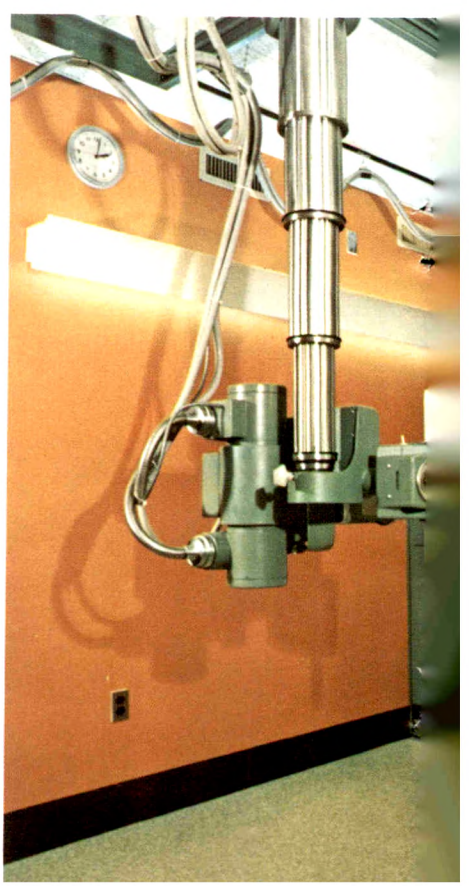
Cassetteless Radiography

Picker is the unquestioned leader in cassetteless radiography, having introduced the cassetteless concept several years ago with the Automatic Chestfilmer. Today, a typical cassetteless installation might include Chestfilmer with/without Rapido®, Rap-X™ film transport system, and Diplomat™ processor(s) to serve the two radiographic units. This equipment can service an estimated 70% of the needs of the average busy Radiology Department, and can do it at lower cost through faster patient throughput and maximum utilization of space and facilities. With cassetteless radiography, diagnostic images are of superb quality . . . free from artifacts caused by dirty, hard-to-handle cassettes.

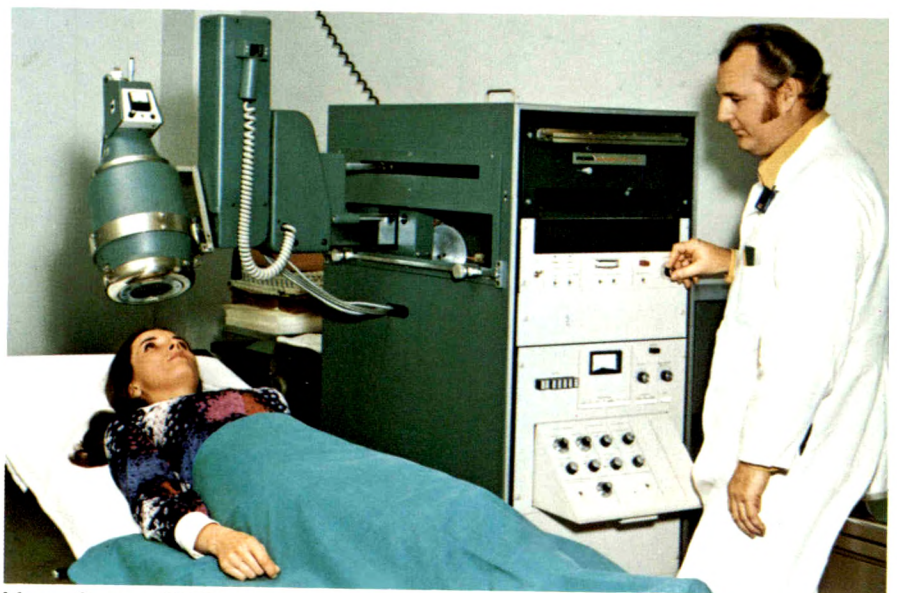


Complete Cassetteless Radiography room contains automatic Chestfilmer and Rapido, both integrated with and feeding into automatic processor via Rap-X film transport system. A single generator with phototimer provides power to both rooms. Photos courtesy St. Mary's Hospital, Lewiston, Maine.

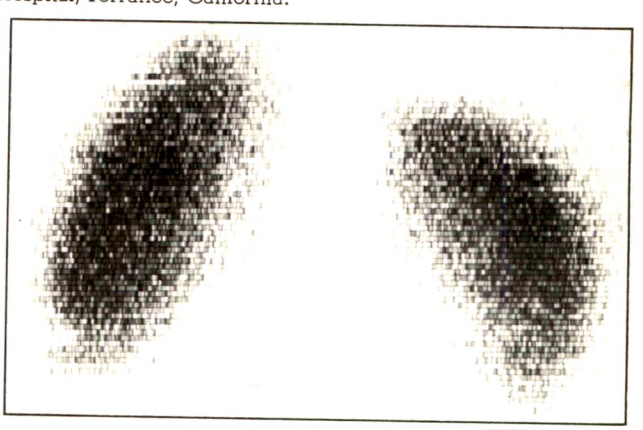


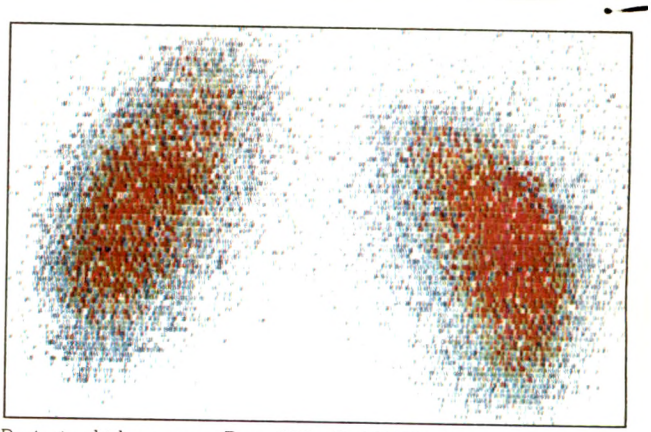


The Picker Magna® Scanners in use today throughout the world probably outnumber all other gamma-imaging devices combined. The new Magna Scanner, and dual Magna Scanner provide virtually automated scan setups, including highest quality color scans. Productivity is high because the instrument gives consistent scans with minimal retakes.

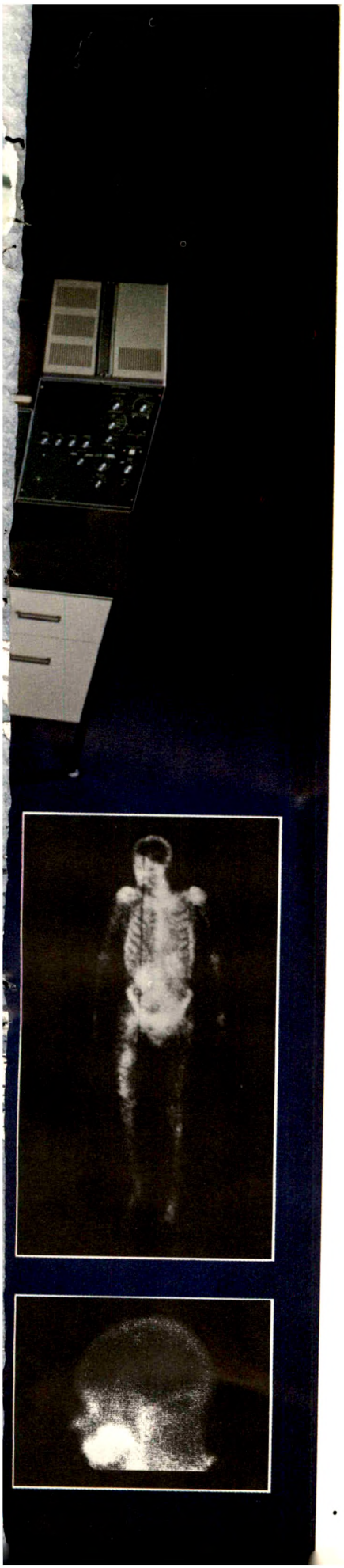


Magna Scanner 500D using 10-degree caudal technic. Photo courtesy Los Angeles County Harbor General Hospital, Torrance, California.





Posterior kidney scan: Dose 300 μCi 197 Hg; line spacing 2 mm; scanning speed 250 cm/min. Impression — Possible cysts in both kidneys.



Technetium 99^{m} Polyphosphate Whole-Body Image

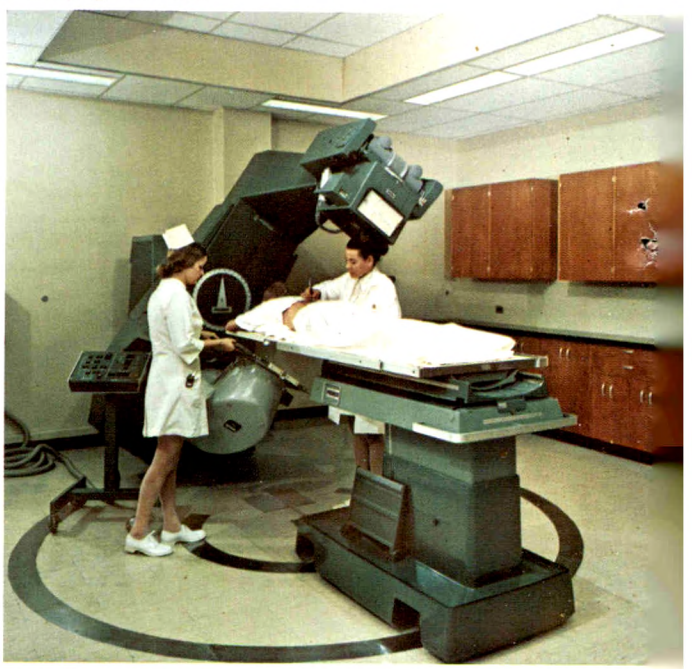
Left Lateral Brain Image from Dyna Camera 2C (50% reduction)

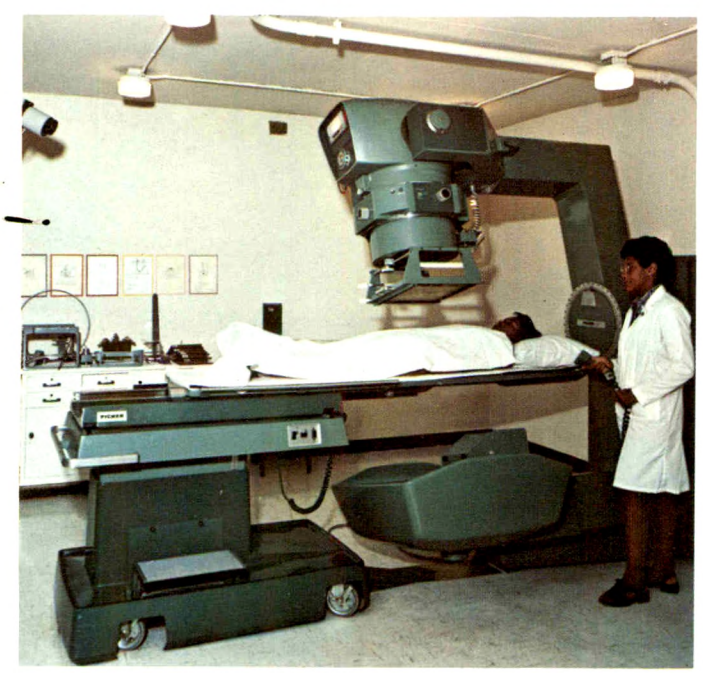
Radiation Therapy

Using diagnostic x-rays, Picker's TherX™ simulator pinpoints tumors, confirms entry and exit ports, and verifies beam coverage at every phase of the treatment to maintain the vital radiation beam/patient relationship. TherX is also invaluable as a teaching tool, or in aiding the radiotherapist in developing radiation treatment parameters.

Picker's credentials in radiation therapy extend from Cobalt-60 capsule manufacture to designing and producing the new C9 and V9 therapy units. These are a fourth-generation technological advance in cobalt radiation-therapy equipment. The C9 unit, featuring wide-ranging capability in moving-beam techniques, also has unprecedented isocentric accuracy, and enormous flexibility for setting up accurate, multiple-fixed-beam therapy. The Picker V9 vertical unit offers a full complement of technical advantages identical to the C9 rotational unit. Safety is paramount in all Picker designs and both units incorporate triple-safeguard systems for source security.

Therapy Simulator Room contains TherX, 300 mA generator, Decalix TV imaging system with isocentric diagnostic stretcher. Photo courtesy Manitoba Cancer Treatment and Research Foundation, Winnipeg, Manitoba.





Therapy Room with Picker C9 Cobalt-60 unit. Photo courtesy Radiology Department, George W. Hubbard Hospital of Meharry Medical College, Nashville, Tennessee.

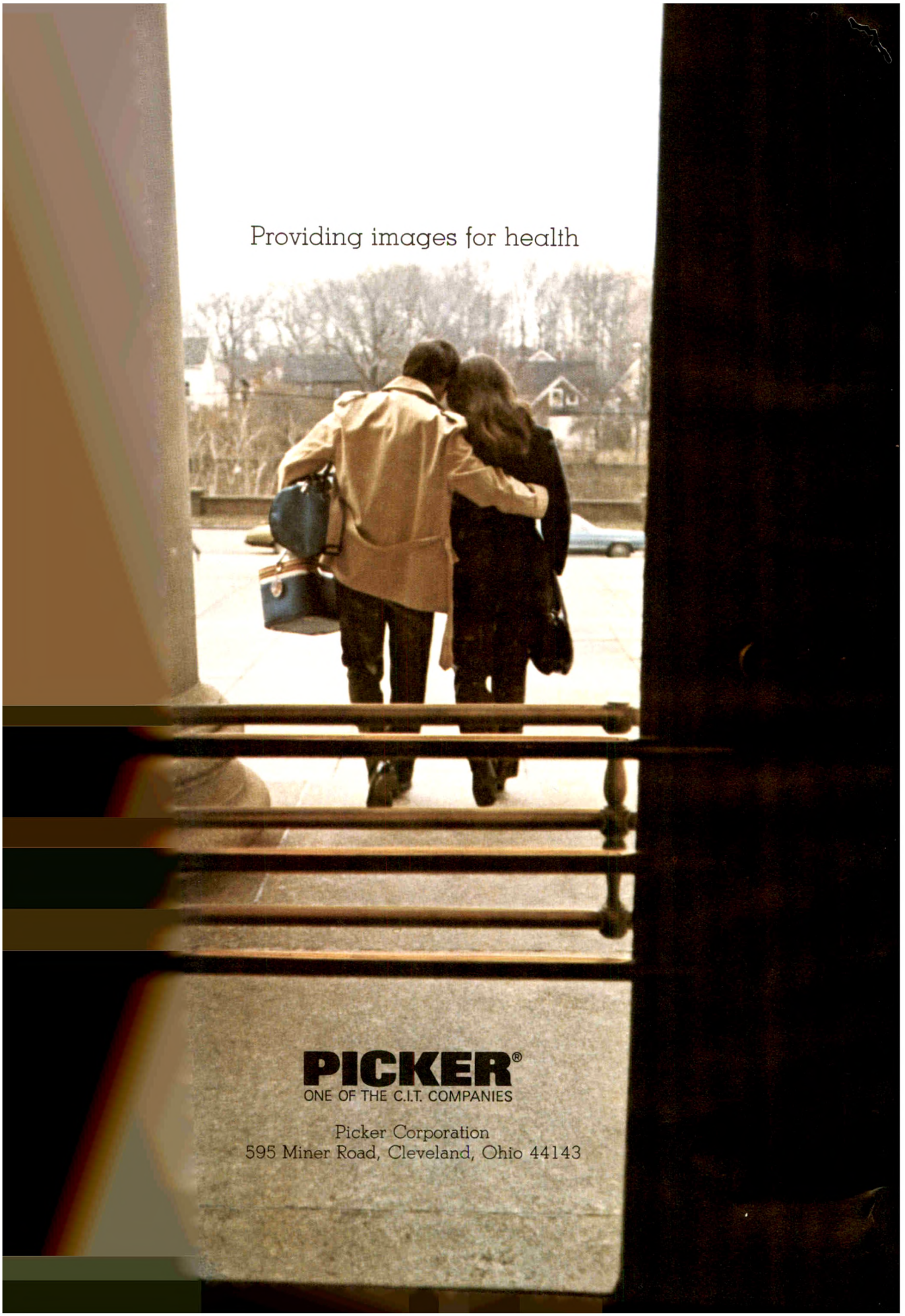


Therapy Simulator Room as seen from control booth. Photo courtesy Manitoba Cancer Treatment and Research Foundation, Winnipeg, Manitoba.

Other reasons to buy from Picker...

Now that you've seen some of Picker's diagnostic visualization and radiation therapy equipment in actual hospital applications, look at other aspects of Picker's working corporate philosophy. For example, our Film Systems and Supplies organization stocks over 3000 individual items to keep your Radiology Department operating smoothly, efficiently and economically. (A complimentary copy of the Picker catalog is available to you on request to your Picker representative.) And to close the circle of total commitment to the medical profession, Picker maintains the largest technical sales/service organization in the industry numbering over 1000 strong.




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WILLIAM B. YOUNG, M.D.
CALDWELL LECTURER, 1971

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 119 SEPTEMBER, 1973 NO. 1

INTRODUCTION OF THE CALDWELL LECTURER, 1971*

By WILLIAM B. SEAMAN, M.D.
NEW YORK, NEW YORK

Dr. William B. Young was born in Shanghai, China. He studied medicine at Oxford University and St. Thomas's Hospital, graduating as a Bachelor in Medicine and Surgery in 1936. He began his professional career as an otolaryngologist. In 1940 he joined the Royal Army Medical Corps, and was posted to Singapore. When Singapore fell to the Japanese, he was taken prisoner, along with Dr. James Bull, another prominent British radiologist, and held for 3½ years. He served as Company Medical Officer in various Japanese prisoner of war camps in Singapore and on the Siamese-Burma railway. At the end of World War II he was decorated by being made a Member of the Order of the British Empire.

Unfortunately, an ulnar nerve palsy resulting from a gunshot wound made it impossible for him to continue on in surgery. He became very friendly with James Bull during their time together as prisoners of war and from him became fascinated with radiology.

Returning to England after the war he studied radiology. At the same time that he took his examinations for certification in radiology he also took the examination for the surgical degree of Fellowship of the Royal College of Surgeons in Edinburgh. In this, the latter, according to Dr. Ronald Murray, another eminent British radiologist and at the time one of his fellow students, he startled the medical community by getting the Gold Medal.

He then served for 10 years in the Colonial Medical Service in Malaya as Director of the Diagnostic X-Ray Services in Singapore and later for the entire Federation of Malaya.

In 1957 he returned to London and worked in St. Thomas's Hospital and the Royal National Orthopaedic Hospital. Shortly afterward he was appointed Director of Diagnostic Radiology of the Royal Free Hospital in London.

His radiologic interests are many, and he has written extensively on the biliary system, tropical radiology, and skeletal system. He has collaborated frequently with Prof. Sheila Sherlock, Prof. of Internal Medicine, at the Royal Free Hospital and is one of the outstanding workers in the field of hepatology.

He currently is an examiner in Radiology and in Surgery for the Faculty of Radiologists as well as being the treasurer and a member of the Council of the same organization. In addition to his scholarly achievements and wide experience, he is a man of great personal charm and has a wide circle of devoted friends.

The subject that he has chosen to speak to you about tonight is "Obstructive Jaundice, The Radiologist, The Surgeon and The Patient."

It is a great pleasure and honor to have the privilege of introducing one of England's most eminent radiologists and tonight's Caldwell Lecturer, Dr. William B. Young.

* Presented at the Seventy-second Annual Meeting of the American Roentgen Ray Society, Boston, Massachusetts, September 28–October 1, 1971.

OBSTRUCTIVE JAUNDICE, THE RADIOLOGIST, THE SURGEON, AND THE PATIENT*

CALDWELL LECTURE, 1971

By WILLIAM B. YOUNG, M.D.
LONDON, ENGLAND

Mr. President, Ladies and Gentlemen, I am deeply sensible of the honor you do me, in inviting me to address you tonight. It is 53 years since Eugene Wilson Caldwell died—the man to whose memory this Lecture is dedicated—and I am the 51st guest in line who has had the privilege to speak to the members of this great Society on this special occasion. I am proud to stand where so many great and talented men have stood before, especially as there are many more able and fitted than I, both in your country and my own, to discharge this pleasant duty.

The study of Caldwell's life must prove an inspiration to any young and aspiring radiologist. As a young man, while training as an electrical engineer at the University of Kansas, he became involved with his chief, Lucien Blake, in experiments and research on submarine telephone communications, culminating with the transmission of the first message from Sandy Hook Lightship to Sandy Hook in 1892.

He continued his work in the fast expanding and exciting field of communications until 1896 when he read Röntgen's papers on his discovery of "x rays." The impact of this must have been something akin to that of Paul's vision on the way to Damascus, for within a year he had thrown in his job with the Metropolitan Telephone Co. of New York and spent his savings buying out a commercial photographer, who, as a sideline, had been dabbling in the "New Photography," which was what the formation of images on photographic plates by x rays was then called. He bought modest accommodations on 53rd Street and installed his apparatus, hoping to become a Consulting electrician to some hospital, and prepared, as his card read, "to do x-ray work for Physicians." In 1899 he invented a liquid interrupter for making and breaking the primary circuit of the induction coil which allowed him to make exposures as short as 1/5,000 second. He pioneered stereoscopic radiology in the United States, and in 1907 constructed a double focus Crookes' tube whereby a stereoscopic effect could be obtained on fluoroscopy, finally replacing this shortly before his death in 1918 by a method he had perfected using 2 self rectifying Coolidge tubes activated alternately to synchronise with an eye shutter device of the kind he had invented with his original design. Among his other inventions were a form of therapy tube for application to body cavities—an osmotic regulator for gas tubes—a vacuum valve tube—and an apparatus for the stereoscopic viewing of films based on the principle of the Brewster refracting prism. He drew attention to the fact that darkroom walls did not need to be painted black and in fact painted his darkroom light red, that both magnifying and reducing lenses were valuable when viewing x-ray plates, and was probably the first radiologist ever to perform a domiciliary x-ray examination in a house without an electrical supply—his ingenuity being such that to power his apparatus he hired an electrically driven taxi and connected his apparatus up to its battery by way of a wire passed through one of the windows.

In 1899 he was invited by the Gibbs foundation to equip an x-ray laboratory for the University and Bellevue Medical College, and was appointed Chief of department. In 1902, at the age of 32, although a very busy man, he embarked on the laborious task of qualifying in medicine, yet found time in 1903, to publish, together with Dr. William A. Pusey, one of the

* Presented at the Seventy-second Annual Meeting of the American Roentgen Ray Society, Boston, Massachusetts, September 28–October 1, 1971.
From the Royal Free Hospital, London, England.

earliest and most important books on radiology written in the United States, titled "The Practical Application of the Roentgen Rays in Therapeutics and Diagnosis," being responsible for the section on diagnosis. In spite of his commitments as a roentgenologist and a medical student, a second revised and enlarged edition was brought out in 1904. He qualified as a doctor in 1905 and in 1907 he published his famous article on "Skiagraphy of the Accessory Sinuses of the Nose," and the term "Caldwell's projection" is still used throughout the English speaking world today. Unfortunately by now he had developed a severe radiation dermatitis of his hands, and in 1908 he resigned his post at Bellevue having never received there the recognition his work deserved; and it was left to the Presbyterian Hospital to honor his achievements by appointing him Visiting Physician to the X-Ray Department in 1910.

In spite of having had to have 2 of his fingers amputated for skin cancer he joined the Armed Services of his country in the first World War—attaining the rank of Major—and as such he died in 1918 at the age of 48, a martyr to his calling.

To my regret I have no personal experience of this courageous man, who had such scientific and inventive ability. He was a Founder Member of your Society and one of its early Presidents: a true pioneer who, with others of his time, laid the foundations on which this great organization has built, and who set an example for all of us interested in the science and art of radiology to follow.

In an article on "The Safe Interpretation of Roentgenograms of the Gall-Bladder Region" published in your Society's JOURNAL *of 1915, he makes the point that "it is very easy to make a diagnosis of gallstones, but the difficult thing is to avoid making such a diagnosis when no stones are present." This was before the days of oral cholecystography, and stones could only be diagnosed with certainty if they contained sufficient calcium to show on the plates, or if they contained enough cholesterol to present as constant negative shadows cast against the soft tissues of the body. When one considers the technical quality of films and the careful observation required to differentiate them from collections of intestinal gas, perhaps we have not made all that much progress in spite of our more expensive and sophisticated equipment.*

Caldwell had always shown an interest in the Radiology of the Biliary Tract, and perhaps, therefore, it is not entirely inappropriate that I have chosen to speak to you on the subject of jaundice, and in particular, surgical or obstructive jaundice, and the part that radiology can play in the preoperative elucidation of its causes.

HISTORIC FACTS AND ANECDOTES

THE Greeks used the word ikteros to describe the yellow discoloration of the skin and eyes which we call jaundice. This was derived from the word ictides, a species of ferret with striking yellow eyes.

In early times jaundice was almost invariably due to infection, for epidemic diseases and malaria were both commonplace. Stones or other forms of mechanical duct obstruction were probably uncommon, and were not described in the earliest literature, for people died young, and in any case postmortem examinations were only performed on slaves or prisoners, many of whom lived on subsistence diets which were largely vegetarian.

Hippocrates, who taught medicine on the island of Cos around 460 B.C. and was himself descended from Aesculapius, the founder of Greek medicine in pre Homeric times, differentiated 4 types of jaundice due to diseases of the liver and 1 he believed to originate in the spleen, and taught that it was "a bad symptom when the liver becomes indurated." Gallstones were recognized, although their connection with jaundice was apparently not fully appreciated.

In early Roman times jaundice was known as Morbus Regis—the King's disease, possibly because of some form of hereditary transmitted hemolytic jaundice. The word jaundice was first used in

the English literature during the 14th century, and was derived from the terms jaunice or jaunes which were in usage during the 12th century, and were corruptions of the French word "jaune" meaning yellow. For instance, in 1340 Humpole wrote "for him that hath jaunes take wormot" (Red. Ant. 1).

Diocles of Karyostos seems to have been the first to have recorded the theory that jaundice could result from obstruction of the bile duct and Aretaeus of Cappadocia, in about 100 A.D., wrote "If the passages which convey the bile to the intestine be obstructed from inflammation or scirrhus, the bladder gets over distended and the bile regurgitates: it therefore becomes mixed with the blood which, passing over the whole system, carries the bile to every part of the body so that it acquires the colour of bile." This is a very accurate description—especially if one considers that it was made nearly 1900 years ago—but the early Roman physicians were ahead of their time and their opinions went disregarded for some centuries.

Since the time of Hippocrates, and possibly dating back even further to Pythagoras (530 B.C.), the theory had been developed that health and ill health depended on a delicate balance or imbalance of certain humors which were formed in the liver and spleen from food carried to it from the intestines, and which were then dispatched to the various parts of the body in the blood, although the pathways by which this was achieved were not properly understood until after Harvey's description of the circulation in 1628.

Galen, who was born in Pergamon on the shores of Asia Minor and later moved to Alexandria in Egypt, was chiefly responsible for the elaboration and dissemination of this theory, for as a result of the publication of his book "de Elementis" in about 180 A.D. it became almost universally accepted throughout the then civilized world. Galen taught that good health necessitated a correct balance between four principal humors, blood, phlegm, yellow bile, and black bile, each of which existed in a natural or beneficial form or in an unnatural or harmful form, so that when the balance between these became disturbed due to an excess of one, or deficiency of another, illness would result (Table 1).

Galen's doctrine of humors was expanded and elaborated by succeeding generations of doctors throughout Europe, perhaps most notably by Guy de Chauliac, and remnants of it persisted in medical teaching in Britain well into the nineteenth century.

The concept of black bile, for which there is no known scientific basis, can be traced back to Hippocrates, who taught that it was formed by the liver, and then delivered, in a retrograde manner, to the spleen *via* the portal and splenic veins—from which, through venous junctions, it was secreted into the stomach to aid digestion. When excreted in excess, it was believed to be responsible for icterus, leprosy and mental disease, among others.

Galen taught that foreign bodies such as grain, kernels of pomegranates and seeds of figs could enter and obstruct the bile ducts, possibly mistaking small stones in the ducts found at autopsy for these substances. He claimed that stones found in the gallbladder were formed by coagulation of bile due to excess heat in the liver, and believed that "bitter bile" could be

TABLE I

Humor	Primary	Element	Season	Temperament
Blood	Hot and moist	Air	Summer and Spring	Plethoric
Phlegm	Cold and moist	Water	Winter	Phlegmatic
Yellow bile	Hot and dry	Fire	Summer and Fall	Choleric
Black bile	Cold and dry	Earth	Autumn	Melancholic

retained in the body as a result of weakness of the excreting and conducting organs, for the accepted concept at that time was that the gallbladder filled by attracting bile into it. He recognized, however, that impairment of the biliary passages could be caused by inflammation, induration or obstruction, or by pressure from surrounding tissues, and that this might result in jaundice.

It was not until 1795 when William Saunders, Senior Physician to Guy's Hospital, London, tied the bile ducts of 2 living dogs, that it was scientifically established that obstructive jaundice was a direct result of bile retention and resorption. Saunders also made the important observation that water injected retrogradely up the bile duct above the ligature would escape readily into the hepatic veins, even though very little force was used—a point to be considered even today by those undertaking operative or T-tube cholangiography, for this would seem to be a possible path for the spread of infection.

Bright in 1836 wrote an excellent description of jaundice and its various causes which in many ways still cannot be bettered, but even at this time the sun of scientific medicine had still not yet fully broken through the clouds of Galenic dogma and the mists of medical superstition which had obscured man's vision and halted the progress of medicine throughout the middle ages.

Morgagni was probably the first to formulate the theory that suppression of liver function would result in bile being retained in the blood. However, until the revolution in medical thought and knowledge (which stemmed from the pathologic studies of Virchow in 1867), the development of anesthesia (ether was first used as an anesthetic for a major surgical operation in 1846 by William Morton, in this very city, at the Massachusetts General Hospital), and the development of antiseptic surgery by Lister (whose first paper was published also in 1867)—all of which were initially reviled, rejected or vehemently opposed by many of the elite in medicine at the time—Galenic teaching, with its concept of humors, continued to influence British medicine as well as national thought and ideas into the middle of the nineteenth century.

For instance, in the Cyclopaedia of Practical Medicine, one of the standard English medical textbooks on medicine, published in 1834, the chapter on jaundice opens as follows: "The circumstances which predispose the system to an attack of jaundice are numerous. The melancholic or leuco-phlegmatic temperament, under mental excitement whether pleasant or painful; an indolent mode of life; a full and stimulating diet; are among the most powerful." It goes on "We have long felt assured that the status of the brain and nervous system has been too little considered in the pathology of jaundice, and many reasonably suppose that any considerable deviation from the sound and vigorous condition whereby they influence secretion and moving powers will be likely to lessen or deteriorate the biliary fluid. We may hope at some future period to clear up many obscure points relating to biliary secretion and arrive at more successful methods not only of removing jaundice but also in many instances of preventing its occurrence."

Indeed remnants of Galenic teaching survive still in our common language and literature. Bile has become synonymous with resentment, and gall with spite. A jaundiced eye denotes embitterment, bias, or prejudice; melancholic denotes a black mood or despair; while choleric is a term interchangeable with irascible, testy, or liverish—adjectives often applied to senior officers of the old Indian and British armies.

Shakespeare, who was very much a man of his time, obviously accepted these connotations without question, and in "Troilus and Cressida," first performed in 1606, Agamemnon asks his army commanders "What griefs have set the jaundies on your cheeks?" In "Henry IV," the hostess

speaking to Captain Pistol cries "I beseech you now aggravate your choler;" while in "Twelfth Night" Sir Toby says to Sir Andrew "Let there be enough gall in thy ink, though thou write with a goose pen no matter."

Dryden in 1687 wrote of "Jealousie, the jaundice of the soul;" he may have taken this concept from the ancient Jewish Talmud, where 83 causes of jaundice were listed, and there is a specific warning that jaundice may be a punishment for jealousy.

Pope in 1709, in his "Essay on Criticism" coined the phrase "all looks yellow to the jaundiced eye," and this—colourful—concept has caught the eye of many a journalist since—jaundiced or otherwise. For instance in 1887 the Western Review contained a report which read "Mr. Chamberlain's view of the Irish people has become suffused with a jaundiced colour." It is possible that, had Mr. Chamberlain been alive today, the sentiment expressed might well have been equally appropriate.

Here in the United States you have a connotation entirely your own. In 1843, Kirby wrote in "Entomology"—"the icteric oriole (incidentally my secretary typed 'icteric creole') is kept by the Americans in their houses to clear them of insects." I would hazard that in this instance the term has nothing to do with Baltimore or New Orleans and is probably being used descriptively for a bird—though the mind boggles at the thought of the modern American home of necessity being turned into an aviary. While, in a typically English manner, I am loath to becoming involved in political issues outside my own country, I must say that perhaps in this particular instance you owe more to insecticides than the antipollution critics might have one believe.

The next great stimuli to the advancement of scientific medicine were provided by Röntgen's discovery of x rays in 1895 and the Curies' discovery of radium in 1898. No doubt those of you who visited the Courtway Library last evening will have noticed that the first x-ray taken by Röntgen was not of his own, but of a lady's and probably his wife's hand! But then, who better? Be that as it may, these discoveries have revolutionized medical and surgical diagnosis and have opened the windows of progress to vistas, the boundaries of which, as yet, are nowhere in sight.

It is to some of the radiologic techniques which have stemmed from these discoveries and which may be applied to a patient suffering from obstructive jaundice in an effort to explain its origin before resorting to surgical exploration, that the rest of this lecture will be devoted.

Before embarking on it however, I should like to pay tribute to my many friends—at the Royal Free Hospital, London and elsewhere—both in the United States and in Britain—for the help they have given me, either in advice or in the loan of slides or illustrations. They are too numerous to mention individually by name and it would be invidious to do so in case I should inadvertently omit some. To them all, therefore, I express my warmest thanks.

CLASSIFICATION OF JAUNDICE

Jaundice is usually classified under three main headings.

1. Hemolytic jaundice, due to overloading of liver cells with bilirubin (such as acholuric jaundice, erythroblastic and sickle cell anemia, malaria, yellow fever, etc.). These are unlikely to present as surgical problems unless there are associated gallstones.
2. Hepatocellular or toxic jaundice, resulting from an inability of the liver cells to excrete bilirubin. This is usually entirely a medical problem, though it may at times pose problems in differential diagnosis.
3. Surgical or obstructive jaundice, due to mechanical interference with the flow of bile down the ducts.

At times there may be a combination of obstructive and hepatocellular factors at work.

CAUSES OF OBSTRUCTIVE JAUNDICE

The commoner causes of obstructive jaundice are as follows:

I. EXTRAHEPATIC OBSTRUCTIVE JAUNDICE

A. *Congenital*
   1. Atresia of bile ducts (presenting at birth)
   2. Choledochal cyst (presentation often delayed for some years)

B. *Inflammatory*
   1. Sclerosing cholangitis
      (i) idiopathic
      (ii) acquired (2° to calculus disease, 2° to ulcerative colitis, etc.)
   2. Oriental cholangio-hepatitis (usually due to worm infestation, *Clonorchis* or *Fasciola*)
   3. Papillitis or fibrosis of sphincter of Oddi
      (i) without stones in the common bile duct
      (ii) with stones in the common bile duct

C. *Traumatic*
   1. Spontaneous rupture of bile duct (rare)
   2. Blunt or direct injury to duct
   3. Attributable to surgery
      (i) immediate, due to loss of duct continuity
         (a) section of duct
         (b) ligation of duct
      (ii) delayed stricture
         (a) following partial section or ligation of duct
         (b) duct fibrosis following exploration of duct
         (c) periductal fibrosis subsequent to leakage of blood or bile around duct

D. *External compression of bile duct*
   1. Nonmalignant
      (i) chronic pancreatitis
      (ii) penetrating duodenal ulceration
      (iii) duodenal diverticulum (rare)
      (iv) hydatid cyst (uncommon)
      (v) aneurysm of hepatic artery or aorta (rare)
      (vi) retroperitoneal fibrosis
   2. Malignant
      (i) carcinoma of head of pancreas
      (ii) carcinoma of gallbladder
      (iii) enlarged glands
         (a) metastatic carcinoma
         (b) reticulosis

E. *Obstruction within the lumen of the bile duct*
   1. Calculus (usually intermittent; occasionally complete or progressive)
   2. Neoplasm (usually progressive; occasionally intermittent)
      (i) benign
         adenoma or polyp of bile duct or ampulla
      (ii) malignant
         (a) carcinoma of bile duct (including ampulla)
         (b) carcinoma or lymphoma of duodenum involving ampulla
   3. Intestinal parasites (*Clonorchis*, *Ascaris*, *Fasciola hepatica*)

II. INTRAHEPATIC OBSTRUCTIVE JAUNDICE

A. *Intrahepatic cholestasis*
   Primary biliary cirrhosis
   Steroid-induced cholestasis
   Jaundice associated with ulcerative colitis
   Phenothiazine drugs (*e.g.*, chlorpromazine)
   Chronic virus hepatitis
   Alcoholic cholestasis
   Benign recurrent jaundice of pregnancy

B. *Intrahepatic bile duct obstruction*
   Intrahepatic bile duct atresia
   Carcinoma of intrahepatic bile ducts
   Primary carcinoma of liver (hepatoma)
   Hydatid disease
   Metastases (usually late)
   Liver abscess (rare)

CLINICAL PROCEDURE

When a patient presents with jaundice the first task of any physician is to determine whether it is hepatocellular or obstructive in origin; and, if obstructive,

whether this is due to stone, benign stricture or carcinoma.

In making his diagnosis he may be assisted by the history—such as the fever, rigors etc. with acute hepatitis; or pain, colic and fever in acute obstructive jaundice. However, in cases of more prolonged jaundice, even after all relevant laboratory tests have been performed and a liver biopsy examined, it may still be impossible to differentiate with certainty between mechanical obstruction and intrahepatic cholestasis—and it is at this stage that radiology and other diagnostic techniques such as radionuclide scanning or ultrasound may prove invaluable, for they then offer the only prospect of making the diagnosis short of surgical laparotomy.

Admittedly in many centers, at this stage, laparotomy is undertaken on the grounds that if an obstruction exists it should, if possible, be relieved or bypassed. But cases suffering from hepatocellular disease are poor operative risks and an anesthetic may be sufficient to precipitate acute liver cell failure or death, so that there may be an advantage—at least to the patient—if the correct diagnosis can be made in advance.

Even when clinically it seems reasonably certain that jaundice is due to large duct mechanical obstruction, it is obviously helpful to the surgeon to know before he makes his incision the exact site of the obstruction, its cause if possible, and the amount and state of the duct available proximal to the obstruction for anastomosis or bypass if this is necessary. Nowhere is this more important than in cases who have undergone previous surgery, especially if there has been more than one previous operation, such as when dealing with postoperative strictures of the duct, or a missed high carcinoma of the bile duct, for normal anatomy may be grossly deformed by scar tissue or concealed by adhesions between different organs in the neighborhood. However, it applies to whatever the cause, for the surgeon can plan his procedure in advance, or make alterations to his list, for a stone will probably present an easier problem than a carcinoma of head of pancreas; furthermore, a high malignant obstruction, or the demonstration of hepatic metastases, may decide the surgeon not to operate, or to undertake a palliative procedure rather than a more heroic surgical resection doomed to failure from the start. When jaundice is due to a stone it is obviously helpful to demonstrate how many are present, although this is not so important as showing later whether any remain in the ducts before the abdomen is closed.

ROENTGENOLOGIC EXAMINATION

PLAIN FILM ROENTGENOGRAPHY[5,12,22]

Plain roentgenograms of the abdomen may show enlargement of the liver and/or spleen and point to primary or secondary malignant disease or cirrhosis as the likely cause of the jaundice (Fig. 1). Occasionally the shadow of an enlarged gallbladder can be made out and indicates, as Courvoisier pointed out, a malignant lesion involving the common bile duct; or varices may be detectable behind the cardiac shadow, or be visible on a plain film roentgenogram of the chest (Fig. 2), and point to cirrhosis as the cause. The chief value of plain film roentgenography, however, lies in the dem-

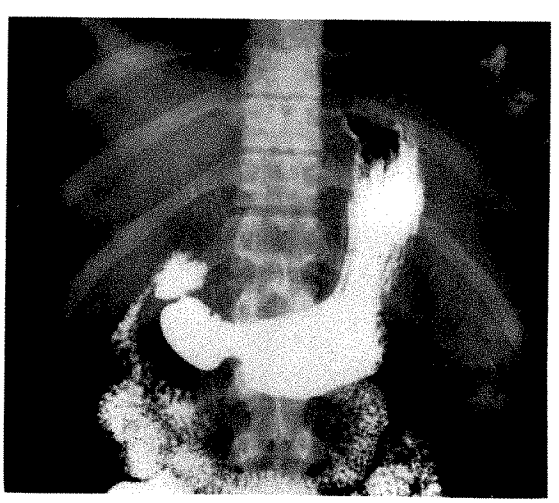


Fig. 1. Cirrhosis of liver. Large liver and spleen; calcification in wall of portal vein; varices in gastric fundus.

onstration of gallstones, various types of calcification of the liver or pancreas, or gas in the bile ducts.

Less than 20 per cent of stones in the gallbladder and only some 5 per cent of stones in the bile duct are sufficiently calcified to show. And even should stones be shown in the gallbladder this does not necessarily mean that they are the cause of the jaundice, for stones are found in the gallbladder at autopsy in some 30 per cent of females, and in 15 per cent of men, over 55 years of age, and are said to be a cause of carcinoma of the gallbladder. They are not uncommonly found in cases of pancreatitis and it is said that the incidence of stones is higher in individuals suffering from cirrhosis of the liver and carcinoma of the head of the pancreas than in others of the same age groups. Similarly, while calcification in the pancreas suggests the presence of pancreatitis, this does not exclude an associated carcinoma. In this context, if previous roentgenograms of the abdomen are available, absorption of previously noted calcification or an alteration in its shape and distribution may indicate the presence of such a growth.

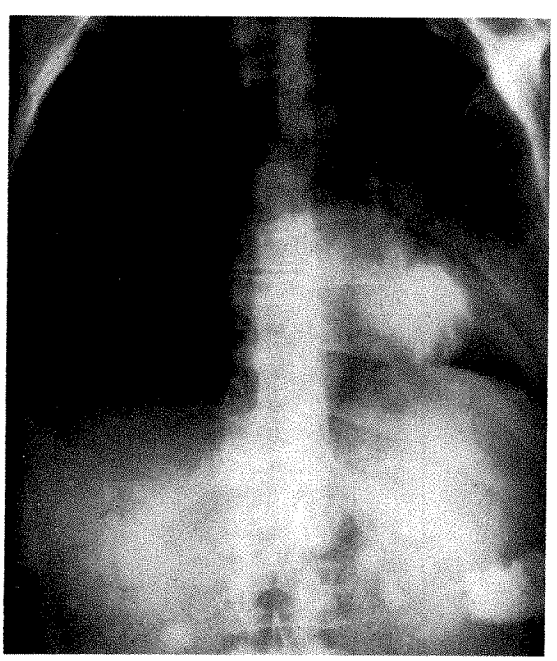


Fig. 2. Cirrhosis of liver. Varices are visible behind the cardiac shadow on the plain film roentgenogram of the chest.

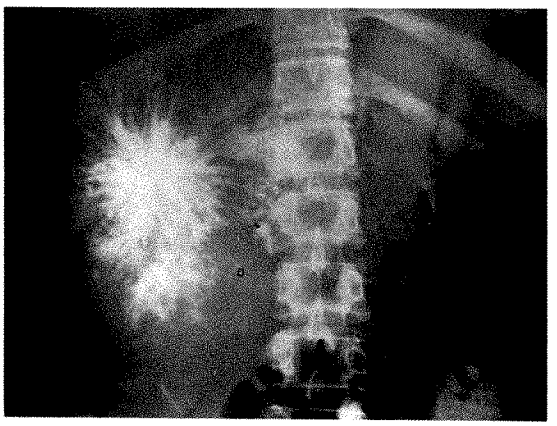


Fig. 3. Bizarre ossification in a hepatoma.

Calcification not uncommonly occurs in a hepatoma and also in metastases from carcinoma; hydatid cysts; angiomas; tuberculomas; or gummas. Calcification in a hepatoma tends to present as a collection of small soft ill-defined opacities of varying sizes but may sometimes present as denser opacities, with a bizarre pattern (Fig. 3). According to Polk, calcification in a carcinoma of the gallbladder is not uncommon.

Calcification in metastatic deposits tends to be ill-defined and soft, mottled or nodular. It is usually secondary to a mucus secreting adenocarcinoma of large bowel, though occasionally from the stomach, and very rarely from the endodermal element of a teratoma. Similar calcification may be seen in metastases from other tumors such as an adrenal neuroblastoma and rarely in metastases from a calcitonin secreting carcinoma of the thyroid gland. Ring calcification in the wall of a hydatid cyst usually indicates that the cyst is dead, but live cysts are not uncommonly present elsewhere in the liver. Patchy or ring calcification may rarely be seen in gummas, tuberculomas or chronic abscesses. In angiomas calcification usually takes the form of phleboliths or wavy or parallel lines. Ossifying metastases may very occasionally be found in the liver secondary to bone osteosarcoma.

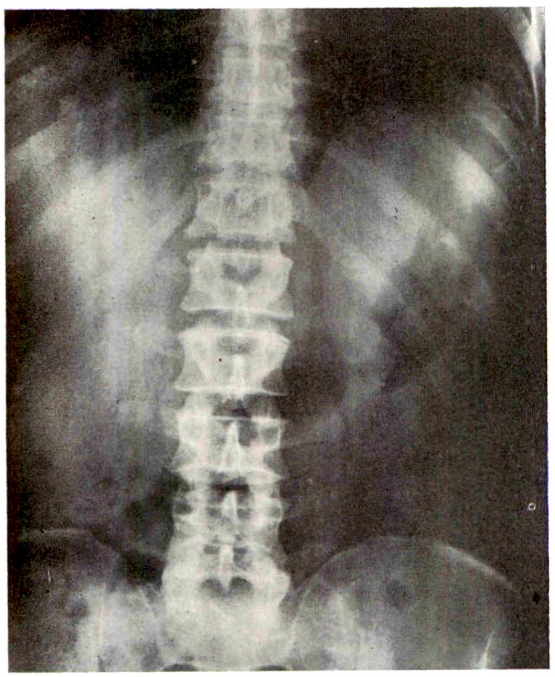


Fig. 4. A carcinoma of the stomach delineated by gas.

Occasionally, a diagnosis can be made from the plain film roentgenogram, as shown in Figure 4, of a gas silhouetted carcinoma of the stomach, with secondaries in the liver and lung.

In a case of unexplained chronic jaundice the wrist and ankles should be examined, for we have found that in approximately 10 per cent of cases suffering from primary biliary cirrhosis, periosteal layering of a type similar to that seen in hypertrophic pulmonary osteoarthropathy will be found, usually with clubbing of the fingers (Fig. 5, *A* and *B*). Similar changes may occasionally be seen in chronic active hepatitis, or in a very slow growing carcinoma of the bile ducts.

Barium Techniques[3,16,19–21,33]

Examination of the upper gastrointestinal tract with barium is mandatory in all cases of persistent jaundice in which the cause is undiagnosed, and, after scans of the liver and pancreas, and, possibly, liver biopsy, should be the first procedure after the plain film roentgenographic examination, particularly if this is negative.

The state of the esophagus—especially the presence or absence of varices, and their size—allows one to assess the risk of passing a gastroduodenoscope, should this be contemplated. Esophageal varices almost always indicate portal venous obstruction and suggest that jaundice is due to some form of cirrhosis, although it should be remembered that there is a definite increased incidence of gallstones and chronic pancreatitis in cirrhotics. There is almost invariable splenic enlargement if the organ has not been removed. A carcinoma or other malignant lesion of the stomach may be demonstrated, or some lesion in the duodenum such as a perforating ulcer, diverticulum or even a carcinoma which may be involving the bile duct. A widened duodenal loop; disturbance of the mucosal pattern on the concavity of the loop; pressure indentations on the loop or Frostberg's reversed E or sigma deformity; or elevation and anterior displacement of the duodenal bulb or pylorus may suggest a neoplasm of the head of the pancreas.

Forward displacement, or anteroposterior compression, of the 2nd part of the duodenum, shown best by lateral decubitus roentgenograms with a horizontal beam; or lack of forward displacement of this part of the duodenum on a left lateral decubitus roentgenogram, compared with the opposite lateral roentgenogram, may provide valuable information regarding fixation which is often missed by failing to take such views. A supine lateral roentgenogram using a horizontal beam is, in my opinion, not very helpful because, although forward displacement of the stomach in relation to the spine may indicate a growth in the body of the pancreas, by the time a growth of the head has extended to this extent it is almost certainly not resectable.

In Britain retroperitoneal pneumography has, on the whole, been neglected or rejected as a worthwhile technique in the search for a growth in the head of the pancreas, possibly for the same reason.

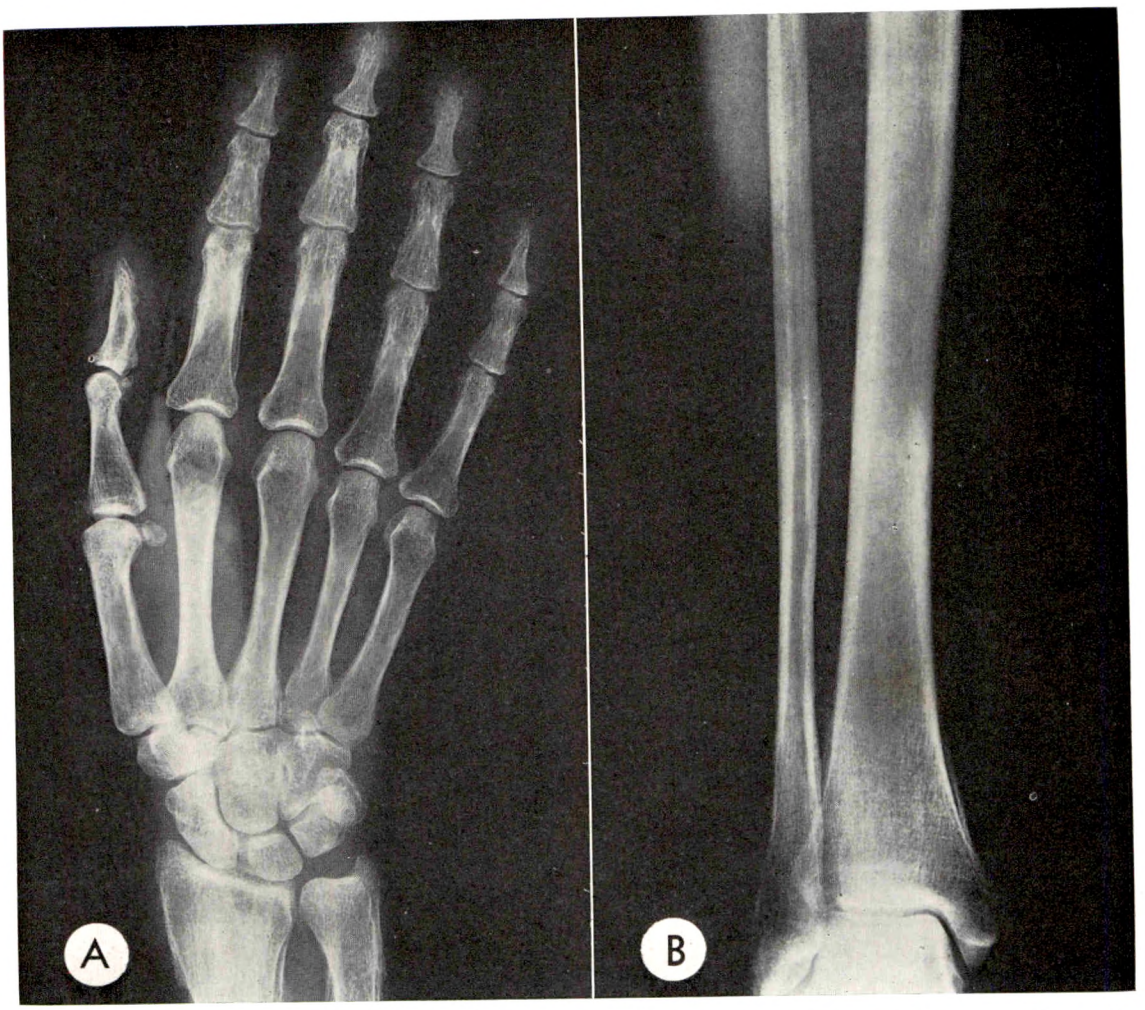


Fig. 5. (*A* and *B*) Primary biliary cirrhosis. Periosteal new bone resembling hypertrophic osteoarthropathy with clubbing of fingers.

Not uncommonly a pancreatic neoplasm will produce a pressure deformity on the lateral aspect of the second part of the duodenum or may encircle it and compress it from side to side (Fig. 6). A filling defect on the concavity of the duodenal loop in the region of the papilla may be due to a dilated common bile duct containing stones or a single large stone (Fig. 7), but almost identical appearance may be produced by a carcinoma of the ampulla (Fig. 8) or even by a carcinoma of the head of the pancreas (Fig. 9), although the margins of the defect when due to stone are usually more clear cut than when due to a growth. A dilated bile duct may impress the duodenum where it passes behind it, usually at the junction of the first and second parts, as in Figure 10, a carcinoma of the head of the pancreas. Occasionally, with a carcinoma of ampulla, or after the recent passage of a stone, a little barium will reflux up the common bile duct.

During the examination the barium filled duodenum should be paralysed by the intravenous injection of some substance such as oxyphenonium sodium, 0.6 mg. (Antrenyl, Ciba). Loss of the coiled spring like folds on the medial side, especially if combined with rigidity or serration of the medial wall with projections resembling rose thorns, suggests a growth in the head of the pancreas (Fig. 11, *A* and *B*). A smooth rigid

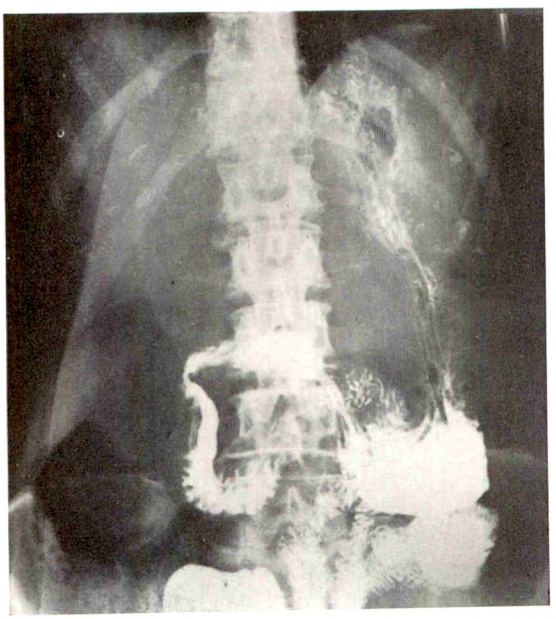
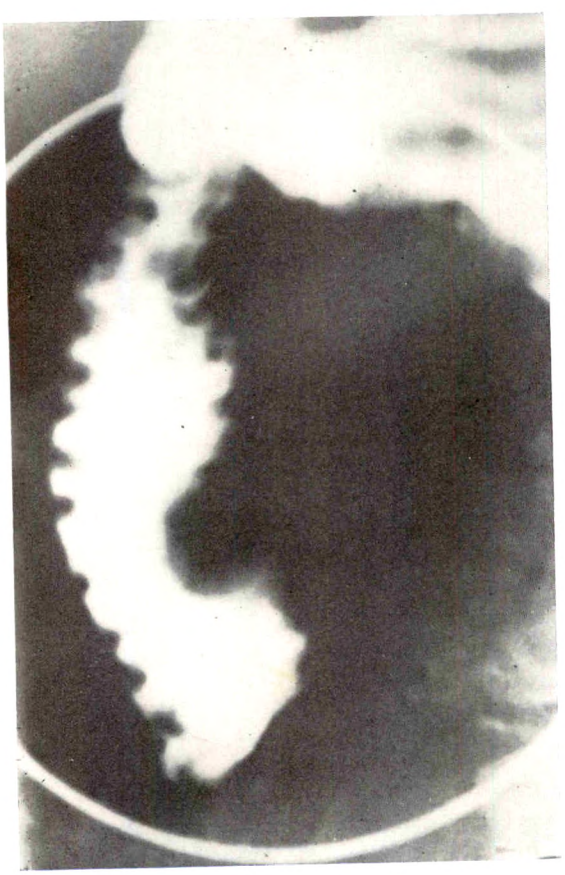


Fig. 6. Barium meal examination. Large gallbladder visible on plain film roentgenogram. The lateral wall of the second part of the duodenum is compressed by carcinoma of the head of the pancreas.



medial wall may indicate chronic pancreatitis (Fig. 12, *A–D*), but it is often impossible to differentiate between the two conditions on appearances alone, as shown in Figure 10, due to a carcinoma (Fig. 13). Calcification in the pancreas usually indicates the presence of chronic pancreatitis, but does not exclude a carcinoma.

Disturbances of the mucosal pattern in the region of the duodenal papilla (Fig. 14, *A–D*), especially if combined with a localised finger-end like pressure defect on the medial duodenal wall, usually indicate an ampullary growth, although this can only be diagnosed with confidence if the defect is constant on two or more films or if a tumor can be silhouetted against gas or air introduced into the duodenum during

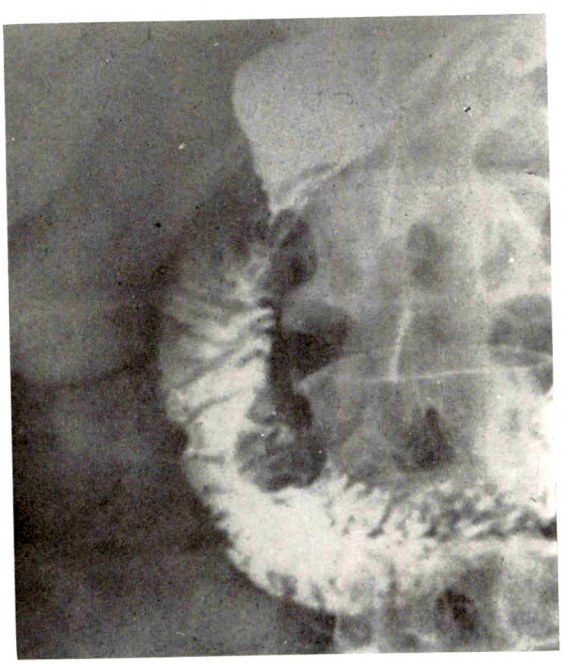


Fig. 8. Barium meal examination. The filling defect is due to carcinoma of the ampulla. Note oblique pressure effect of dilated bile duct on the upper part of the descending duodenum.

Fig. 7. Barium meal examination. The filling defect in the region of the ampulla is due to a stone in the bile duct.

hypotonic duodenography. Even so, it may be impossible to differentiate a primary carcinoma of duodenum, which, although uncommon, is only rarely situated sufficiently close to the ampulla to involve it.

It has been claimed that cine-roentgenographic examination of the barium filled duodenal loop increases the chances of diagnosing a carcinoma of the head of the pancreas by demonstrating significant differences in the pattern of duodenal peristalsis or flexibility of the medial duodenal wall. Any method that may increase the accuracy or scope of diagnosis is worth trying or combining with accepted techniques, but in my experience cine-roentgenography, in itself, is little if any more effective than conventional methods in reliably distinguishing between carcinoma and chronic pancreatitis, which is what really interests the surgeon. The findings are not always easy to interpret, and false positive diagnoses are not uncommon. While, therefore, it may have a place in the examination of selected cases, the additional radiation delivered has to be weighed against its possible value in routine examinations. Video-tape recording does not suffer from these disadvantages and when reproduction is improved it may become the method of choice.

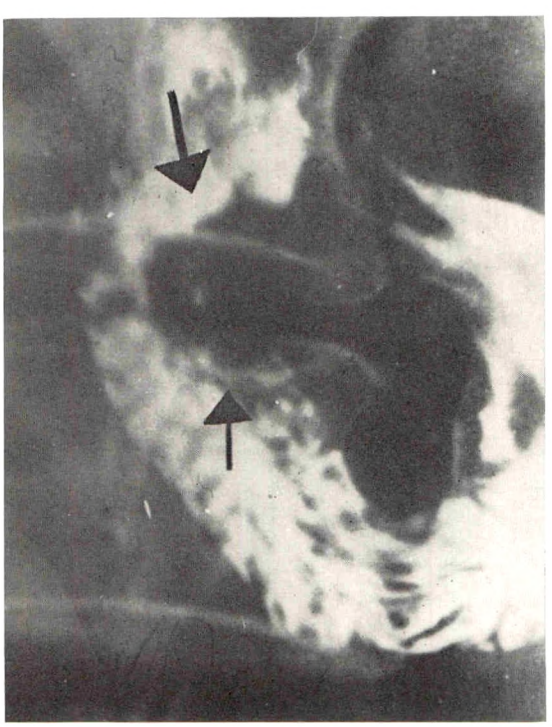


Fig. 9. Barium meal examination. The somewhat irregular filling defect on the duodenal concavity is due to a carcinoma of the pancreas (arrows).

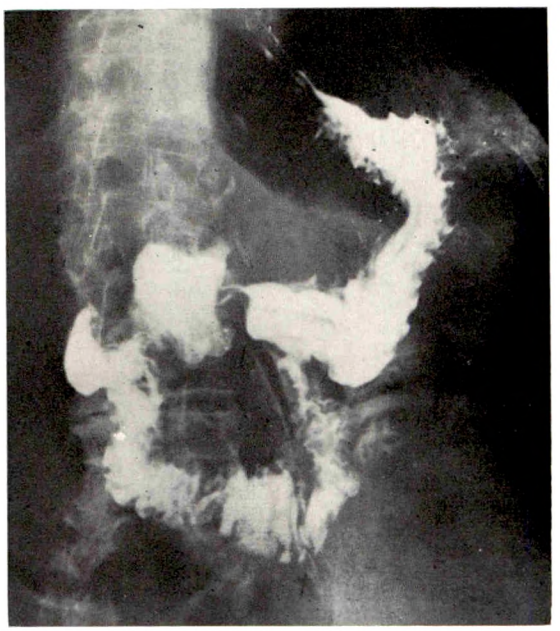


Fig. 10. Note oblique pressure effect on proximal duodenum by a dilated bile duct. A carcinoma of the head of the pancreas was obstructing the duct lower down.

Attention should always be paid to the caliber and mucosal pattern of the small bowel, for in chronic pancreatitis, though also occasionally with a slow growing carcinoma of the head of the pancreas, prominent plical folds and increased caliber may result from deficiency of normal pancreatic secretion.

Unfortunately, none of the barium techniques described, however carefully performed, offer much prospect of a correct diagnosis of malignant lesions of the pancreas while still small enough to offer hope of adequate resection unless the growth early involves the terminal segment of the bile duct or the duodenum in its immediate vicinity. Even then, as has already been shown, a nonopaque stone impacted in the lower end of the bile duct or a carcinona of the pancreas or ampulla may present almost

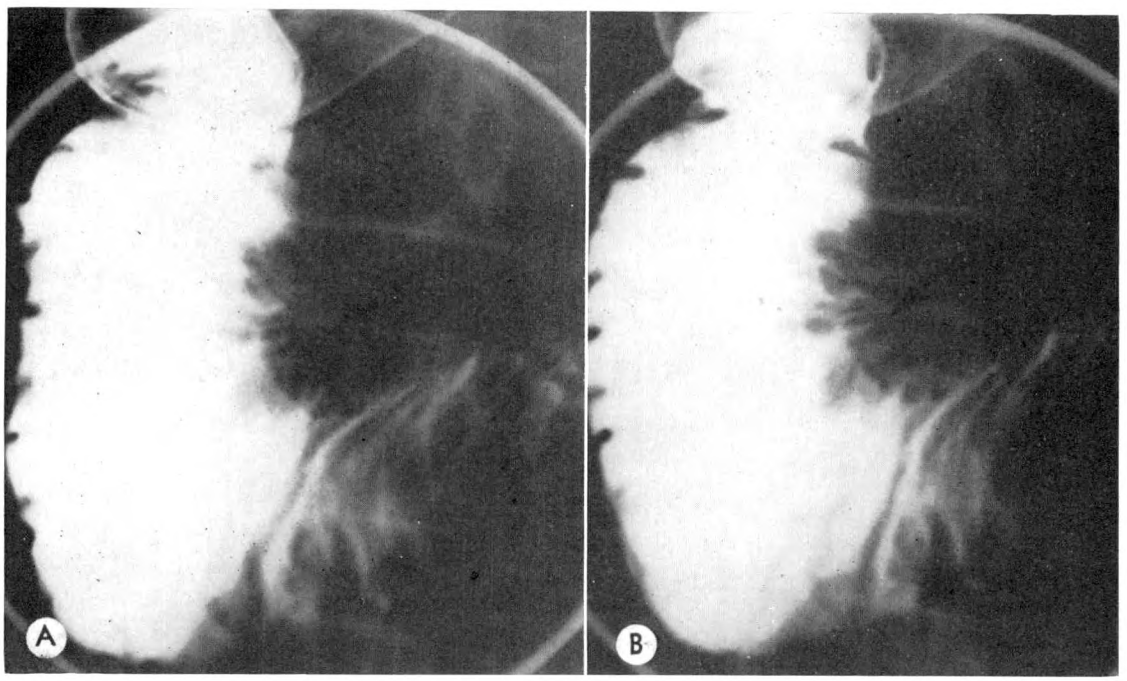


FIG. 11. (*A* and *B*) Hypotonic duodenography. Typical rose thorn projections due to carcinoma of pancreas. (Courtesy of Dr. L. Kreel.)

identical appearances. Should any abnormality be noticed, however, further investigations, and preferably laparotomy, should be undertaken as a matter of urgency to determine the true situation.[11,13,26,29,39,45]

Similarly a carcinoma of the head of the pancreas may produce changes which are indistinguishable from those usually attributed to chronic pancreatitis.[19,31]

Enlargement of the duodenal loop is very difficult to assess, for individual variation is so great, and in hypersthenic subjects the loop normally takes a wide sweep. Definite compression with narrowing of the second part of the duodenal loop, if constant, is highly suggestive of a carcinoma of the head of the pancreas, but the inverted E or sigma sign of Frostberg is, in my experience,

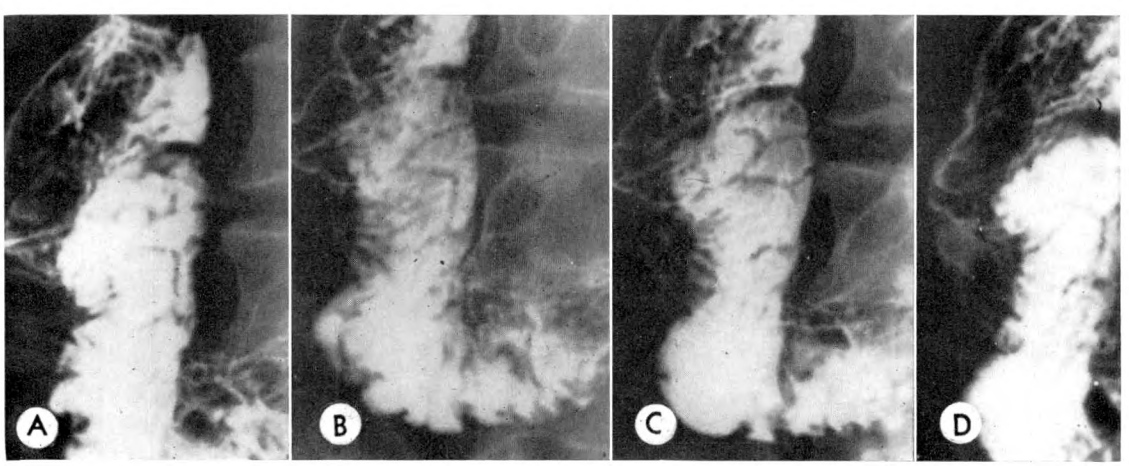


FIG. 12. (*A–D*) Chronic pancreatitis. Hypotonic duodenography without gas distention. Smooth rigid medial wall of second part of duodenum.

only present in some 5 or 10 per cent of cases, and usually only when growths are too large to offer much hope of successful surgical resection. It may occasionally be seen in otherwise apparently normal individuals.

Hypotonic duodenography is, in most cases, only a little less unreliable than a conventional barium examination except in the diagnosis of intraluminal lesions in the region of the ampulla, for rose thorn serrations of the medial wall may occasionally be found in apparently normal individuals, especially if the lumen is not fully distended with gas. Subtle changes in the mucosal pattern of the second part of the duodenum, whether with air distention or not, are often very difficult to assess and the differential diagnosis between carcinoma, chronic pancreatitis and variations of normality may at times be impossible. Although the commonest cause of malignant obstruction of the bile ducts is a carcinoma of the head of the pancreas, false positive diagnoses are not uncommon, and not infrequently a case is diagnosed as a carcinoma of the head of the pancreas on a faulty assessment of changes in the duodenal mucosal pattern, or because the recognised signs are themselves unreliable, when no carcinoma exists or when the site of obstruction lies above the level of the pancreas. It is perhaps therefore fortunate that newer techniques of gastroduodenal endoscopy made possible by the development of flexible fiberscopes have developed to a stage where endoscopy can provide the physician with additional diagnostic information and complement existing and longer established radiologic techniques.

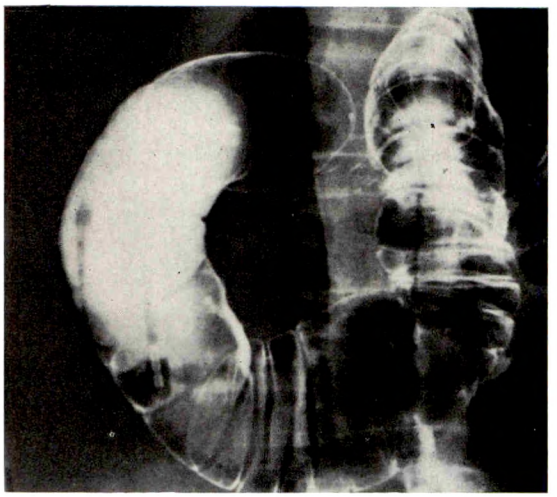


Fig. 13. Hypotonic duodenography. The appearances are more those of chronic pancreatitis but are due to carcinoma of the pancreas.

Cholecystography and Cholangiography[2,39,43,44,47,51,52]

The major weakness in the radiologic investigation of jaundice is that none of the opaque media at present available for cholecystography and cholangiography, because of the suppression of liver function, is consistently effective in the presence of hepatocellular or sustained obstructive jaundice, and the gallbladder and ducts therefore cannot be opacified by usual contrast methods. In my experience satisfactory opacification of the ducts by intravenous cholangiography, whether by injection or infusion, even with the use of more recent and more expensive opaque media, can rarely be satisfactorily achieved if the serum bilirubin level is above 4.0 mg. per cent and often fails in the 2–4 mg. per cent range, especially if the jaundice has been prolonged or is increasing in intensity. This is in accordance with the opinions of experts such as Wise[52] and Baker and Hodgson,[2] who have unparalleled experiences in this field. Intravenous cholangiography may be effective within this range of serum bilirubin levels when the jaundice is intermittent or decreasing in severity, and therefore success is more likely when obstruction is due to stone than neoplasm. Should opacification of the duct system be achieved, a stone may be shown by tomography or inferred by abnormal stasis in the duct with increasing opacification of the dilated duct system over a period of 1–2 hours, as first described by Wise and O'Brien[51] in their time density concept. The administration of substances such as penditan to reduce renal tubular excretion of bilirubin rarely seems to improve the degree of opacifica-

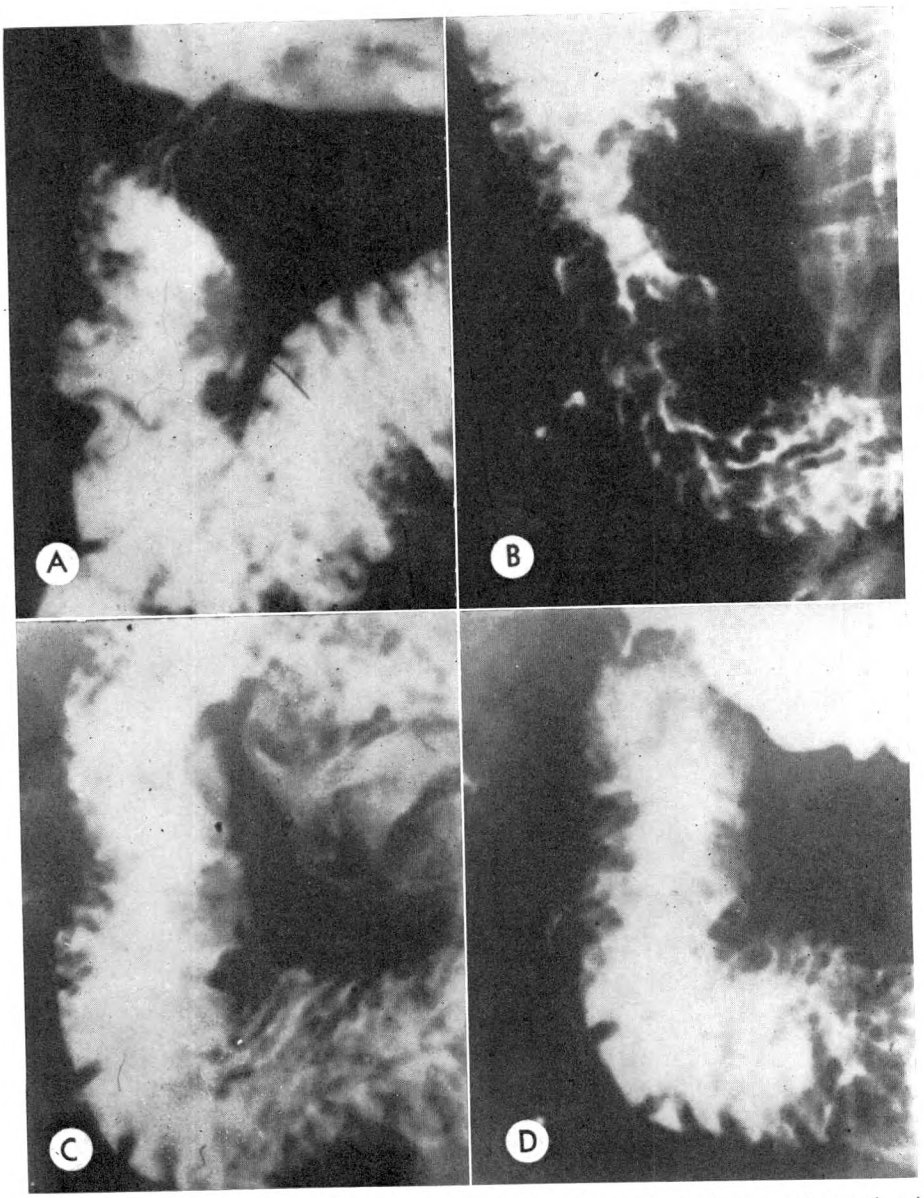


Fig. 14. (*A–D*) Carcinoma of the ampullary region of the bile duct. The barium meal examination shows a constant filling defect in the region of the papilla, proving the value of serial spot roentgenograms.

tion and may be dangerous. A drip infusion method using glucose saline may be slightly more effective in opacifying the ducts than simple intravenous injection of the opaque medium, but this is based on subjective impressions and in man is almost impossible to prove. Whether so or not, the method has less side effects, is probably safer, and is usually less unpleasant for the patient.

When conventional oral and intravenous methods have failed to opacify the ducts and the gallbladder, the dribble technique described by Salzman *et al.*[40] in 1959 may have occasional success in demonstrating a calculus, if this is the cause of the jaundice. They so beautifully demonstrated, that calculi coated with bile pigment may absorb sufficient opaque medium onto their surfaces to render them opaque to roentgen rays, as, for instance, in one of our cases, a

lady of 51 with jaundice of 5 weeks' duration and a serum bilirubin of 1.8 mg. per cent. Plain film roentgenograms of the abdomen and a barium series proved negative. No opacification of the biliary tree was obtained with intravenous biligrafin, but after taking telepaque 1 gm. t.d.s. for 4 days, 3 stones were clearly visible (Fig. 15) in a dilated bile duct. Occasionally, the gallbladder or bile duct will be opacified by this method when others have failed and non-opaque stones may thus be demonstrated as negative shadows in it as in Figure 16, a man of 49 who had had painless jaundice for 5 weeks. A malignant lesion was considered, even though the serum bilirubin was only 2.2 mg. per cent, for the serum phosphatase level was 70 K.A. units and the jaundice seemed to be deepening. Biloptin was administered and after 4 days a faintly opacified gallbladder containing nonopaque stones was demonstrated. No opacification of the stones was achieved, and another nonopacified stone was found in the common duct at operation. Salzman *et al.*[40] claim that approximately 70 per cent of duct stones and 10 per cent of stones in the gallbladder are coated with sufficient biliverdin to be demonstrable if bile concentrations of opaque medium are adequate. In my experience, some 30 per cent of gallbladder stones and 35 per cent of stones in the ducts can be demonstrated provided the serum bilirubin level is below 2–3.0 mg. per cent.



Fig. 15. Dribble telepaque cholecystography in a case of failed oral and intravenous cholecystography. The preliminary roentgenogram was negative. Three stones are shown in the dilated duct after taking telepaque 1 gm. t.d.s. for 4 days.

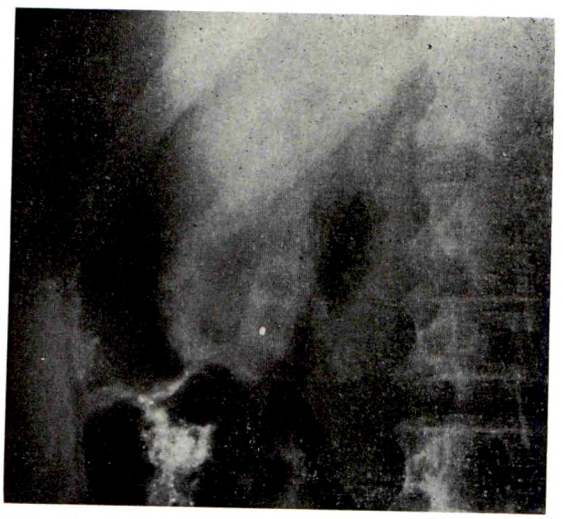


Fig. 16. Dribble biloptin cholecystography. Plain film roentgenography and cholangiography negative. There is a faint filling of the gallbladder containing nonopaque stones after 4 days administration of biloptin.

Angiography[4,24,27,28,30,35,36,39]

Seldinger's technique of arterial catheterization has made it possible to demonstrate the vascular supply of the liver, pancreas and gallbladder with a high degree of accuracy, although that of the bile duct is as yet less easy to determine. It is possible, however, that this will be better achieved by the use of magnification techniques.

The arteriographic diagnosis of a carcinoma, whether of the head of the pancreas or the bile duct, depends on the demonstration of one or more features, such as enlarged feeding arteries; the presence of pathologic tumor vessels or lakes; a tumor blush; displacement, invasion or stenosis of normal arteries or their branches; or by early filling of, displacement, or invasion of veins (Fig. 17).

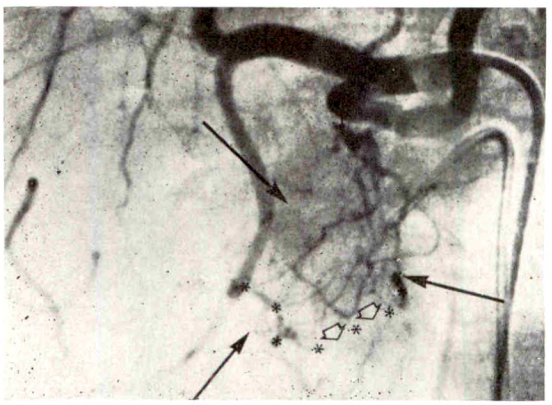


Fig. 17. Carcinoma of the head of pancreas. Combined celiac axis and superior mesenteric arteriogram. Subtraction film shows pathologic vessels (arrows), amputation (open arrowheads), and encasement (asterisks) of normal vessels and tumor stain. (Courtesy of Dr. H. Herlinger.)

Enlargement of the gallbladder can usually be deduced from the degree of displacement of the cystic artery and its main divisions, and, if present, should raise the suspicion of malignant involvement of the distal part of the bile duct. An abrupt angulation or cut-off of the gallbladder vessels should make one consider a growth in the gallbladder. Uneveness in the thickness of the opacified gallbladder wall, as pointed out by Abrams, is an important diagnostic feature, and if present lends support to the diagnosis.

Normal arterial anatomy of the region may be considerably altered if a growth obstructs a major vessel, and the nutrition of important structures or organs such as the liver may be jeopardised if these collateral pathways on which their blood depends, such as arcade vessels of the gastroduodenal artery or the main artery itself in which the direction of flow may be reversed, are resected at surgery together with the duodenum as in the classical Whipple type operation. It is, therefore, of considerable value if the surgeon can be forewarned of this hazard.

A valuable side product of celiac, or hepatic arteriography is the occasional demonstration of unsuspected lesions in the liver such as a hepatoma, intrahepatic bile duct carcinoma, hydatid cysts, or metastases. A hepatoma is usually highly vascular with a distinctive appearance of numerous pathologic tumor vessels and vascular lakes (Fig. 18), although occasionally avascular forms occur. The celiac trunk may be displaced or there may be lack of filling of the other lobe due to preferential filling of the tumor. Normal intrahepatic vessels may be displaced and arterial invasion or a tumor blush can often be shown. Sinusoidal filling is common.

Metastases tend to be more peripherally situated and multiple. They commonly produce an early and sustained blush (Fig. 19), and, depending on size, tend to distort and stretch normal vessels rather than to invade them. Vascular lakes and sinusoidal filling are less common than in hepatoma (Fig. 20).

Avascular forms occur and may be suspected by the demonstration of stretched vessels and negative areas in the hepatogram stages. Unless secondary to a carcinoma of bile duct or head of pancreas, they are usually large and numerous before jaundice is produced.

Multiple lesions may occasionally be due to multifocal liver cell (Fig. 21), or bile duct carcinoma, or hydatid cysts or abscesses.

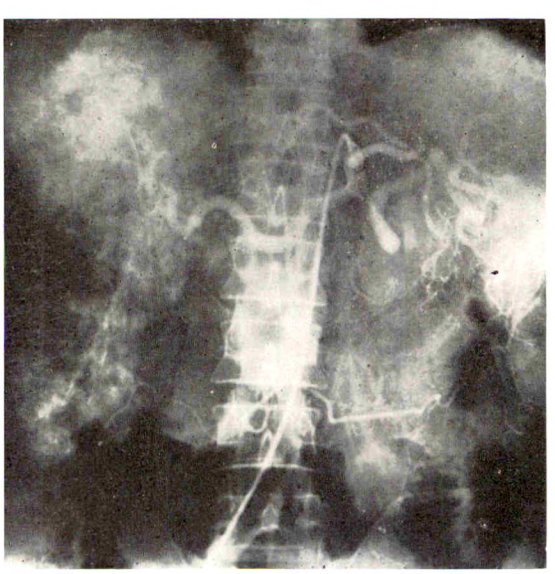


Fig. 18. Hepatoma. Celiac arteriography.

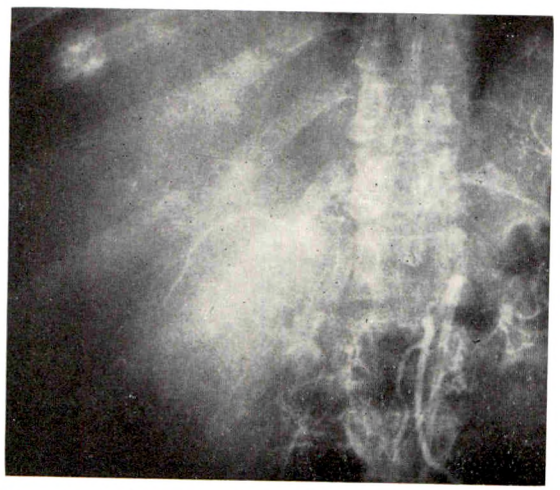


Fig. 19. Celiac axis arteriography. Small blushing metastasis shown during the investigation of a case of jaundice. (Courtesy of Dr. R. Dick.)

Occasionally, intrahepatic systemic-portal shunts in the Budd-Chiari syndrome may mimic the blush of scattered metastatic nodules. A round avascular area with a dense peripheral opacified margin in the hepatogram stage is probably pathognomonic of hydatid cyst (Fig. 22).

Increased vascularity may be present in regenerative nodules in cirrhosis and normal vessels may be displaced or stretched, though not obstructed. Tumor vessels and

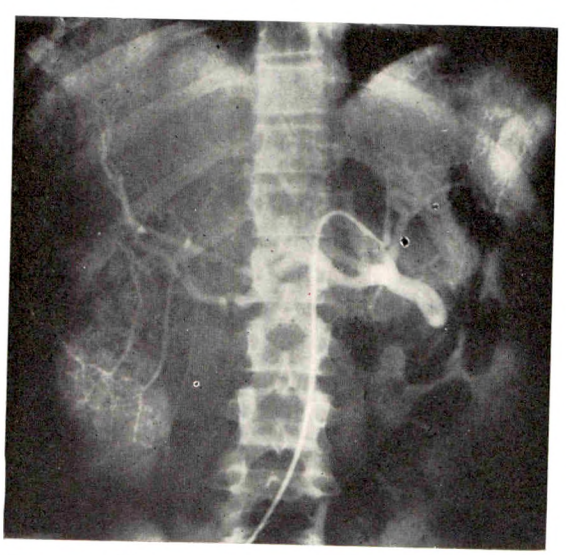


Fig. 20. Celiac axis arteriogram. Metastases (from gastrointestinal tract) are shown in a case with a large liver and jaundice.

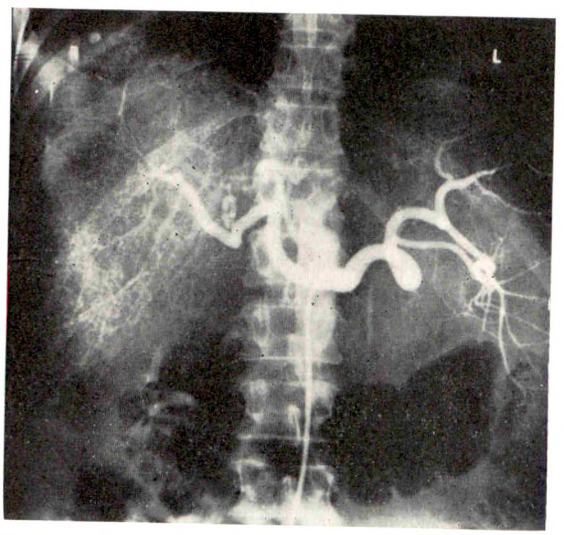


Fig. 21. Celiac axis arteriogram. Multifocal liver cell carcinoma. (Courtesy of Dr. R. Dick.)

vascular lakes are not seen, and the hepatic vascular pattern with collateral venous channels and varices visible in the venous phase is usually characteristic of cirrhosis.

Displacement or obstruction of the

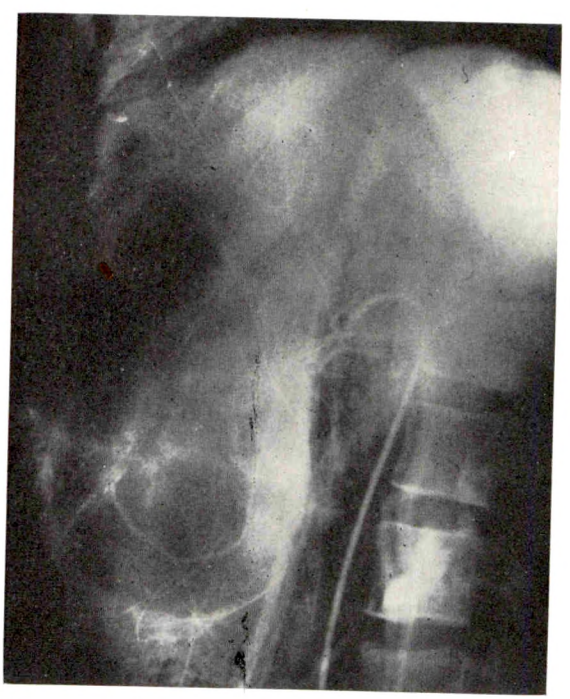


Fig. 22. Celiac axis arteriogram. Hydatid cyst. Typical opacified ring around the periphery of a rounded avascular area in the liver. (Courtesy of Dr. R. Dick.)

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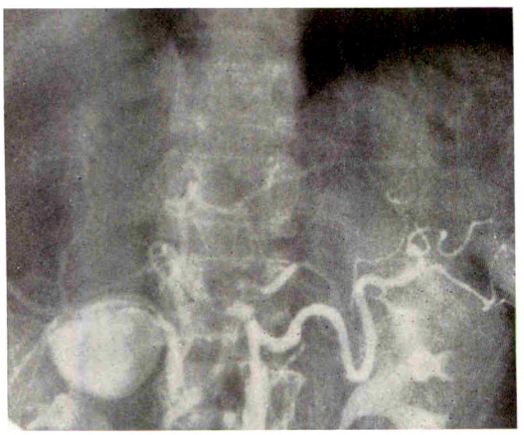


Fig. 23. Celiac axis arteriogram. Aneurysm of the hepatic artery in the region of the porta hepatis compressing and displacing the artery. In this position pressure on the bile duct can be expected.

splenic vein in the later stages of the examination should raise the suspicion of a carcinoma of the pancreas. Dilated intrahepatic bile ducts associated with an extrahepatic obstruction of the bile duct may produce a streaky appearance converging to the hilum during the hepatogram stage. Involvement of the portal vein in any way is a strong indication that a growth is unlikely to be resectable and that palliative surgery or other treatment is the most that can be expected.

If perfusion chemotherapy of the liver is contemplated, preliminary angiography of the celiac axis or hepatic artery is, of course, essential to demonstrate the vessels supplying the growth, and to make sure that the perfusion catheter is properly sited.

Occasionally, some benign condition will be found to be responsible for the jaundice. Aneurysms of the aorta have been known to compress the duct and produce jaundice. In this case an aneurysm of the hepatic artery (Fig. 23) was demonstrated.

It would seem that the best chance to demonstrate a small neoplasm involving the bile duct directly is by a combination of a free flush examination of the celiac axis and superior mesenteric arteries followed by selective catheterization of the gastroduodenal artery or any other vessel which seems to be contributing to the blood supply of the tumor. Subtraction is essential and Herlinger believes that the results are improved still further by tomography, although this requires special apparatus. As in most fields of endeavour, the standard of result depends on the ability of the man carrying it out, although there remain at present certain definite inherent problems which present a challenge to be overcome. For instance, most adenocarcinomas of the biliary tree or of the head of the pancreas are poorly vascularised, and although Herlinger, and Nebesar and his colleagues[27,28] have occasionally demonstrated lesions less than 1 cm. across (Fig. 24), few tumors of less than 2.5 cm. in diameter can be detected with any regularity or certainty even by the most expert arteriographer. Yet by the time these lesions have reached this size many will have passed beyond any hope of radical resection (Fig. 25, *A* and *B*).

Ampullary carcinomas or carcinomas of the bile duct seem to be especially difficult to demonstrate because of the scarcity of abnormal angiographic features.

Intrahepatic bile duct carcinomas are more easily demonstrable than extrahepatic forms, but even these are rarely obvious unless they are more than 2–3 cm. in diameter.

With tumors over 3 cm. in diameter, whatever the site, the success rate improves, being almost proportional to the size of the tumor. But this is the case with



Fig. 24. Celiac axis arterio-tomogram. Subtraction film. Note the small branch of the dorsal pancreatic artery supplying the tumor and the tumor blush (arrows). Early carcinoma of head of pancreas. (Courtesy of Dr. H. Herlinger.)

most of the other radiologic and nonradiologic techniques available and usually is of little help to the patient, although it is of value to the surgeon, who is warned beforehand of what he may expect at laparotomy.

Unless a carcinoma is well vascularised, whatever its size, arteriography is of little help in differentiating between carcinoma and chronic pancreatitis. It is of no value when jaundice is due to stone, benign stricture, or intrahepatic cholestasis.

False positive diagnoses of carcinoma of the head of the pancreas vary inversely with the experience and directly with the enthusiasm of the operator, and probably in experienced and competent hands are in excess of 10 per cent.

A negative arteriogram does not exclude disease of the head of the pancreas whether malignant or inflammatory, although, as Bouchier has pointed out, taken in conjunction with a negative scan the odds against malignancy are high.

Arteriography is reasonably safe and simple in experienced hands, although even so there is a definite morbidity rate, and more than an occasional hazard to a limb or even life in sick or elderly patients, many of whom already suffer from degenerative arterial disease. These factors have to be weighed carefully against any probable advantages that may accrue from a positive arteriogram, and it should therefore be undertaken only when other simpler methods have failed. It is unwarranted in nonmalignant states.

It is, however, the only method whereby the exact size and extent of a carcinoma may be revealed, and may demonstrate a growth still small enough for surgical resection. If it is to be performed at all it should be as soon as possible after the onset of jaundice in any case in which the possibility of carcinoma arises, for the only information of real importance to the surgeon is whether the growth is still limited to the right of the superior mesenteric and portal veins so that a Whipple type operation can be attempted.

Metastases in the liver can usually be shown if they measure 2 cm. in diameter or more, and may sometimes be shown when smaller than this. If none are shown, and the growth seems reasonably small, surgical resection should offer some prospect of cure. If metastases are present some simpler palliative procedure to relieve the jaundice, pruritus, and obstructive vomiting if present, can be undertaken, instead of embarking on an heroic attempt at resection doomed to failure from the start.

Whether the portal vein is involved by the growth or not may make all the difference between an attempt at radical resection or a palliative operation. Strangely enough, radiologists are rarely requested to provide this information. On the assumption better late than never, if preliminary splenovenography has not been performed,

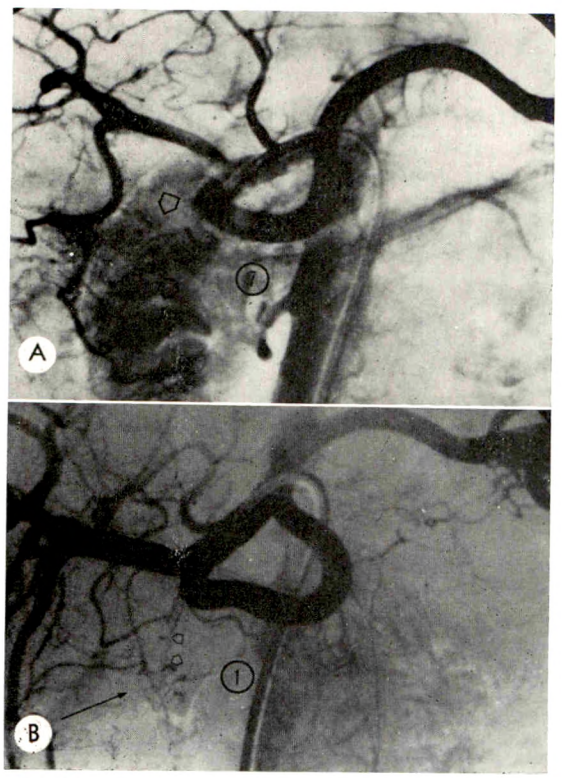


Fig. 25. (*A*) Celiac axis arteriogram. Typical appearances of a large carcinoma of the head of the pancreas (large open arrowheads). (*B*) Arteriogram 1 year previously shows pathologic vessels in a small tumor (small open arrowheads). The patient refused operation. (Courtesy of Dr. H. Herlinger.)

improved radiographic facilities in operating theaters might help the surgeon considerably when faced with a difficult dissection or decision.

GASTRODUODENAL ENDOSCOPIC INTUBATION OF THE BILE DUCTS[8,25,33,46]

I have no personal experience of this technique which was originally developed in Japan, but which is now effectively used in many centers throughout the world. In expert hands, using a special Olympus fiberscope, a catheter can be introduced under direct vision into the mouth of the bile or pancreatic duct and opaque medium injected into them under screen control. It is unlikely to become an universal routine every day type of examination, for it is very time consuming in unpracticed hands and the success rate is low to start with, although 2 or 3 cases can be examined in an hour by an expert when all goes well.

If the bile duct can be intubated, opaque medium is injected to show the site, extent and cause of the obstruction. When jaundice is due to pathology in the head of the pancreas, it may be possible to demonstrate this by injecting opaque medium into the pancreatic duct.

Its main limitation would seem to be that it cannot demonstrate the duct proximal to a complete obstruction; yet this is what the surgeon is usually most interested in, for he wants to know how much is available for anastomosis; nor can it demonstrate the ducts after a choledochojejunostomy has been performed.

PERCUTANEOUS CHOLANGIOGRAPHY[1,6,9,10,15,16,18,32,45–50,53]

At times, after all clinical, laboratory and radiologic investigations have been carried out, including organ scanning, arteriography and liver biopsy, doubt may still remain as to whether jaundice is due to intrahepatic cholestasis, such as in primary biliary cirrhosis; or following the taking of drugs such as chlorpromazine; or to mechanical obstruction of the ducts. Yet, it is essential to know, for the treatment and prognosis are very different. At this stage surgical exploration is commonly employed to exclude or alleviate a mechanical obstruction, should one be present, either by removing or bypassing it. Laparotomy, however, may prove hazardous for patients suffering from intrahepatic cholestasis because of hepatocellular decompensation and an anesthetic may be sufficient to precipitate acute liver cell failure and death, so that surgery is to be avoided, if possible, in this type of case.

Even when there is no doubt that jaundice is due to mechanical obstruction, it is obviously of help to the surgeon to know with certainty, before making his incision, whether it is due to a stricture, benign or malignant; or to stone; or to pressure on the ducts from without. Perhaps it is even more important to know the exact site and extent of the obstruction, the amount of normal duct above and below available for reconstruction or anastomosis to bowel should this be necessary, and the extent to which normal anatomy is distorted, be it due to some developmental anomaly or pathologic process.

When a duct has been mechanically obstructed for any length of time the ducts above the obstruction are usually dilated. The extent to which they dilate seems to depend on a number of factors such as the degree, duration and site of the obstruction. There are exceptions, such as in the case of acute or subacute obstruction of the bile duct due to a calculus, when the common duct may for a time look normal in caliber; or in the presence of chronic cholangitis or jaundice associated with cirrhosis of the liver, when the liver is hard and contracted and there is little if any dilatation of the intrahepatic ducts.

When jaundice is due to intrahepatic cholestasis the ducts seem to be somewhat sparse with diminished caliber, whereas in cholangitis ductal caliber is often irregular with beading.

If, therefore, the duct system can be opacified, the various types of jaundice can usually be differentiated with a fair degree

of accuracy, and the site and usually the extent and often the cause of an obstruction can be shown.

The proximal extent of an obstruction is always best demonstrated from the liver side, whereas the lower end may be better shown from below. In my experience, it is easier to show an incomplete obstruction from above—probably because the patient is examined in an X-Ray Department on a tilting table, under screen control, usually with image intensification, rather than in the more primitive conditions pertaining in an operating theater, for if the duct system can be intubated it is usually possible to show the whole extent of a stricture unless it is complete; and should this be so its lower extent can be demonstrated subsequently by cholangiography when the patient comes to operation.

At the Royal Free Hospital, London, we perform percutaneous cholangiography in the following circumstances:

1. To differentiate between intrahepatic cholestasis and obstructive jaundice when clinical examination, conventional radiology, liver biopsy and biochemical tests have proved inconclusive.
2. In known obstructive jaundice when information is required as to the cause of the obstruction and its site and extent.
3. In all cases of suspected or known traumatic stricture of the ducts prior to surgical repair.

We undertake the examination only as a preoperative procedure to be followed by laparotomy either immediately or within a few hours. The skin is, therefore, prepared for major surgery and the patient is premedicated in the ward before coming to the X-Ray Department, arrangements having been made to proceed to operation if a mechanical obstruction is demonstrated.

In the majority of cases, especially when the case may have to be fitted into an operation list in progress, local anesthetic is employed, although a general anesthetic is given to children.

The prothrombin test and platelet count are checked beforehand and corrected if necessary.

In cases of known obstruction due to stricture, and in all cases where cholelithiasis is suspected, the chances that the bile is infected are over 60 per cent. Antibiotics are therefore administered shortly before the examination and continued at least until the results of the culture of aspirated bile are known.

A 20 gauge needle with a polythene sheath (size p.e. 180) threaded over it is introduced through the skin into the liver, a small incision in the skin and a track through the soft tissues having first been made with a knife blade and a fine pair of forceps. The needle is used only as an introducer and is withdrawn immediately the puncture is made. The polythene tube is then connected to a syringe containing saline by way of a Seldinger type tap adapter—the saline being used to flush it through periodically. Suction is applied while the tube is slowly withdrawn. Once a duct is entered, bile is aspirated and can be easily recognized by its color and consistency. The tube is then fastened to the skin by adhesive tape.

When the duct system is dilated an intrahepatic duct is usually easily found, but it is advisable to make 5 or 6 punctures before assuming that the intrahepatic ducts are not dilated and giving up. In cases of suspected traumatic stricture, especially when one or more previous attempts at repair have been made, it is so important for the surgeon to know the anatomy of the duct system he is being called on to repair that as many as 10 or 12 punctures can be made using both the anterior and lateral intercostal routes, and finally using the needle alone without the polythene sheath, as this enables smaller ducts to be found. The patient seems to suffer no additional hardship by this practice.

A little bile is aspirated and sent for culture and a syringe containing the opaque medium is then attached to the tube using a medium length polythene cistern (we use

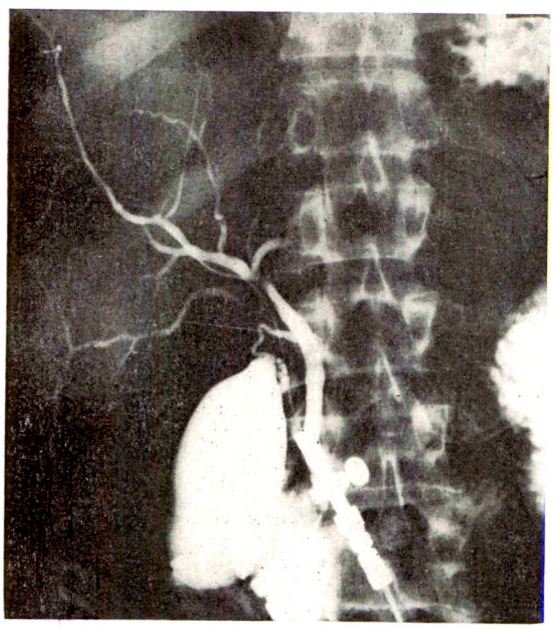


Fig. 26. Primary biliary cirrhosis. Percutaneous cholangiography. Spidery intrahepatic ducts with sparse branches. The ducts have smooth outlines and taper normally.

35 per cent hypaque). The duct system is then gradually filled, the amount injected depending on the degree of dilatation of the duct system. Spot films are exposed in those positions thought best to show any lesion present, and as a routine we take 30×25 cm. supine, supine oblique, erect, lateral and prone views as well. A lateral decubitus roentgenogram using a horizontal beam is advocated by Jacobson to separate the main divisions of the right hepatic duct.

Posterior and lateral intercostal approaches to the liver have been recommended by a number of different workers, the former to achieve an extraperitoneal approach and the latter to lessen the risk of creating a bile venous fistula. Others inject the gallbladder or insert the needle between the layers of the falciform ligament during peritoneoscopy. Some recommend the use of polythene or teflon tubing, while others use a flexible needle alone. I prefer an anterior right subcostal approach inserting the needle at a point shown to overlie the tip of the 2nd lumbar transverse process on the preliminary roentgenogram. From here the needle is initially directed slightly cephalically along a line to the right shoulder. If unsuccessful, further injections are made with the needle fanned to the right or left. I prefer the anterior approach because it enables either the right or left duct system to be injected separately or simultaneously when one wants to know whether a high obstruction involves one or both intrahepatic ducts, whereas it is impossible to inject the left duct system by the right intercostal route. An additional advantage is that the puncture sites are easily visible and accessible at laparotomy should any leakage or hemorrhage complicate the procedure. However, if the anterior route proves unsuccessful, I usually make 2 or 3 additional attempts to intubate a duct, using the right lateral intercostal route.

The demonstration of normal ducts, or ducts of decreased caliber, with free flow of the opaque medium into the duodenum indicates that the jaundice is not due to large duct obstruction.

Figure 26 is a roentgenogram taken of a lady aged 37, who had been jaundiced for 6 weeks, in whom laboratory tests and liver biopsy were equivocal, and in whom it was clinically impossible to decide between intrahepatic cholestasis or extrahepatic duct obstruction. The roentgenologic appearances were typical of primary biliary cirrhosis and as a result surgery was cancelled and she was returned to the ward, being spared an operation that might have done her harm. There were no complications. Figure 27 shows the ducts of a man of 45 years of age, with a history of recurrent attacks of jaundice and fever over the past 2 years. Because of the absence of stones a diagnosis of idiopathic sclerosing cholangitis was made and he was spared an unnecessary operation.

It is usually possible from the roentgenologic appearances and the history to diagnose with a high degree of accuracy the cause of any obstruction shown. For instance, an obstructing stone classically

shows a convex or meniscus like cut-off (Fig. 28), although this may be mimicked by a benign polyp. A malignant stricture usually presents a tapered irregular, or finger like termination (Fig. 29), often with a small protruding nipple (Fig. 30). Traumatic strictures are usually fairly short. If incomplete, the duct tends to taper smoothly to the stricture, but the narrowing may be abrupt. If the obstruction is complete the distended proximal duct may look identical to a malignant obstruction, so that the history is of some importance. Chronic pancreatitis always affects the infraduodenal segment of the duct, which tends to be straightened and its normal convexity to the left is lost (Fig. 31). As a rule the duct is fairly uniformly narrowed as it passes through the pancreas, although there may be a smooth unevenness of its caliber, with only slight proximal dilatation of the proximal duct system unless narrowing is pronounced. Complete obstruction is rare and in fact jaundice is not a very common presentation.

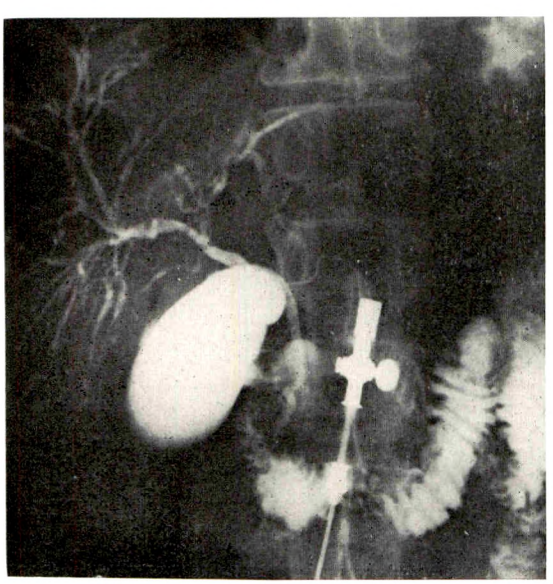


Fig. 27. Idiopathic sclerosing cholangitis. Percutaneous cholangiography. The pattern of the intrahepatic ducts is distorted. Note the irregular caliber of the intrahepatic ducts due to local narrowings and dilatations. There is evidence of calculus disease.

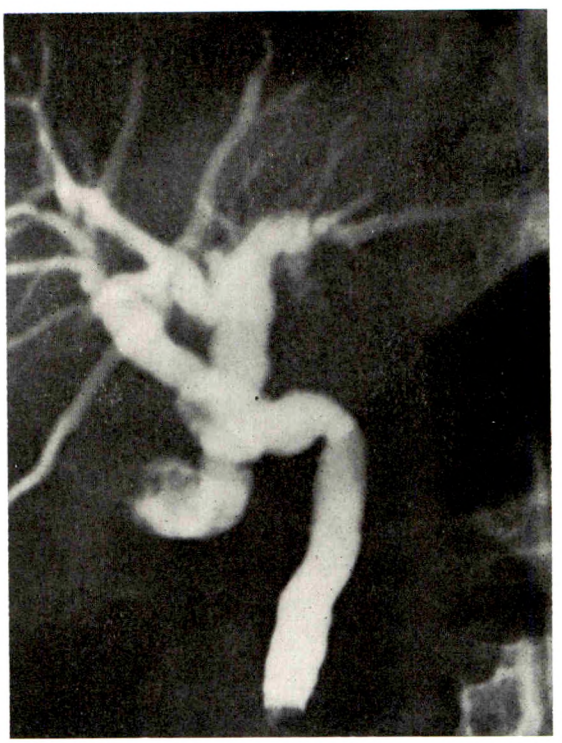


Fig. 28. Calculus disease. Percutaneous cholangiography shows stone obstructing the lower end of the common duct. Typical meniscus. Stones are also present in the shrunken gallbladder.

ANATOMIC VARIATIONS AND SURGICAL CONSIDERATIONS

In some 20 per cent of cases the anatomy of the duct system varies from normal, and it is of value to the surgeon to be forewarned of this, for it will reduce the chance of accidental damage to the bile duct. Occasionally, jaundice may be associated with choledochal cyst. More often the duct anomaly takes the form of a long cystic duct implanted low into the common duct, which may itself be diseased or harbor a stone. Less often there is an abnormal situation of the papilla, for the bile duct may enter the duodenum anywhere from the first to the middle of the third part of the duodenum. Thorek states that in 10 per cent of individuals the cystic duct drains directly into the right hepatic duct; and in 3–5 per cent there is a connection between the dorso-caudal division of the right hepatic

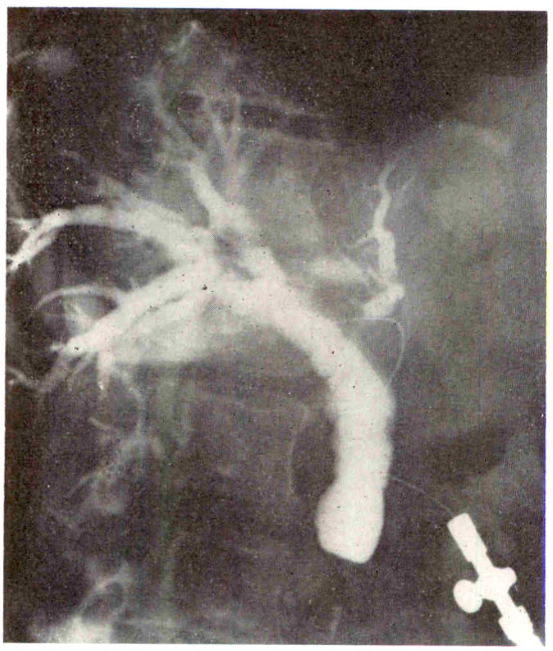


Fig. 29. Carcinoma of the ampulla. Percutaneous cholangiography shows finger end appearance of complete obstruction. There was a corresponding pressure defect on the concavity of the barium filled duodenum.

duct and the gallbladder or cystic duct, both situations in which there is a real risk of injury.

Percutaneous cholangiography has certain inherent advantages over cholangiography performed at operation. False positive diagnoses are almost unknown, and difficulties arising from confusing shadows such as air bubbles do not arise if a proper technique is followed. Because we perform the examination in the X-Ray Department on a tilting table with image intensification and compression spot roentgenograms, detail obtained is far superior. The films are examined by a radiologist in a good light and discussed calmly and objectively with the surgeon instead of, as is usually the case, being examined by the surgeon alone, often under poor viewing conditions. The surgeon can plan his operation and approach before making his incision knowing what he can expect to find, or later can change his list to suit the circumstances, for the removal of a duct calculus will take less time than a Whipple type resection.

A stricture in the bile duct can be better shown from above than below, for the transition from a distended duct to a narrowed one seems to make more impact than the reverse, and distention is achieved less readily from below for the opaque medium tends to flow preferentially into the duodenum unless it is injected under pressure, or the duct below the site of injection is gently occluded. If the stricture is complete, and especially if due to previous surgery, it is impossible to show its full extent from below even if it is possible to find the lower duct remnant because of surrounding fibrosis, etc. In any case the surgeon is more concerned to know the amount and condition of the duct proximal to an obstruction available for anastomosis to bowel, and where to look for it. The demonstration of a patent or obstructed cystic duct when obstruction is due to a

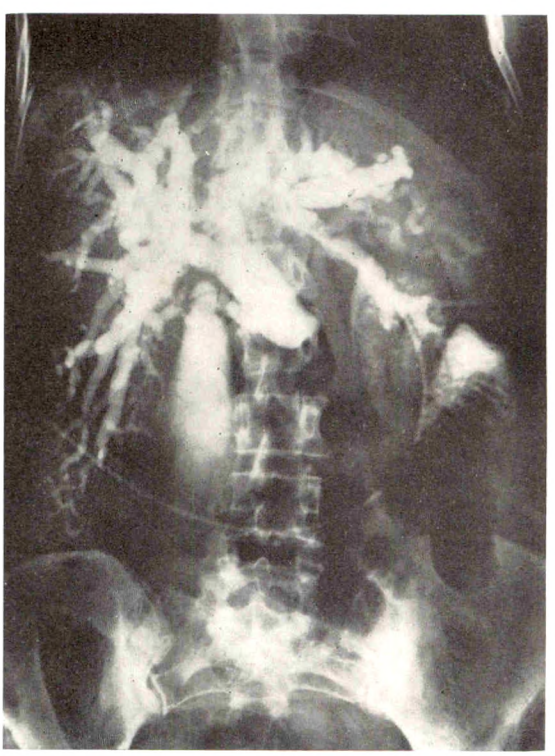


Fig. 30. Carcinoma of the head of the pancreas. Percutaneous cholangiography shows complete obstruction of the common bile duct with distention of the gallbladder and of the proximal duct system.

carcinoma may, in itself, be an important observation, for this will often decide whether the gallbladder can be used for anastomosis or not.

The following cases illustrate some of the points made.

REPORT OF CASES

Case 1 (Fig. 32). A lady aged 35 developed jaundice some 8 months after cholecystectomy for calculi. Cholangiography had not been performed at operation and the ducts were not explored. It was thought likely that a stone had been left behind in the bile duct, but jaundice was increasing, and another cause could not be excluded. Percutaneous cholangiography was requested, and a short incomplete stricture—almost certainly traumatic—was demonstrated with biliary debris above it. It also informed the surgeon than an end-to-end anastomosis might be feasible.

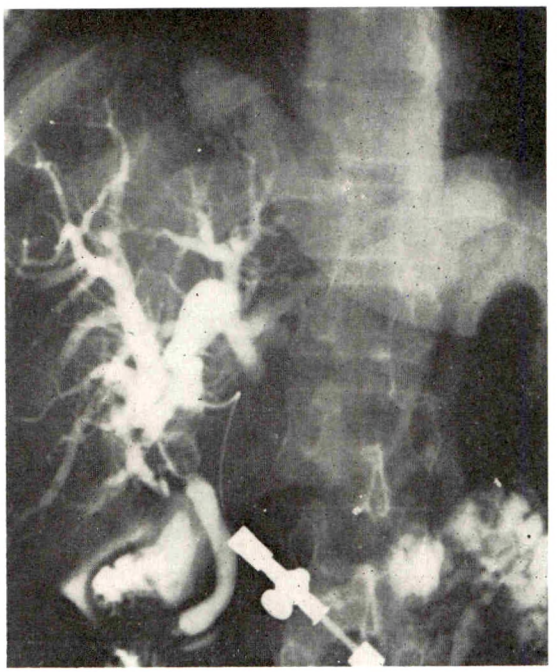


Fig. 32. Case 1. Percutaneous cholangiography. Short traumatic stricture of the bile duct (post-operative). Biliary sludge and small stones are shown above the stricture.

Case 11 (Fig. 33). A middle-aged lady developed severe and increasing jaundice over a period of 3–4 months following cholecystectomy for stones. Operative cholangiography had not been performed and the ducts had not been explored. The probable diagnosis lay between a residual stone or traumatic stricture. Percutaneous cholangiography revealed a surprisingly high complete stricture, and the surgeon was warned that a high anastomosis of the type described by Langmire or Rodney

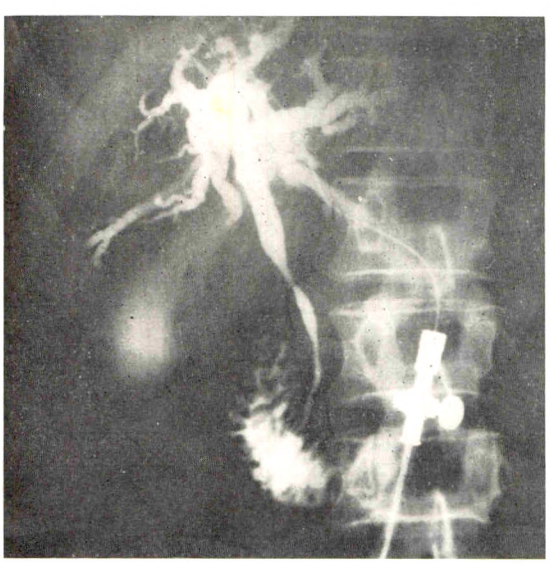


Fig. 31. Chronic pancreatitis. Percutaneous cholangiography. There is typical gradual narrowing of the pancreatic segment of the bile duct with straightening of the duct and proximal dilatation.

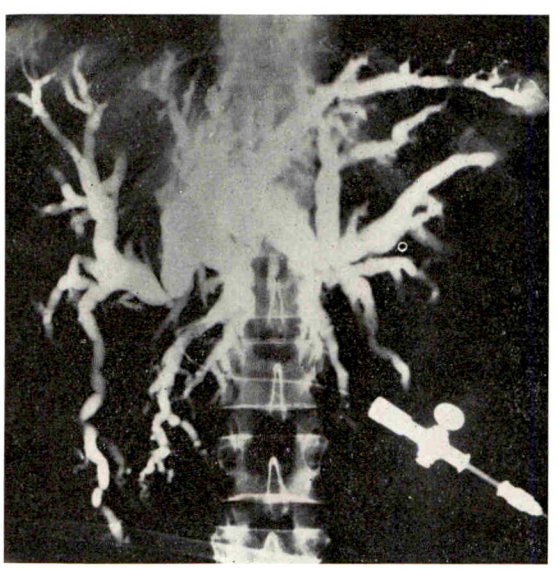


Fig. 33. Case 11. High traumatic stricture of the duct following cholecystectomy. Percutaneous cholangiography shows involvement of the origins of both main intrahepatic ducts.

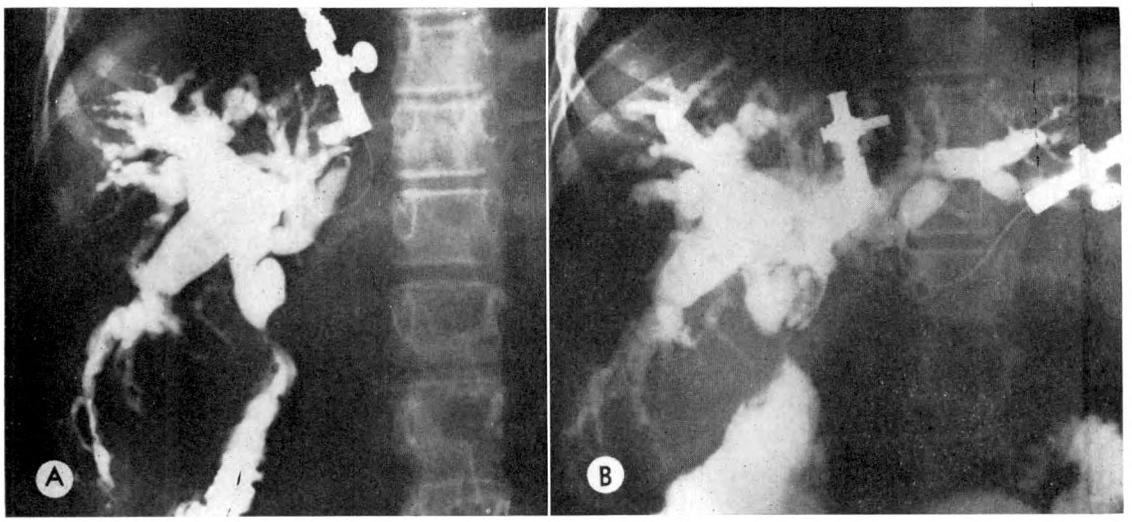


Fig. 34. Case III. (*A*) Operative traumatic stricture. Choledochojejunostomy performed 18 months previously to relieve jaundice. Percutaneous cholangiogram shows only the right hepatic duct to be filled. There is a narrow opening communicating with the jejunum. (*B*) Second puncture fills the left hepatic duct. There is marked stenosis of the anastomosis with considerable dilatation of both intrahepatic ducts.

Smith would be required, so that he was able to plan accordingly.

Case III (Fig. 34, *A* and *B*). The patient, a lady of 49, complained of increasing jaundice with attacks of fever. An attempt had been made to relieve an operative stricture 8 months previously, but details were lacking. Percutaneous cholangiography was undertaken and only the right intrahepatic duct system filled. A second puncture was therefore made into the left lobe of the liver and the left duct system opacified. It became apparent that a high anastomosis had been performed between jejunum and the confluence of the hepatic ducts and that this had become stenosed. As a result, and only after a difficult dissection, the anastomosis was reconstructed and the patient has remained symptom-free since.

After a choledochojejunostomy has been performed there is, of course, no other method of satisfactorily demonstrating the state of the anastomosis before surgery.

Case IV (Fig. 35). A man aged 34, recently returned from the Far East, developed jaundice which was thought to be hepatocellular in origin and was treated as such. Liver function tests and biopsy were equivocal. The jaundice deepened in spite of treatment. Percutaneous cholangiography showed the typical appearances of a carcinoma of the bile duct. Because of its position, it seemed unlikely to be completely resectable and this proved to be so. The proximal ducts were anastomosed to intestine.

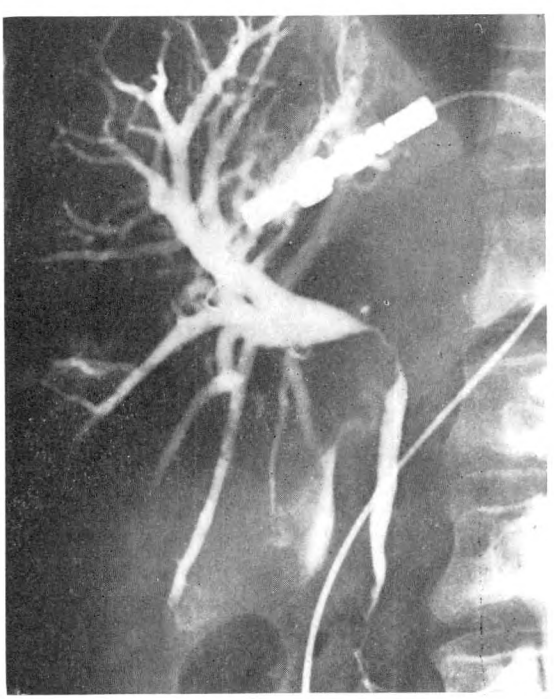


Fig. 35. Case IV. Carcinoma of the bile duct. Percutaneous cholangiography demonstrates tapered stricture above the cystic duct close to the confluence of the hepatic ducts.

Case v (Fig. 36). A lady of 65 with painless deepening jaundice of varying intensity over 6 weeks with loss of weight was thought likely to be a case of carcinoma of the head of the pancreas. Percutaneous cholangiography showed a large stone ulcerating into and partly destroying the bile duct, with a fistula into the duodenum. The stone was removed and a choledochojejunostomy performed. She made a good recovery.

Case vi (Fig. 37, *A* and *B*). This man of 42 had had an operation for jaundice several years previously at another hospital. Jaundice had recently returned and was getting worse. There were no notes or operative details available, although he said that he had been told he had a cyst of his bile duct. Percutaneous cholangiography was performed. On the first puncture (*A*) the right duct system only was demonstrated, and showed the right hepatic duct draining into a sac-like structure. A second puncture was then made into the left duct system (*B*). This was shown also to communicate with the sac which now had a convex lower end, thought to be due to biliary mud, probably obstructing the anastomosis which was assumed to have been performed between a choledochal cyst and the jejunum. After a difficult dissection, the sac, which lay partly behind the head of the pancreas, was found half-filled with stones and biliary mud obstructing the previous anastomosis to jejunum. The stones and biliary debris were emptied from the sac and a fresh anastomosis to bowel was achieved. The patient made a good recovery. Without a clear indication of the anatomy the surgeon would have probably found it impossible to sort out the situation.

Case vii (Fig. 38). An Arab boy aged 15 was admitted with a history of having been jaundiced since early childhood. He was thin and emaciated and deeply jaundiced. After laparotomy 6 months previously the parents had been told that there were enlarged glands compressing the bile duct, and that the boy was suffering from some form of malignant reticulosis. Percutaneous cholangiography showed a high complete stricture of the common hepatic duct. Because of the history it was suggested that this was likely to be either traumatic or congenital in origin. At laparotomy the surgeon found the bile duct to be very small, although no other abnormality could be found on inspection and palpation. Because of the roentgenographic findings, the duct was opened and a probe passed up it. A resistance was felt which suddenly gave way, to be followed by a gush of white bile. It was assumed that the obstruction was due to a congenital diaphragm, for a cholangiogram obtained before closing the abdomen showed the opaque medium to flow freely into the hepatic ducts. The jaundice rapidly cleared and the boy started to put on weight, eventually making a full recovery.

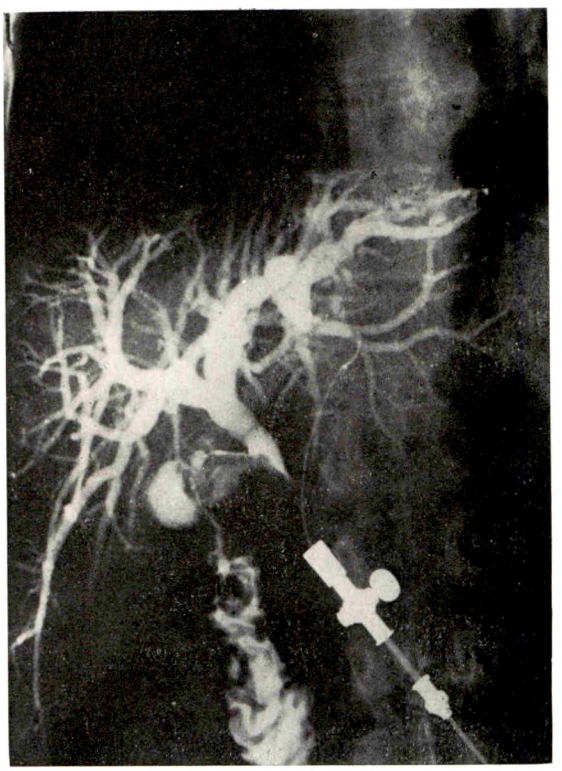


Fig. 36. Case v. Percutaneous cholangiography shows a large stone ulcerating through neck of gallbladder into common duct. Fistula into duodenum.

Case viii (Fig. 39, *A* and *B*). A lady of 50 was referred with increasing painless jaundice over a period of 6 weeks. She had an attack of jaundice with pain 8 months before which was thought to be due to the passage of a gallstone. A barium meal examination on admission showed a filling defect in the barium filled duodenum and it was thought that this was probably due to a dilated duct containing stones (*A*). Percutaneous cholangiography showed numerous stones in the common bile duct (*B*), but on close examination it was apparent that there was a proximal carcinoma of the bile duct.

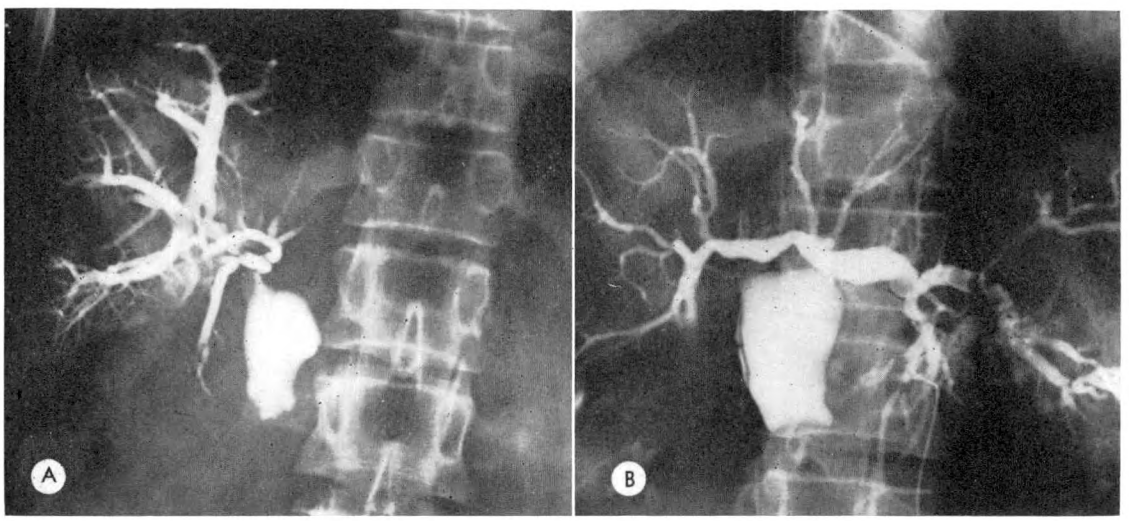


FIG. 37. Case VI. Choledochal cyst. Previous anastomosis of cyst to bowel. (*A*) Percutaneous cholangiogram. First puncture shows right duct system only opacified. (*B*) Second puncture. Left duct system opacified. Both ducts are shown to communicate with a sac-like stricture. No obvious communication with the small intestine. Convex lower end of the sac suggests that a large stone is present obstructing the anastomosis.

It was possible to pass a tube through the narrowed segment and the patient survived for nearly 4 years.

CASE IX (Fig. 40, *A* and *B*). Percutaneous cholangiography was carried out in a patient with obstructive jaundice of uncertain origin. The typical appearance of a nonopaque stone obstructing the lower end of the bile duct was shown (*A*). Another film exposed a few minutes later (*B*) showed no evidence of any stone in the duct. This suggested that it had been passed, but the surgeon thought it wiser to operate. At laparotomy no stones were found in the duct, so it would appear that in this case the examination was instrumental in the passage of the stone.

DISCUSSION

When the extrahepatic ducts are patent, the larger subdivisions of the main intrahepatic duct system do not exceed 3 mm. in diameter and their branches are considerably smaller. In our material there is a relatively high percentage of cases suffering from intrahepatic cholestasis in which an unsuccessful examination is to be expected, and we have managed to intubate only some 15–20 per cent of ducts which have subsequently been shown at surgery not to be dilated. Other workers seem to have had greater success, and no doubt a higher percentage success rate could be obtained if a needle of 20–21 gauge is used alone

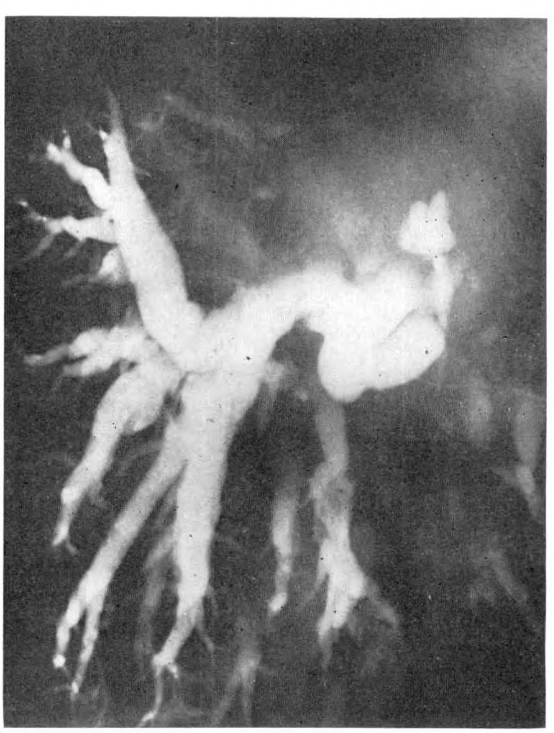


FIG. 38. Case VII. Congenital diaphragm of the bile duct. Percutaneous cholangiography shows complete high duct obstruction.

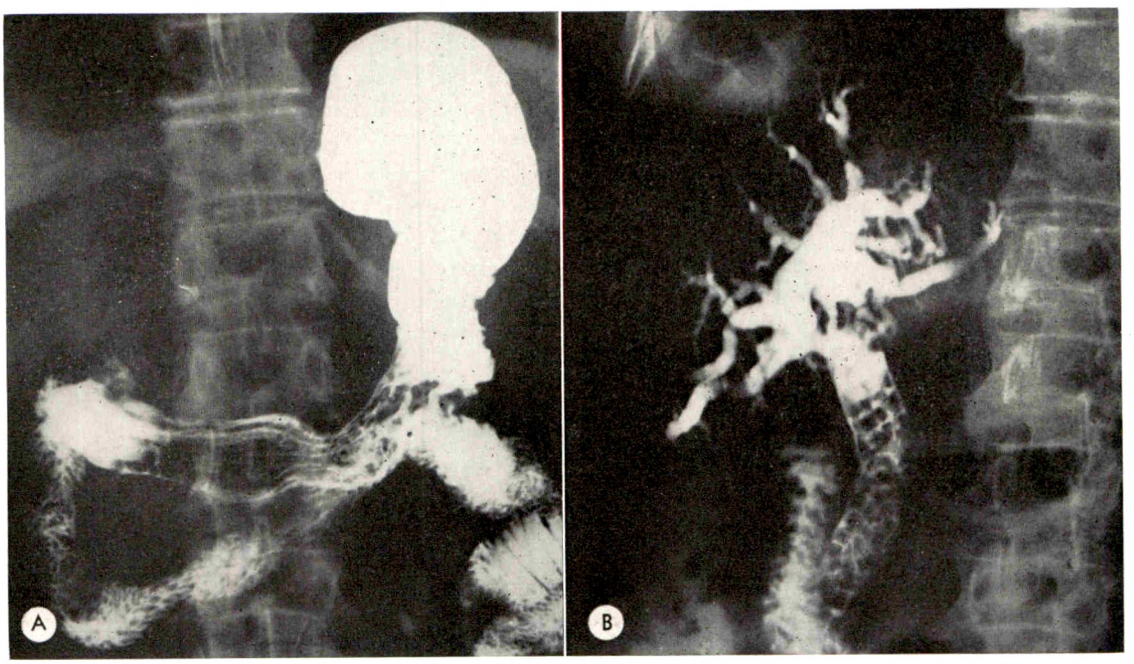


Fig. 39. Case VIII. (*A*) Barium meal examination shows a filling defect on the concavity of the 2nd part of duodenum. Diagnosis of distended duct, probably due to calculi, was made. (*B*) Percutaneous cholangiogram shows bile duct distended with stones. However, note the proximal stricture which is typical of carcinoma of the bile duct.

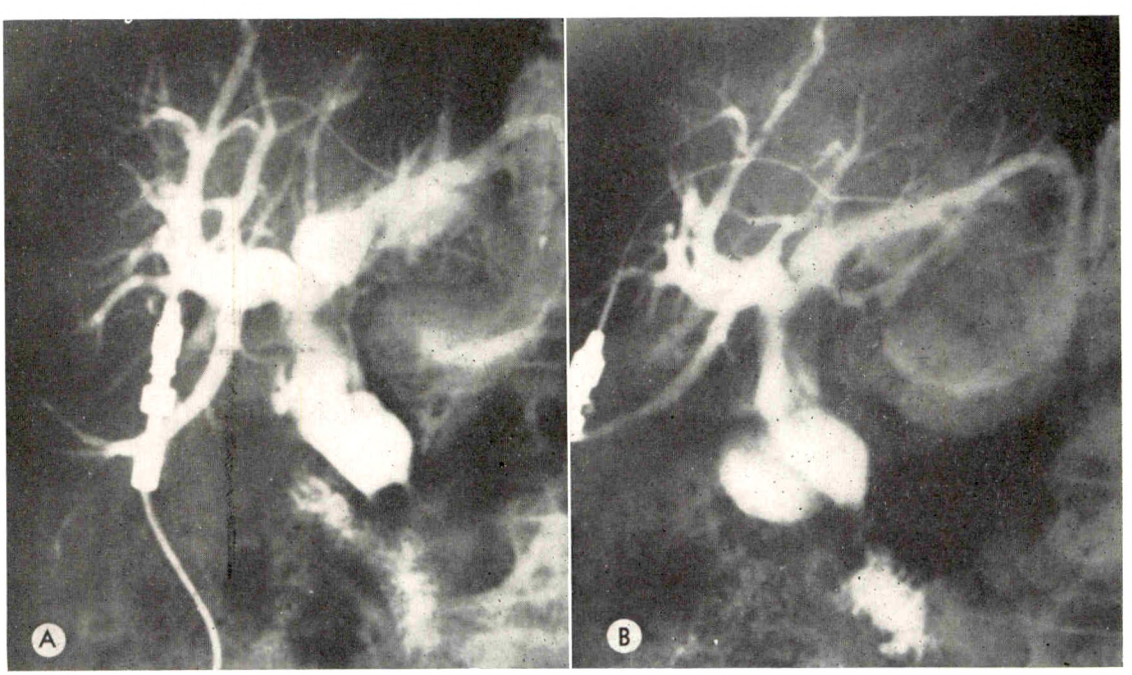


Fig. 40. Case IX. (*A*) Percutaneous cholangiogram shows a stone impacted in the lower end of the common duct. (*B*) Film exposed a few minutes later. No evidence of stone. (Courtesy of Dr. Chapman.)

instead of the type of tube we advocate, although we consider that a tube is less likely to damage the liver than a needle when left in position throughout the examination, for it does not restrict movement of the patient or screen in any way, and there is little risk of hepatic damage if he should cough or move suddenly. But even so, failure to find a duct system suggests that it is not significantly dilated and that the jaundice is more likely to be due to some hepatocellular cause or intrahepatic cholestasis than extrahepatic mechanical obstruction. This assumption is, however, not infallible, for in adults the intrahepatic ducts may occasionally be narrowed by a diffuse infiltrative carcinoma, or as a result of sclerosing cholangitis, with or without stones; and at other times surprisingly large ducts may defy attempts to demonstrate them. Intrahepatic ducts may not, or be only slightly dilated above a partial postoperative stricture, or when jaundice is due to calculus disease, even though the extrahepatic ducts are increased in diameter, sometimes to quite a marked extent. Our failures have been greatest in cases of partial postoperative stricture, in which our rate of successful intubation has been approximately 75 per cent, and of intermittent mechanical obstruction of the duct by stone, especially when complicated by proximal cholangitis, in which our results have been only a little better. On the other hand, we have had very few failures, less than 3 per cent, in demonstrating a malignant obstruction when one existed and most of these have been due either to patient intolerance of the procedure or operator inexperience, although one must admit that at times one can hardly believe one's failure to intubate ducts shown to be so grossly dilated at surgery.

Success rates and failure rates quoted in different series are uninformative if a mechanical obstruction is subsequently not proven by surgery or necropsy. Figures showing over-all success rates are also of little value, for they will vary according to the proportion of large duct obstruction to intrahepatic cholestasis in the series. Even when applied to cases of known duct obstruction, success will vary according to the type and site of the obstruction, the degree of impedance to the flow of bile and the experience or fortune of the operator, and what is considered to be adequate filling of the ducts. Success rate should therefore be measured according to the proportion of correct diagnoses in different pathologically proven conditions if it is to mean anything, for tabulation of over-all results on a percentage basis can be almost meaningless.

I believe that in the interests of the patient the examination should be essentially a preoperative one as the result of consultation between physician, surgeon and radiologist. It has no place in the investigation of jaundice in a patient too ill or too old to undergo a simple bypass or drainage procedure should the need arise, unless, in the rare instance, it is performed as a deliberate drainage procedure to relieve intractable pruritus. We have had only modified success in 2 cases in which we have intubated for this purpose, for it is difficult to maintain satisfactory drainage, although it should not be too difficult to insert another larger tube into one of the larger ducts under direct visual control once the duct system has been opacified if a definitive drainage procedure is being attempted. It should be undertaken only in special circumstances if there is clear evidence of intrahepatic sepsis, and it is contraindicated if there is a likelihood of hydatid disease.

It has been stated that percutaneous cholangiography is a dangerous procedure because of the risk of peritonitis due to leakage of bile, and in the past a number of deaths were reported from this cause; as a result the technique fell into disrepute. I believe this to be unjustified, and if the examination is performed as an immediate preoperative procedure the risk of peritonitis is negligible. Normally, bile is sterile and, following spontaneous or traumatic rupture of a major duct, a large quantity may accumulate in the peritoneal

cavity for some weeks without producing anything much in the way of symptoms. In animals ligature of the ducts is usually followed shortly by infection, although this does not seem to follow invariably in man. Bile may become infected after anastomosis of the duct to intestine or after sphincterotomy and is infected in about 60 per cent of cases suffering from chronic stones in the bile duct.

When ducts have been shown to be distended and the intraductal pressure is raised—this can be gauged by the speed of backflow into the tube once a duct is intubated, or more accurately by taking pressure recordings before injecting the opaque medium—there is obviously a risk of leakage between the time of the examination and the opening of the abdomen. For that reason we always leave the tube in place to drain the ducts until the surgeon is ready to remove it. Once the obstruction has been relieved or bypassed by surgery, bile will tend to flow along normal pathways rather than collapsed needle tracks and there seems to be little risk of subsequent bile peritonitis. This danger is probably further reduced by the fall in intraduct pressure that occurs after any exploratory operation on the bile ducts due to a reduction in the amount of bile secreted by the liver. Any bile or blood that may have leaked into the peritoneal cavity is evacuated during peritoneal toilet before the abdomen is closed and in our experience of over 500 cases we have had no serious case of peritonitis and none of our patients has died or suffered any really serious morbidity as a result.

If it is clear that the duct system is not dilated and there is free flow of opaque medium into the duodenum, the patient can safely be returned to his bed, there being little if any danger of subsequent leakage of bile. If no duct is intubated when the diagnosis between obstructive and cholestatic jaundice is in doubt, we have found it safe also to return the patient to the ward for further observation and treatment on the grounds that the chances are against obstruction. If, however, the patient's condition is reasonably good, to be on the safe side and to make sure of the diagnosis, laparotomy is undertaken as arranged. Operative cholangiography should be mandatory, whenever in a case of jaundice the bile duct is found not to be distended, for it is essential under these circumstances to obtain good filling of the intrahepatic ducts. In a period of 10 years 21 cases with prolonged jaundice were referred to the Royal Free Hospital. All had had 1, and several 2 previous negative laparotomies. In all it had been noted that the common duct was not distended. In spite of this, operative cholangiography was performed only on 5 and, presumably because attention was focused on the lower bile duct and flow into the duodenum, in none did the surgeon appreciate that the proximal ducts had not properly filled. Percutaneous cholangiography performed shortly after admission revealed a high duct carcinoma in every one. Clemmett has reported an almost identical experience in the United States.

In my opinion the risk of septicemia is greater than that of bile peritonitis and may result by direct transference of organisms in infected bile to the blood stream during the insertion or withdrawal of the tube; or as a result of the formation of small traumatic bile-venous fistulas; or simply as a result of backflow into the venous system if the duct system is not decompressed or if opaque medium is injected too forcibly. If a pyelogram is obtained during what otherwise seems to be a clean intubation of a duct, it can reasonably be assumed that a communication with the blood stream has been opened up and suitable measures taken if, as a result of the examination, it is thought likely that the bile is infected.

It is our practice always to aspirate some bile for culture before injecting the opaque medium. In all cases of known or suspected chronic cholelithiasis or sclerosing cholangitis, or a duct stricture or duct anastomosis to bowel, we administer antibiotics immediately before the procedure and con-

tinue until the sensitivity of any organisms cultured is known. Gentamycin, Kanamycin or Colistin are probably the most effective drugs to deal with the organisms usually found, such as *E. coli*, *B. proteus*, *Klebsiella*, etc., and Ampicillin is possibly the next best. Rifomycin and Tetracycline which are normally excreted in relatively high concentration in bile are not so effective in jaundice because of impaired liver excretion. In carcinoma of the head of the pancreas or primary carcinoma of the bile ducts, the bile is rarely infected, although it may be at times when a carcinoma involves the ampullary region.

Occasional deaths have been reported from hemorrhage. We have had no serious problems with bleeding, although it has been quite severe in 2 cases, both suffering from multiple metastases in the liver. In both cases it was easily controlled by the surgeon once he opened the abdomen. This is, of course, an added reason for performing the procedure as close to surgery as possible, and for the use of an anterior approach which is more accessible than a lateral one. As a precaution we never undertake the procedure if the patient has any serious blood coagulation defect, and the clotting time is always checked beforehand. Vitamin K is always administered to any case of obstructive jaundice. It is not uncommon for the surgeon to find some blood clot in the bile duct if he opens it. This is usually easily extractable and the duct is flushed through with saline. We have never encountered significant postoperative duct obstruction due to clot. Margulis[24] has described an ingenious method of irrigating the duct system and dispersing the clot if a T-tube has been left in the duct by using a catheter inserted over a Seldinger type guide wire, but so far we have not had reason to try this.

Percutaneous cholangiography has proved to be of such value to my surgical colleagues, and it is considered so safe, that it has now become almost a routine preoperative examination in any cases of persistent or increasing jaundice lasting more than 3–4 weeks, especially when the cause or site of the obstruction is in doubt. When successful, it provides a map of duct anatomy and pathology from which the surgeon can determine his procedure in advance, and to which he can refer during his operation, instead of embarking on a blind adventure in a field where normal anatomy may be considerably distorted by previous surgery, adhesions or malignant infiltration. This is particularly the case when an operation is undertaken to repair damage to the bile duct caused by previous surgery where the search for a duct remnant by blind dissection may prove extremely anxious, frustrating and, at times, hazardous. This aspect of percutaneous cholangiography has frequently been emphasized by my surgical colleagues, who, once they have experienced the help it can give, stress the confidence and moral boost it provides at times when faced with what looks like an almost impossible task, for they are encouraged to persevere, knowing with certainty that somewhere there is a duct available for anastomosis and where to look for it. Operation and anesthetic times are shortened, not only directly, but because it obviates the need for operative cholangiography with its occasional attendant frustrations, so that the total benefit to the patient is considerable.

CONCLUSION

At present, just as in a traffic light the yellow is a precursor of red, the yellow of jaundice should always be regarded as a warning of danger. When it is due to malignant disease, by the time it becomes apparent, or the reason for it is discovered, the growth is usually so large as to be beyond reasonable hope of radical excision and diagnosis is tantamount to the confirmation of a sentence of death.

In certain situations, however, such as when jaundice is due to a carcinoma of ampulla or the common bile duct, or a carcinoma of the head of the pancreas arising in the immediate vicinity of the duct, it may be the sign of a growth small enough

to allow a surgeon to resect it completely if only the warning is heeded and the diagnosis is made in time; and the patient will then have a chance of survival.

A delay in diagnosis of some weeks probably does not matter when jaundice is due to stone or to a large carcinoma of the head of the pancreas, which is already inoperable; or malignant disease of the liver; and most forms of hepatocellular and intrahepatic cholestatic jaundice will run their natural courses more or less regardless of how they are treated. But jaundice, especially in the middle-aged and over, should be considered a surgical emergency until proved otherwise, and the medical profession and the public require to be educated to this way of thinking.

Small growths, wherever situated, cannot be demonstrated by radionuclide scanning, and only rarely by hypotonic duodenography. They may be found by selective arteriography, but a small carcinoma, unless it causes jaundice, is unlikely to produce symptoms sufficiently severe to bring it to notice, so that arteriography rarely has an opportunity to show its true worth, especially since carcinomas of the bile duct and ampulla do not tend to produce specific changes until quite late. Surgical exploration with cholangiography is not entirely free from risk, when jaundice is due to liver cell damage, and there is a possibility of precipitating severe acute liver cell failure and even death.

At present the most reliable methods of detecting lesions in or close to the bile duct which involve it early and manifest themselves by jaundice, are gastroduodenoscopic intubation of the duct or percutaneous cholangiography. These techniques are not infallible, but they are safe, and present no serious ordeal to a patient.

Percutaneous cholangiography performed by an experienced operator should demonstrate dilated ducts due to mechanical obstruction in a high percentage of cases—in the case of carcinoma in 97 or 98 per cent and when due to calculus in 90 per cent or more. The effectiveness of the technique is lower in the case of postoperative stricture, but depending on the degree of obstruction and whether the ducts are filled with gas following some previous anastomosis, success should approach 80–90 per cent. A failure to intubate a duct makes it probable that the jaundice is due to some cause other than extrahepatic obstruction. I believe that percutaneous cholangiography deserves a better reputation and wider recognition and usage than it does, for its full value does not seem to be appreciated by our surgical colleagues until they experience it.

I should like to end by quoting another passage of Caldwell's article on gallstones to which I have already referred. He wrote "the x-ray, like many other useful things, is capable of harm as well as good. The dangers of the x-ray are due chiefly to those who use it. These dangerous persons may be classified in two groups. In the first group are those who call themselves Roentgenologists who think they know more about the subject than anyone else. In the second group are Physicians and Surgeons who make use of x rays, either themselves, or by proxy, who are also convinced that they know more about the subject than anyone else."

"The greatest danger of x-rays is in faulty interpretation—usually due to reading into the plates something that is not there. It is easy for an experienced observer, however honest he may be, to fall into this error."

One should "refuse to allow the clinical record to cloud one's mind and affect one's interpretation of the plates. Much suffering may result. . . ."

And he concludes, "The especially dangerous zone is in the right upper quadrant. It is in this danger zone that the number killed and wounded resulting from faulty interpretation is greatest."

These words were written in 1915. They remain true today.

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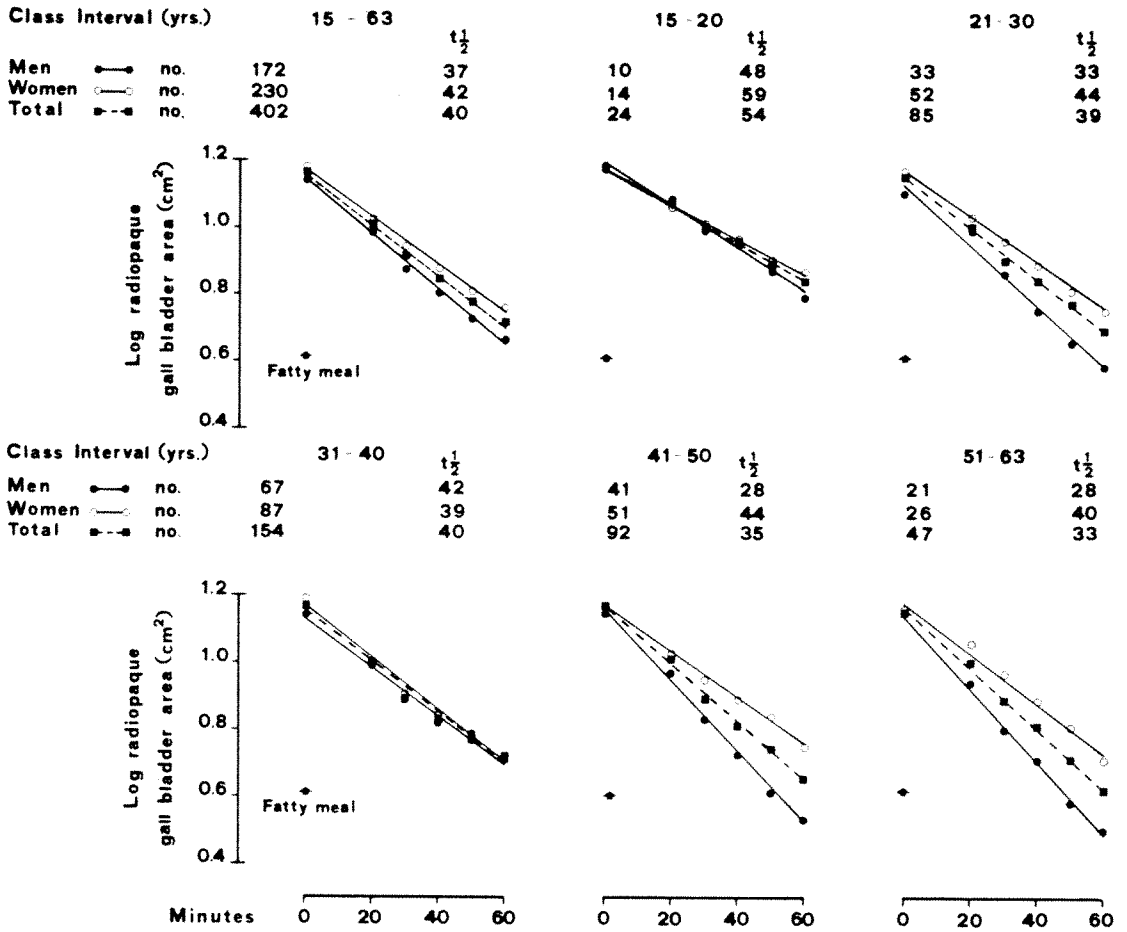


Fig. 1. Gallbladder emptying in healthy subjects induced by a fatty meal (2 egg yolks); linear regression of radiopaque gallbladder areas (cm.²) on time estimated with the least squares method.

sensitive to show even small differences in emptying rate.

This is confirmed by the results of this study carried out on a representative sample of people who, on the suggestion of their doctor, were undergoing cholecystographic examination; very frequently (in about two-thirds of the cases) no anatomo-functional abnormality was found in the extrahepatic biliary tract. Ansay[1] also found only about 30 per cent of the outpatients presenting for cholecystographic examination abnormal on the basis of roentgenography. This study,[1] which could be extremely useful as regards emptying times, since it was carried out on 701 normal subjects, unfortunately gives no information on the make-up of the sample (age, sex), nor any detailed measurements. The gaps in descriptions of material and methods make it difficult to compare our results with those obtained by other authors. Many years ago, Boyden[2–4] studied gallbladder emptying induced by a fatty meal (egg yolk) in subjects with no gastrointestinal diseases. Experimental data available on individual cases (volumetric measurements according to Boyden) have been used to calculate $t_{\frac{1}{2}}$ values, which indicate faster emptying in older subjects (55–78 years) as compared to younger subjects.[3,4] Values for $t_{\frac{1}{2}}$ calculated on the planimetric figures given by Cosmacini and Peco[7] also point to faster emptying in subjects over

50 years, as Comino[6] also concluded.

The results of the present research confirm the above observations, providing the additional information, that it is the men who cause this increase in emptying rate in older subjects; the rate for women over 20 years hardly varied between any of the age groups. The faster emptying in older healthy people than in young persons, although some authors have not noted it,[8,11,15] was definitely observed in the experimental conditions employed by us. The reasons for this are not known, and any theory put forward to interpret it (increased production and/or earlier availability of cholecystokinin and/or reduced production of anticholecystokinin) can only be suggested as a working hypothesis.

Another point which deserves analysis is that the females' emptying rate was slower than that of the males. This was particularly noticeable in the 21–30, 41–50, and 50–63 year age groups, where the differences between the sexes were significant. One convincing explanation might be that cholecystokinin is available earlier in males. This should not be altogether disregarded, as Buckler[5] also found a tendency to faster gastric emptying in men. Gastric and gallbladder emptying rates also vary according to the phase of the menstrual cycle at which research is carried out, and seem to be fastest about half way through (gastric emptying,[13] gallbladder emptying[14]). Since the gallbladder emptying rate seems also affected by the presence of premenstrual tension,[14] it may reasonably be assumed that hormonal changes are able to modify not only the gastrointestinal smooth muscle ability to respond to a stimulus, but also secretion of the digestive tract (*i.e.*, gastroduodenal hormones). This could explain some of the contradictory results obtained on gallbladder emptying rate in fertile age women.[2,4,14,16–18]

CONCLUSIONS

The adoption of strict criteria for admitting subjects to the trial and uniform methods of research has given reliable information for quantitative study of gallbladder emptying after a fatty meal (2 egg yolks) in healthy subjects.

By planimetric measurement of the radiopaque areas, it was possible to prove that the emptying rate varies with age and sex, and is higher in older subjects and men.

SUMMARY

Gallbladder emptying induced by a fatty meal (2 egg yolks) was studied by oral cholecystography in 402 subjects without gastrointestinal disorders.

Roentgenograms were made at intervals up to the 60th minute after the meal, and the radiopaque areas were measured planimetrically to provide a quantitative assessment of emptying.

The pattern of emptying was compatible with an exponential function. The mean value for $t_{\frac{1}{2}}$ was higher in the women; emptying tended to be slower in younger subjects.

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OPERATIVE CHOLANGIOGRAPHY USING A 6:1 CRISSCROSSED GRID*

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RADIOGRAPHY in a surgical suite has been often compromised by the surgical environment. This observation is supported by reviewing publications concerning operative cholangiography. Many authors have had to resort to drawings along with their roentgenograms in order to convey the information that they hoped to reveal on their films.

We believe that a major factor resulting in missing stones during operative cholangiography is the direct result of accepting suboptimal quality roentgenograms. This is the most chagrining error of the biliary tract surgeon and his radiologist. Hall *et al.*[2] have recently pointed out reasons for failure in stone detection. These reasons mostly result from suboptimal roentgenographic technique, in their opinion.

The primary objective of our effort was to accomplish optimal diagnostic accuracy. The substance of this report is the description of a current technique consisting of well established features from the past, the addition of some new features and, finally, the effects of this effort on the utilization of operative cholangiography in our institution.

We advocate routine operative cholangiography on all patients undergoing cholecystectomy. The reasons have been well presented by others.[1,2]

To demonstrate the results of these changes, we have selected, at random, a

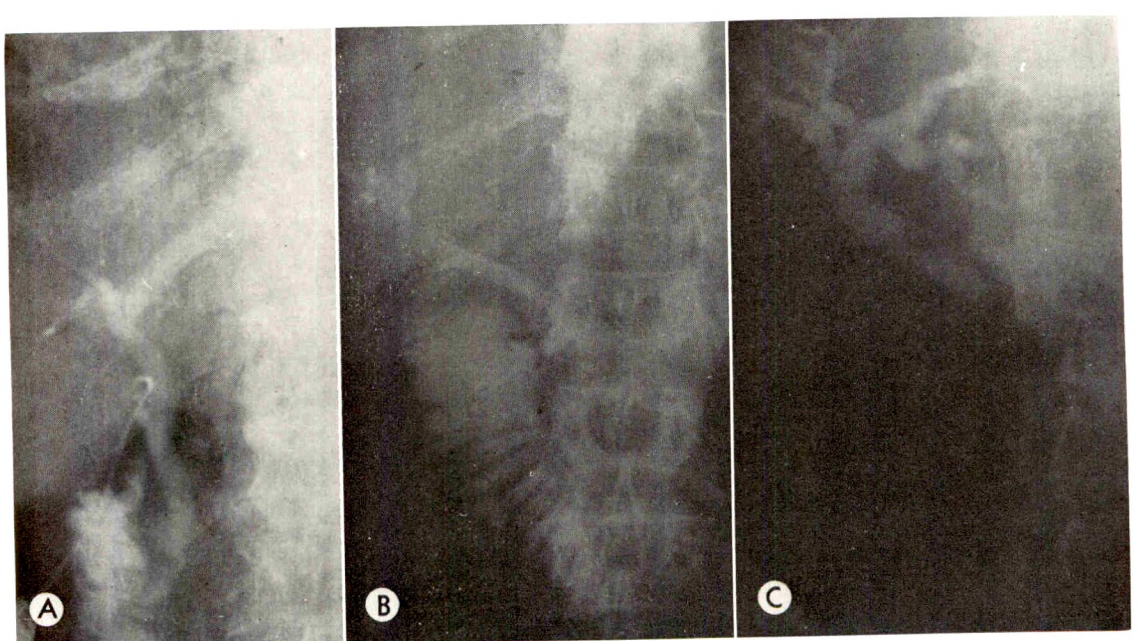


FIG. 1. (*A–C*) Random specimen films of 3 separate cholangiograms using a 6:1 grid cassette. This is the more prevalent practice and our former technique.

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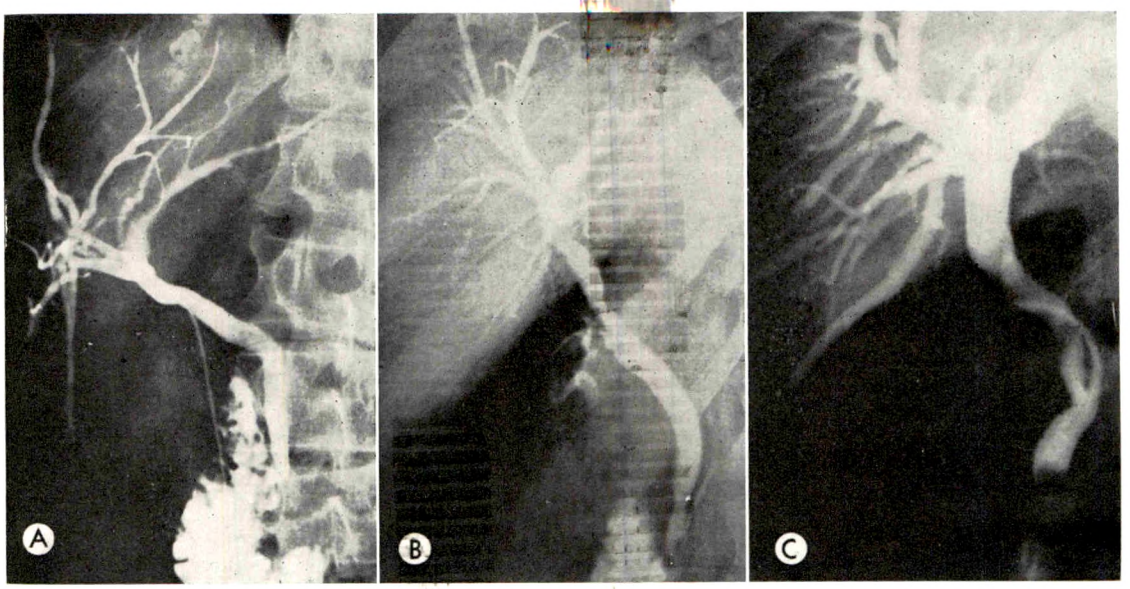


Fig. 2. (*A–C*) Random specimen films of 3 separate cholangiograms using our new 6:1 crisscrossed grid technique.

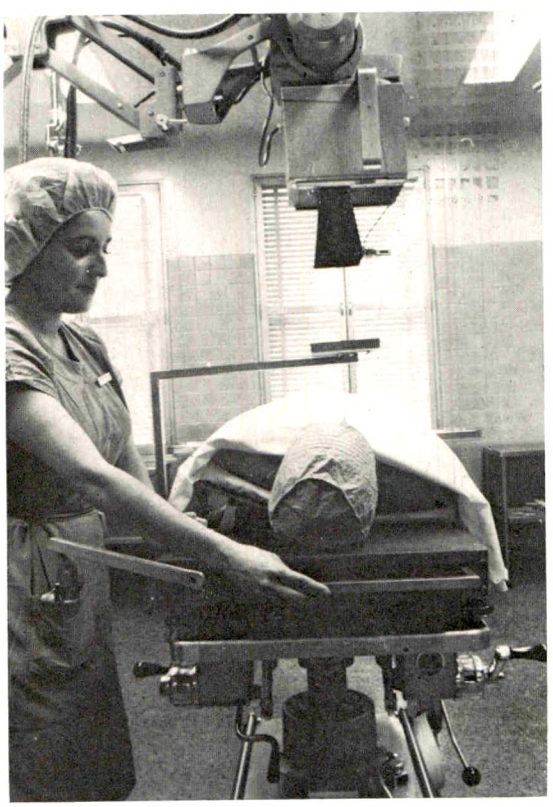


Fig. 3. Head-end view with a patient on the film tunnel. The localizing paddle is in position for centering the tube.

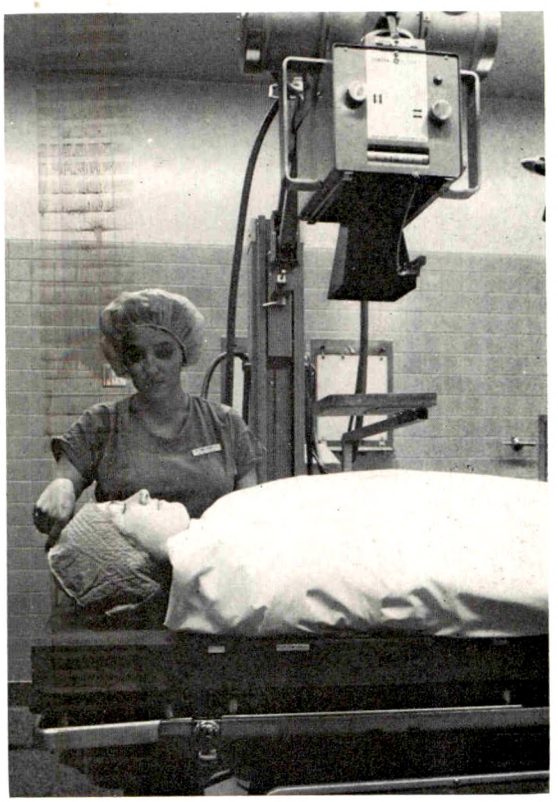


Fig. 4. View of film tunnel from the surgeon's side.

cholangiographic film from each of 3 old cases (Fig. 1, *A–C*) for comparison with single films selected at random from 3 separate recent operative cholangiographies (Fig. 2, *A–C*). The older films represent that quality resulting from the use of a 6:1 grid cassette so widely employed today.

THE 6:1 CRISSCROSSED GRID APPARATUS

This crisscrossed 6:1 ratio, 85 line, Liebel-Flarsheim Grid is equivalent to a 12:1 Bucky in the improvement of film quality. Positioning is critical. Because of this, these grids have been generally avoided.

In order to use this type of grid in surgery with a portable roentgen-ray machine, a special tunnel was constructed to help the technologist obtain the required precise positioning of the tube. Figures 3 and 4 show a patient positioned on the tunnel prior to a cholecystectomy.

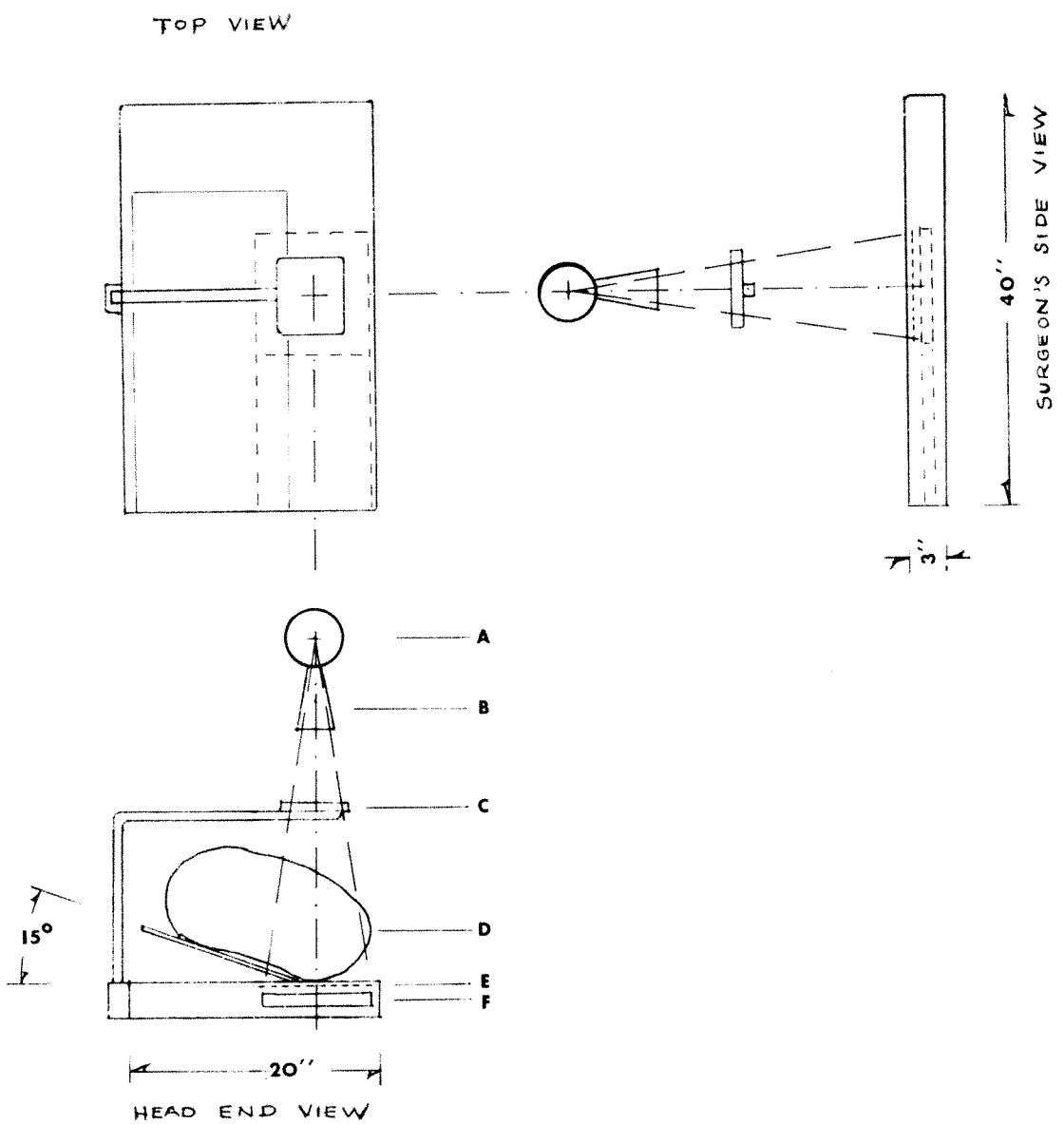


FIG. 5. Three-dimensional drawing of the operative cholangiographic apparatus.

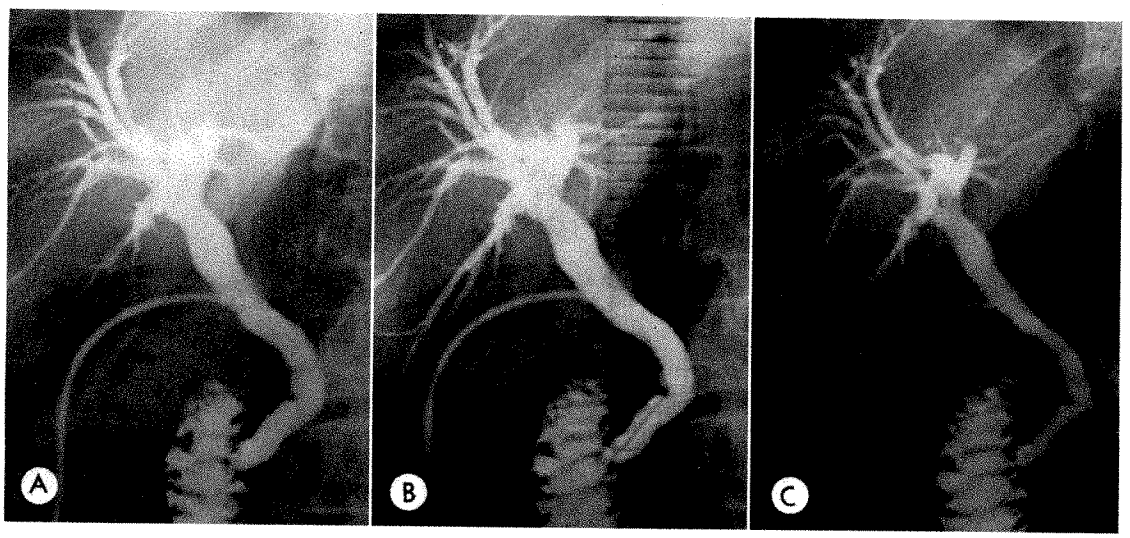


Fig. 6. (*A–C*) A series of 3 ascendingly exposed films that were accepted as adequate and interpreted as normal.

Figure 5 is a three-dimensional drawing of the apparatus. We can more clearly explain its features from this drawing. A represents the 200 ma. portable roentgen-ray tube. The commercial localizer has been modified by an extension cone (B) that reduces the 10×12 inch field to 9×11 inches. Means, not illustrated, were provided to obtain 2 dimensional vertical alignment of the central beam. C represents a field localizing paddle. This is required to precisely reposition the tube after the operation is underway and when the technologist can no longer find the grid because of the drapes. The paddle is removed during the operative phases. D represents a patient tilted 15° into the right posterior oblique position. A lever accomplishes this and, of course, only during roentgenography. The obliquity usually projects the common duct free of the spine. E is the 6:1 crisscrossed grid fixed above the film cassette. F represents a slot at the head end of the apparatus into which a cassette is loaded. A crank moves it into position beneath the gallbladder area.

SURGICAL AND ROENTGENOGRAPHIC TECHNIQUE

Before the surgeon begins the operation, a qualified technologist positions and bolsters (pads) the patient over the tunnel. A test roentgenogram is routinely obtained.

The surgeon exposes and cannulates the cystic duct using a No. 16 Jelco Teflon needle or its equivalent. After a tube has been connected to the needle the surgical field is overdraped. The localizing paddle (Fig. 5C) is covered by a sterile transparent plastic bag and brought into position. Precise repositioning of the tube follows by using the light localizer on the paddle. The paddle is then removed and the films are exposed without cut-off.

Injections must be air free. The anesthesiologist must produce complete apnea. We now use 20 per cent Hypaque according to the following schedule:

Dosage Schedule for 20 Per Cent Hypaque

Duct Size	*1st Film*	*2nd Film*	*3rd Film*
Under 10 mm.	3 cc.	3 cc.	5 cc.
Over 10 mm.	3 cc.	5 cc.	8 cc.

Ascending Kilovoltage Feature

From the following exposure chart one notes that we utilize increasing kilovoltages in our series of 3 films in the hope of better penetrating, or seeing through, the common and hepatic ducts.

*Exposure Chart**

Patient Size	ma.	Time	At kvp.
Small	200	0.5 sec.	80–85–95
Medium	200	1.0 sec.	80–85–95
Large	100	3.0 sec.	80–85–95

* Other technical data: Par Speed Screens, High Speed Film, 30 sec. in 80° F. Med Developer.

Figure 6, *A–C*, is a series of 3 films exposed at increasing kilovoltages. They were interpreted as normal. Figure 7 is a postoperative cholangiogram on the same patient as in Figure 6. The latter cholangiogram demonstrates a missed stone. The original cholangiograms should have been made with less dense contrast and less quantity of the opaque material. The diagnosis on the erroneously interpreted series of Figure 6 should have been, "unpenetrated biliary ducts, repeat the cholangiogram."

Figure 8, *A–C*, on the other hand, illustrates optimal results from good technique. We see through the ducts and can be certain that there are 2 stones and not more.

DISCUSSION

About 15 surgeons on our staff performed 161 total cholecystectomies and common duct explorations during the past year. Operative cholangiograms were carried out in 92 of these patients. This is an average of 58 per cent of the total group. As opposed to 3 years ago, this is a sharp rise in utilization. At that time we performed operative cholangiography on about 10 per cent of our patients having biliary surgery. Many factors may be stimulating this increased use of "routine operative cholangiography," but one fact is evident: a technique must be adequate to assure the surgeon of a high degree of diagnostic accuracy or he would not extend his operative time to utilize it. Further on this point, we must mention that good technique is not the result of just physical factors. A well-trained technologist, a cooperative operating room crew and experience must all be present. With these factors present and after the cystic duct has been cannulated, complete cholangiography routinely requires less than 10

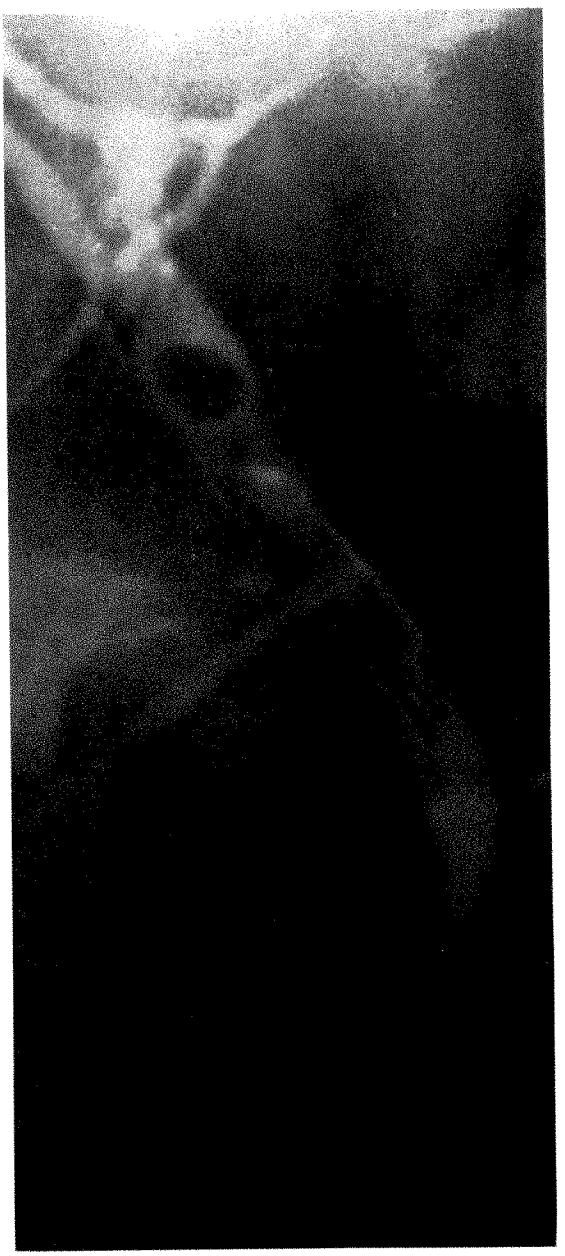


FIG. 7. Postoperative T-tube cholangiogram on the same patient as in Figure 6, using less dense contrast agent and heavier exposure that demonstrates a missed common hepatic duct stone. Radiation penetration of the ducts is an obligation of the cholangiographer.

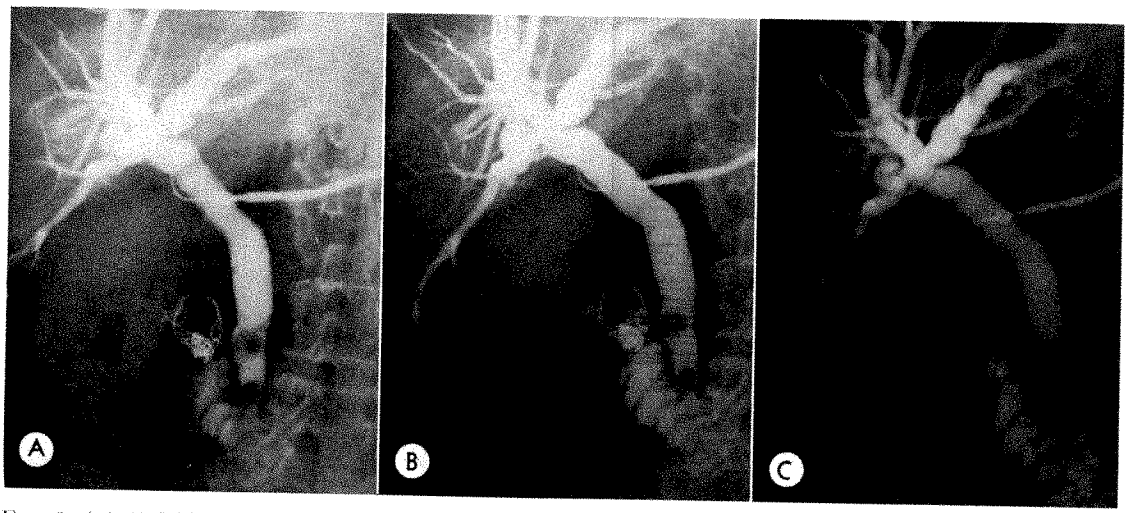


FIG. 8. (*A–C*) This series of 3 films at increasing kilovoltages demonstrates 2 common duct stones. The films are assuring that there are not more than 2.

minutes, and is a diagnostic procedure not replaceable by any other means.

SUMMARY

An improved crisscrossed grid technique for operative cholangiography is described in detail.

Common pitfalls leading to error in diagnosis are discussed.

Efforts in the improvement of operative cholangiography in one hospital are documented.

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REMNANT CYSTIC DUCT IN T-TUBE CHOLANGIOGRAPHY*

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IN patients with previous gallbladder surgery, recurrent biliary calculi and cholangitis may occur postoperatively. An incompletely excised cystic duct may contribute to stone formation and infection. An analysis of postoperative T-tube cholangiograms was undertaken to assess the incidence of such cystic duct remnants as well as their characteristic features.

A specific objective of this study was to clarify the criteria so that they might be applied more precisely to the intravenous cholangiogram in order to enhance its accuracy in the detection of cystic duct remnants.

MATERIAL AND METHOD

Seventy postoperative T-tube cholangiograms obtained between 1968 and 1971 were reviewed. Patients in whom this did not represent the first exploration of the common duct were excluded from the analysis. In all cases a cholecystectomy had been performed at the time the tube was placed into the common duct intraoperatively.

For postoperative cholangiography, a solution of 30–60 per cent renografin or 25–50 per cent hypaque had been injected into the T-tube under fluoroscopic control. Filming in various degrees of filling of the biliary radicles was done in anteroposterior, right posterior oblique and left posterior oblique projections. Reliance increasingly was placed upon fluoroscopic spot roentgenograms to the point that roentgenograms obtained by the overhead tube were omitted entirely during 1971.

All distances and lengths were measured directly on the films. Magnification was discounted because consistent correction factors could not be applied to all patients. Flexible rulers were used whenever the course of the ducts was undulating. When

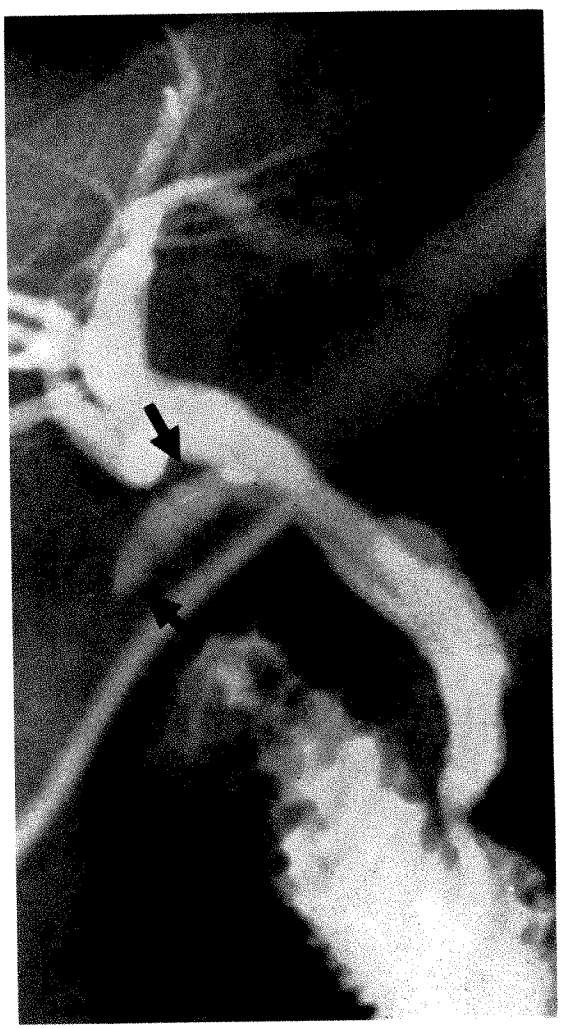


FIG. 1. Angular cystic stump. The cystic duct joins the common hepatic duct laterally forming a right angle junction. Note the associated short hepatic and much longer common bile duct.

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Supported in part by USPHS grant GM01910.

the cystic duct stump measured less than 5 mm., the case was considered negative.

All cases with remnants measuring 5 mm. or more were categorized into 2 major groups depending on the type of junction that the stump formed with the common hepatic-common duct radicle. One major group consisted of those patients in whom the junction was clearly angular (Fig. 1). The second group was composed of patients in whom the cystic duct ran parallel to the common hepatic duct before joining (Fig. 2, *A* and *B*).

RESULTS

Of the 70 cholangiograms reviewed, 44 (63 per cent) had a cystic duct remnant (Table 1). No significant difference was found between the mean age of the negative or positive group (56 and 51, respectively). Of the 44 remnants, 30 (68 per cent) were angular and 14 (32 per cent) were parallel.

If only the parallel component was considered in the parallel category, the remnant length was similar to the angular group, showing a range of 10–40 mm. and a mean length of 22 mm. (Table II). Significantly, however, all but 3 of the cases in this group had in addition an angularly oriented portion of cystic duct left behind. The total length of remnant in this group was therefore significantly higher: a mean value of 34 mm. as compared to 20 mm. in the angular group.

There were significant differences in the length of the common hepatic and common bile ducts between the angular and parallel groups (Table II). These measurements showed that when the cystic duct junction was angular, it occurred high, leaving a short hepatic and a much longer

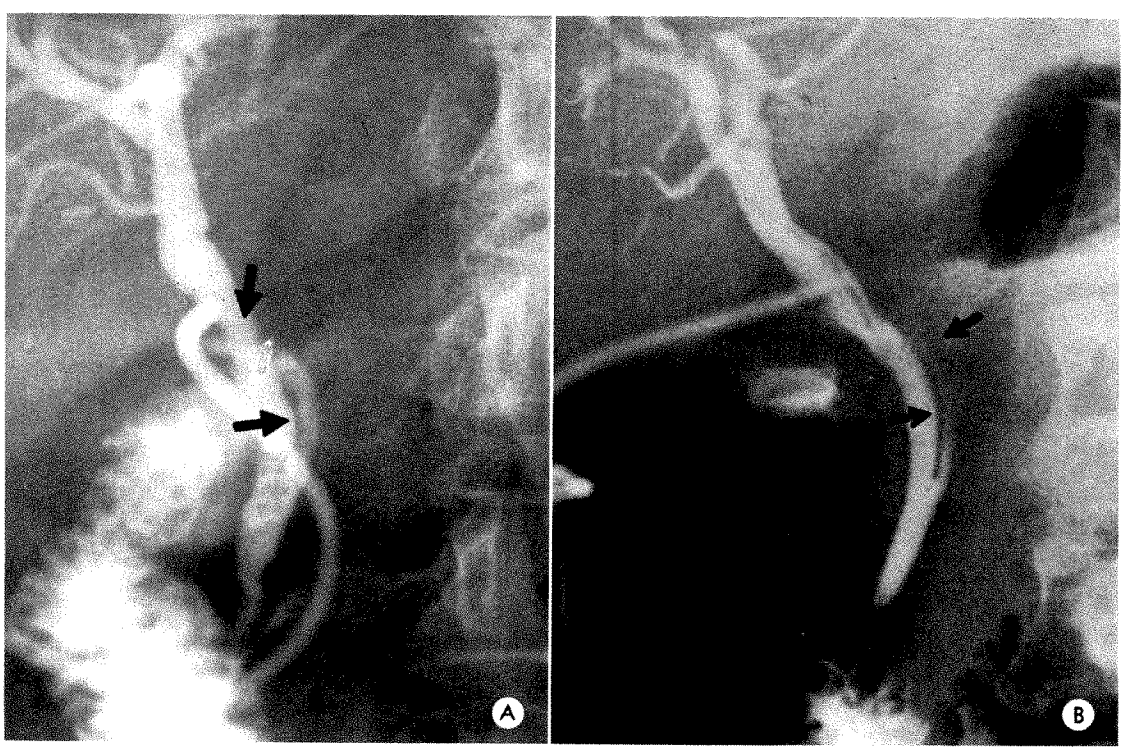


FIG. 2. (*A*) Parallel cystic stump. The cystic duct joins the common hepatic duct after coursing parallel to it for some distance and frequently medially. Note the associated angular component which after crossing the common hepatic duct terminates laterally. (*B*) Parallel cystic stump. Parallel cystic stump without associated angular component, a much less frequently encountered type. Here the common hepatic duct is characteristically long; the junction is low and embedded in the pancreatic parenchyma resulting in a short common duct.

TABLE I

INCIDENCE AND CATEGORIES OF CYSTIC DUCT REMNANT

Cholangiograms	70
Negative	26 (37%)*
Positive	44 (63%)†
Cystic duct remnants	44
Angular	30 (68%)
Parallel	14 (32%)

* Mean age: 56 years.
† Mean age: 51 years.

common bile duct. The reverse was true in the parallel group, indicating that the junction was much lower. Indeed when the point of junction was related to the upper border of the duodenum, it was superior to the upper border of the duodenum in the angular group by an average distance of 17 mm. When the cystic duct was parallel, the junction point was usually inferior to the duodenal border, averaging 12 mm. below this point.

Because oblique views were routinely obtained, the course of the parallel cystic duct could in most instances be precisely assessed. The majority (8 out of 14) were found to take a medial course to the common hepatic duct. In the remaining cases it showed posterior, lateral or intermediate positions (Table III).

TABLE II

LENGTH OF CYSTIC DUCT REMNANTS AND MAJOR EXTRAHEPATIC BILIARY DUCTS

	Angular (mm.)	Parallel (mm.)
Length of Cystic Duct Remnant		
Range	5–55	10–60 (10–40)*
Mean length	20	34 (22)*
Common Hepatic Duct		
Range	10–105	30–85
Mean length	38	59
Common Bile Duct		
Range	45–90	30–75
Mean length	70	43

* Parallel component only.

TABLE III

COURSE OF PARALLEL CYSTIC DUCT

Medial	8
Posterior	2
Lateral	1
Intermediate (usually posteromedial)	3

DISCUSSION

When the gallbladder is removed and the common duct explored, complete excision of the cystic duct is not considered to be prudent even by experienced surgeons. The excessive traction necessary to accomplish this could lead to subsequent stricture of the common duct.[2,5,7] Leaving a short remnant of no more length than 5 mm. is therefore considered good surgical practice.[2] It was for this reason that cases with such short stumps were classified as being negative in this review.

In a postcholecystectomy patient who develops or continues to have abdominal distress, a cystic duct remnant cannot be assumed to be responsible for these symptoms.[7] When intravenous cholangiography is performed routinely in completely asymptomatic postcholecystectomy patients, a remnant frequency of 40 per cent has been reported.[1] Much higher figures have been reported by Millbourn,[7] who in an analysis of intraoperative and postoperative cholangiograms found remnants in 83 per cent of patients. Our lower incidence of 63 per cent probably reflects the current reduced intraoperative risk permitting more time for careful dissection. If patient age can be used as a gross indicator of the difficulty that may have been encountered intraoperatively, it is worthwhile to point out that the mean age was the same in our positive and negative groups (Table I).

Although it may be of little consequence for most postcholecystectomy patients that a cystic duct stump is left behind, the remnant in other patients may be the site of infection and stone formation.[4] Higher

frequencies of stumps are found in those patients who have common duct stones or recurring cholangitis after gallbladder removal in an unselected group of such postoperative cases.[1] Similarly, remnants tend to be longer in these cases than in patients who are mildly symptomatic or asymptomatic.

It has previously been stated that remnants of the cystic duct are usually only left unexcised when the duct is parallel in course with a low insertion.[8] Our series with a 68 per cent incidence of angular and a 32 per cent incidence of parallel stumps actually shows the same frequency of duct types that are normally found in anatomic studies.[3,6] This indicates that angular and parallel cystic ducts are just as likely to be left incompletely excised. Undoubtedly, some of the parallel cystic ducts could have been more extensively excised and some of the remaining angular remnants completely removed had their presence been recognized intraoperatively. Only a few of the remnants are actually demonstrated on intraoperative cholangiograms even on a retrospective review. This demonstrates the limitation of intraoperative cholangiography with presently available techniques. It also emphasizes that a properly controlled cholangiogram, during which graded amounts of contrast material can be injected under fluoroscopic control and filming can be done in various projections, is the best method for accurately assessing duct architecture and spatial relationships. Such cholangiography is far superior in demonstrating biliary duct architecture than the most meticulous surgical exploration.

In the present study, it was also shown that parallel duct stumps had a much lower point of insertion, with the junction in most cases below the duodenum embedded in the pancreatic parenchyma. This, frequently combined with a medial course, explains why these ducts are not easily accessible to surgical dissection. Similarly, some may actually be separated from the common duct by a membrane lacking a true wall, so that dissection and excision could produce serious injury. While it cannot be assessed from available data how often this was actually the case in our patient group, the common hepatic duct and parallel stump diameter measurements strongly suggest that the medially coursing remnant tends to be more intimately part of the common hepatic duct (Table IV). These measurements also indicate that parallel ducts in other than medial positions do not share these characteristics. Under these circumstances, the hepatic duct is wider at this level with diameter measurements similar to those in the negative group and the cystic duct is a much more separate structure. When such medial remnants become dilated because of calculi or infection, dilatation could easily compromise the narrower than normal lumen of the common hepatic duct and cause extrahepatic biliary obstruction early in the course of the disease.

In spite of this, it is disturbing that the mean length of the parallel remnants is as great as 34 mm. Interestingly, an associated laterally oriented component left behind in most cases is primarily responsible for the difference in lengths of remnants in the two major groups. If this had been recog-

TABLE IV

HEPATIC DUCT DIAMETERS IN PARALLEL REMNANT AND NEGATIVE GROUP

Common Hepatic Duct	Cystic Stump
Parallel Remnants (medial course—6 cases)	
Range 3–8 mm.	2–7 mm.
Mean 6 mm.	3.6 mm.
Parallel Remnants (other course—5 cases)	
Range 6–10 mm.	4–7 mm.
Mean 8 mm.	5.6 mm.
Negative Group (18 cases)	
Range 4–12 mm.	
Mean 8.4 mm.	

nized intraoperatively, a more extensive resection would have been probably performed in the majority of cases.

SUMMARY

An analysis of 70 postoperative T-tube cholangiograms is described. Our findings indicate:

1. Cystic ducts are just as likely to be left incompletely excised whether they are angular or parallel in type. Significantly longer remnants, however, are left in the parallel group.

2. Application of cholangiographic techniques in the operating room similar to those presently available in the postoperative setting will undoubtedly reduce the over-all incidence of remnants as well as the mean lengths of those which cannot be excised completely.

3. A medially running remnant is much more intimately associated with a common hepatic duct than other types. Although such a remnant is frequently difficult and in some instances impossible to resect, it probably also causes extrahepatic obstruction early with only minimal dilatation.

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CHOLEDOCHAL CYST: A CONCEPT OF ETIOLOGY*

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CHOLEDOCHAL cyst is a rare malformation of the pancreaticobiliary ductal system. Many theories[1,3,4,15,18,26,27] have been proposed regarding its etiology. None of these, however, explains all the findings. In 1969 one of us (D. P. B.)[2] presented a new concept of etiology based on an anomaly of the duct system found on cholangiography in 3 cases. Since that time, an additional 4 cases have been collected. In addition, 12 other examples of the anomaly have been found in the literature, making a total of 19 cases. In all cases with adequate cholangiographic studies delineating the distal ductal system, the anomaly has been evident. It is the purpose of this communication to indicate that this anomaly is the etiology of choledochal cyst.

NORMAL EMBRYOLOGIC DEVELOPMENT

The normal embryologic development of the pancreaticobiliary ductal system[5,16] is illustrated in Figure 1. In the normal relationships of the pancreatic duct and common duct, the 2 channels are a common channel over a distance of 5 mm. or less. The normal and anomalous relationships between the common bile duct and the pancreatic duct found in our cases are shown in Figure 2. In this anomaly, the relatively small distal common bile duct enters the duct of Wirsung at a right angle approximately 2 to 3.5 cm. from the ampulla of Vater. This anatomic arrangement is likely due to embryologic failure of the normal separation of the pancreatic and common bile ducts. Some authors have reported anomalies of the pancreaticobiliary ductal system associated with choledochal cyst and their illustrations have demonstrated an arrangement similar to that found in our cases.[6,9,10,13,15,21,23] However, the etiologic significance of this anomaly has not been stressed. We feel that this is due principally to inadequate study of the distal choledochus and its relation to the pancreatic duct.

DIAGNOSIS

The diagnosis of choledochal cyst is often difficult and in many cases the diagnosis is not made until the time of surgical exploration when the correct diagnosis may be made by direct observation and by operative cholangiography.[5,14] On occasion, the entire pancreaticobiliary ductal system may be opacified by operative cholangiography. Often, however, the cyst obscures much of this anatomy (Fig. 3) or the examination is technically inadequate. For these reasons, postoperative cholangiography under fluoroscopic control is advocated. This is possible, if some access to the biliary tree, *e.g.*, a cholecystostomy or T-tube, is left at surgery. Technically poor cholangiograms are probably responsible for failure to demonstrate the distal ductal system in many cases in the literature.

NEW CONCEPT OF ETIOLOGY

A constant anomalous arrangement of the pancreaticobiliary ductal system has been found in our 7 cases (Fig. 4–10) of choledochal cyst and an identical arrangement has been found in the illustrations from previous reports of this entity. It is proposed that this anomaly, which may be

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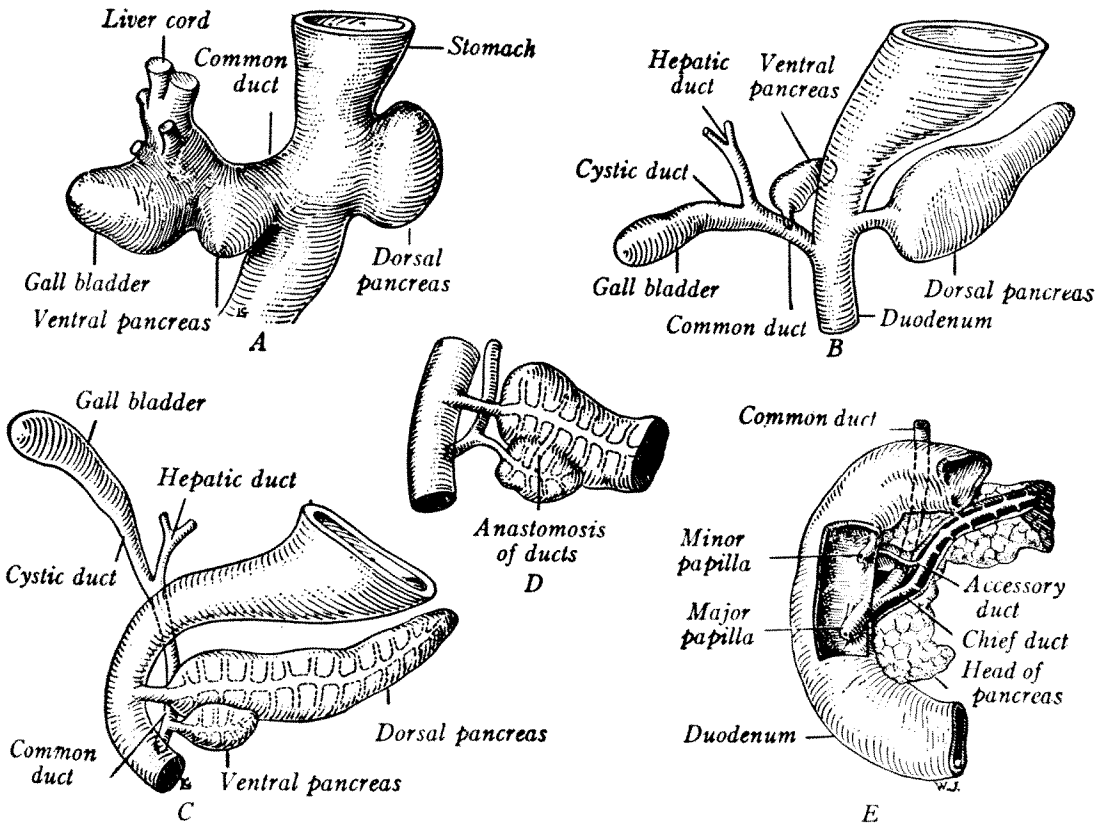


FIG. 1. Development of the human pancreaticobiliary ductal system viewed from the left side. The ventral and dorsal pancreatic buds arise on opposite sides of the duodenum. (*A*) Of the 2 pancreases, the dorsal one grows more rapidly. (*B*) The dorsal pancreas is an elongated nodular structure with a centrally coursing duct. (*C*) Unequal growth of the duodenal wall moves the common bile duct dorsally, bringing the ventral pancreas into the dorsal mesentery. The 2 pancreases interlock intimately. (*D*) The dorsal pancreas forms all of the adult pancreas except for the head which is derived primarily from the ventral pancreas. The ductal system fuses in such a way that the long distal segment of the dorsal duct plus the entire ventral duct serve as the chief line of drainage (duct of Wirsung). (*E*) The proximal segment of the dorsal duct constitutes the duct of Santorini and may or may not persist into adult life. (Adapted from Arey.)

found in the absence of choledochal cyst,[11] precludes the development of a sphincter at the junction of the pancreatic and common bile ducts. This sphincter is present when the pancreaticobiliary ductal system develops normally.[8]

Since the maximum pressure of the pancreatic duct is 30 to 50 cm. of water, while the maximum pressure of the common duct is 25 to 30 cm. of water,[8] this pressure differential would allow flow of pancreatic juice into the bile ducts if there is no sphincter present to prevent it. There is evidence that such flow does occur since the fluid aspirated from choledochal cysts has high amylase content.[1,22]

Free flow of pancreatic juice into the common duct would produce recurrent bouts of cholangitis.[12] The clinical triad of pain, jaundice and an intermittently present right upper quadrant mass is readily explained by this etiologic concept. Recurrent episodes of cholangitis result in thickening of the wall of the common duct. The walls of choledochal cysts vary from 2 to 10 mm. in thickness[12,19] and histologically are fibrous without an epithelial lining,[7,12] even in the newborn with choledochal cyst.[25]

As the common duct dilates further, it expands anteriorly since expansion in other directions is relatively restricted. The

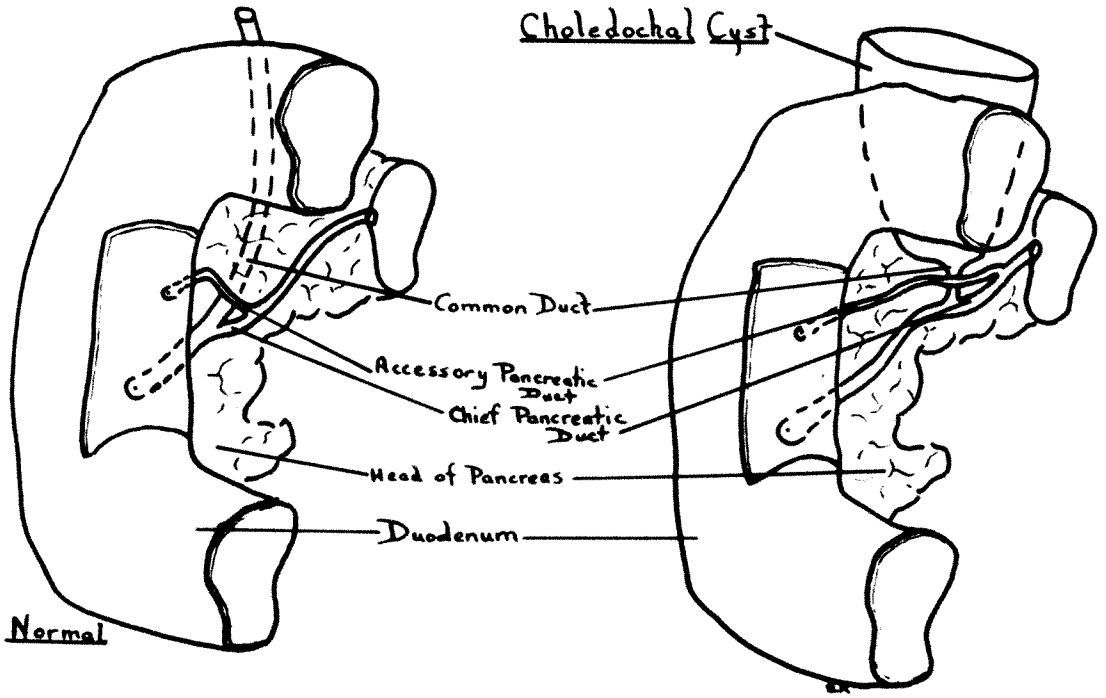


Fig. 2. The normal pancreaticobiliary ductal relationship is shown on the left. On the right is a sketch showing the anomalous pancreaticobiliary ductal relationship in a choledochal cyst. Note the shortness and small caliber of the distal common duct and its insertion into the duct of Wirsung at a right angle at an unusually long distance from the ampulla of Vater.

common duct could become obstructed at the distal end of the cyst from edema or eventually, fibrosis. This explains cases described as atresia of the common duct. To ascribe such cases to congenital atresia of the distal common duct is illogical since such an occurrence would result in unrelenting jaundice from birth.

SURGICAL IMPLICATIONS

The surgical approach to this entity has been a matter of controversy.[1,6,12,15,18,19,24] Alonso-Lej *et al.*[1] advocated complete excision of the cyst with anastomosis of the remaining biliary tree to the duodenum or jejunum. It has been shown that if patients survive complete excision of the cyst, they have a lower morbidity. However, most recent reports oppose this approach because of the relatively high mortality. Instead, they favor duodenocholedochostomy or jejunocholedochostomy, either direct or by Roux-en-Y anastomosis.[20] In acutely ill patients, decompression of the cyst may be

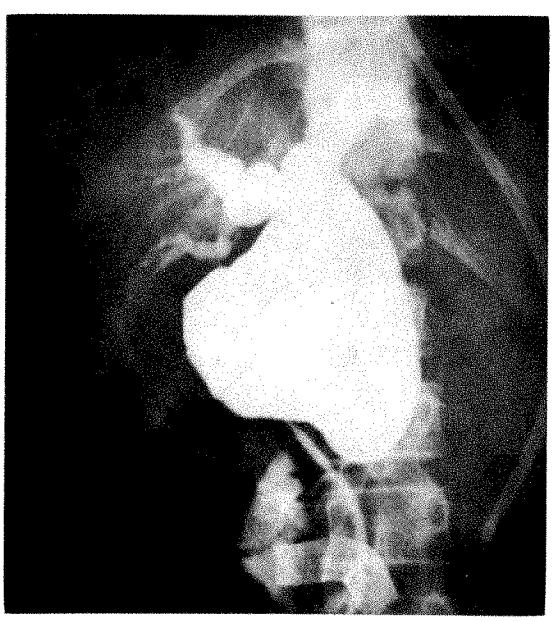


Fig. 3. Operative cholangiography demonstrating the large choledochal cyst with a small duct extending from its inferior aspect into the duodenum. This illustration demonstrates the obscuration by the cyst of the true relationship of the choledochal cyst to the pancreaticobiliary ductal system.

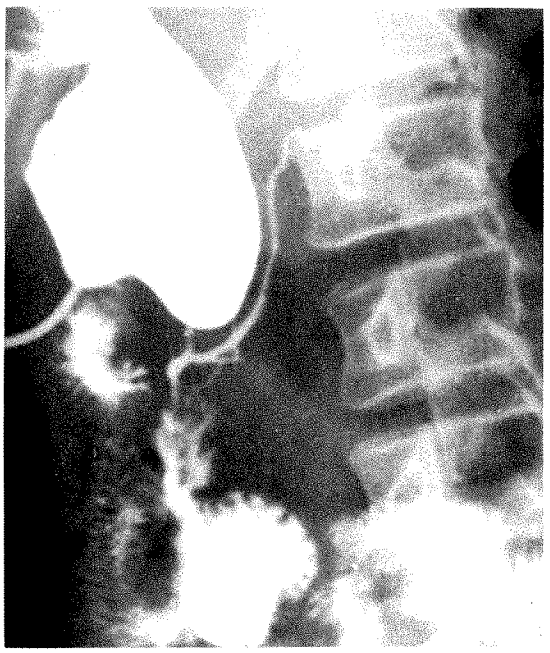


FIG. 4. Postoperative cholangiogram obtained through a T-tube on the same case as Figure 3 in the oblique projection, demonstrating the short segment of common duct entering the pancreatic duct which was obscured in the previous operative cholangiogram.



FIG. 5. Choledochal cyst with the characteristic anomalous arrangement of the ducts.

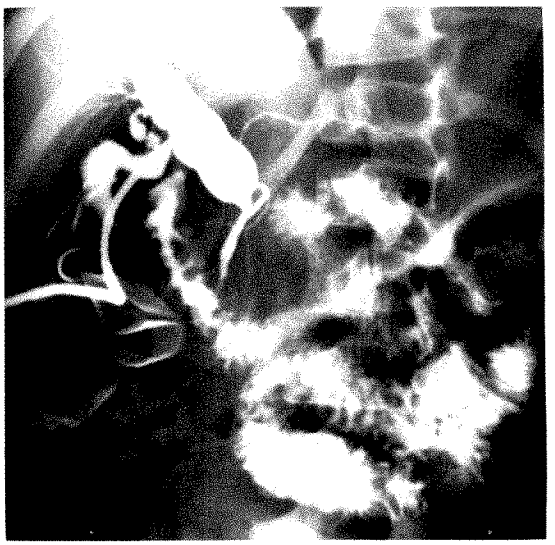


FIG. 6. Choledochal cyst with the characteristic anomalous arrangement of the ducts.

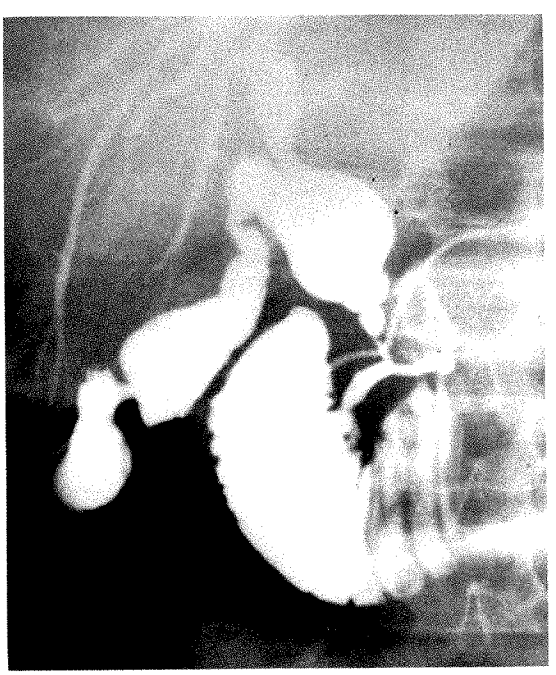


FIG. 7. Choledochal cyst with the characteristic anomalous arrangement of the ducts.

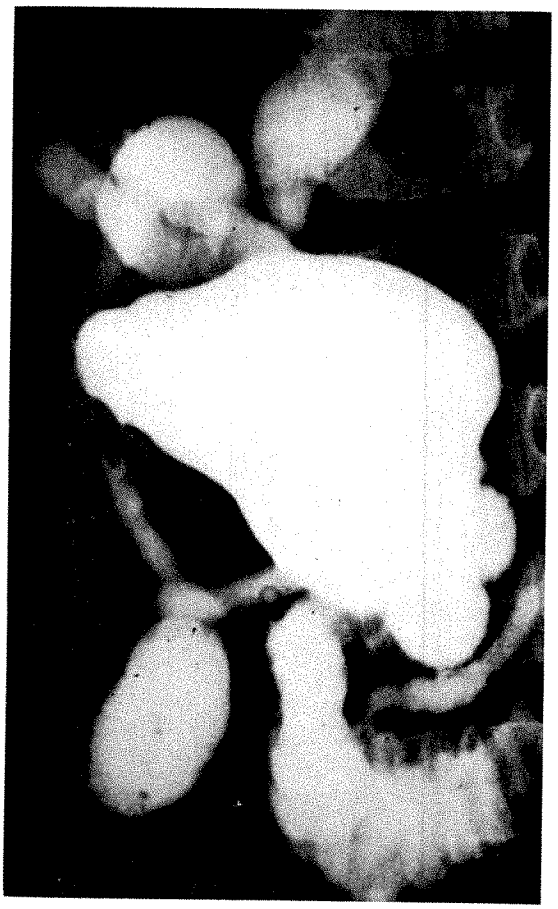


Fig. 8. Choledochal cyst with the characteristic anomalous arrangement of the ducts.

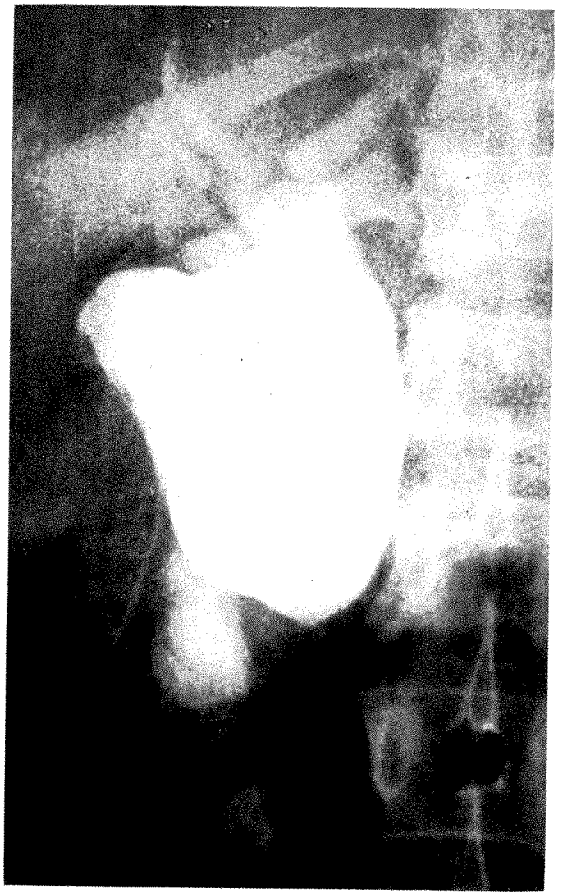


Fig. 9. Choledochal cyst with the characteristic anomalous arrangement of the ducts.

all that is possible with correction of the abnormality at a later time.

In light of our demonstration of the free communication of the biliary and pancreatic ducts which allows pancreatic juice to enter the biliary ducts, it is suggested that division of the cyst at its distal end would terminate this communication. A direct anastomosis of the remaining portion of the cyst to the duodenum or jejunum could be done, or to a Roux-en-Y anastomosis. The latter would possibly be preferable because of less contamination of the cyst by intestinal contents.[17,20] It is apparent that if such a surgical approach is contemplated, cholangiographic demonstration of the pancreaticobiliary junction is imperative.

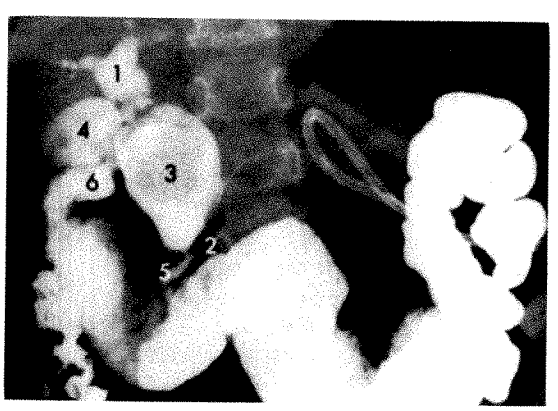


Fig. 10. Characteristic configuration of the ducts with choledochal cyst: (1) dilated intrahepatic ductal system; (2) pancreatic duct; (3) choledochal cyst; (4) gallbladder; (5) common channel of common duct and pancreatic duct; (6) duodenum. (Reproduced with permission of Drs. H. Taybi, A. R. Margulis and H. J. Burhenne; and C. V Mosby Co., St. Louis.[23])

SUMMARY

1. A new concept of the etiology of choledochal cyst is presented. The etiology of this entity is based on an anomalous relationship between the common bile duct and the pancreatic ducts.

2. This anomaly allows reflux of pancreatic juice into the common bile duct. Pancreatic juice leads to cholangitis and dilatation of the common duct.

3. The roentgenographic evaluation of choledochal cyst with special emphasis on cholangiography is presented.

4. The surgical implications of this anomaly are suggested.

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THE MERCEDES-BENZ SIGN: INSIGHT INTO THE DYNAMICS OF FORMATION AND DISAPPEARANCE OF GALLSTONES*

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THE "typical" clinical history of cholelithiasis is often absent. In the investigation of a patient with nonspecific abdominal complaints, the initial diagnosis may be made by the radiologist when calcifications in the right upper quadrant are incidentally observed in the course of examination of another body system. Up to 90 per cent of gallstones are nonopaque, however, and require adequate opacification of the bile for their identification. While cholecystography has immeasurably advanced the clinical diagnosis of diseases of the gallbladder and cholelithiasis, a recent editorial has stressed the frequent poor opacification of the gallbladder on first-dose oral cholecystography.[4] Yet even when single or multiple *nonopaque* gallstones are present, the diagnosis may on occasion be made on plain film roentgenogram by the observation of stellate radiolucencies in the area of the gallbladder. These reflect gas-containing fissures within the calculus, a phenomenon first observed by Bauer[3] in 1931. It can be aptly referred to as the "Mercedes-Benz" sign (Fig. 1), since such radiolucent fissures usually present as a triradiate pattern. The filling defect produced on oral cholecystography or perhaps a faint rim of calcification about the stone, often unnoticed until the sign is observed, encircles the lucent radii. Twenty-eight cases have been previously reported.[1,6,7,9,12,15,16,18,20,22,25,30,31]

In addition to permitting a plain film roentgenographic diagnosis of nonopaque gallstones, these *in vivo* roentgenographic observations regarding the Mercedes-Benz sign provide an insight which can be correlated with recent investigations dealing with the basic physiochemical processes of gallstone formation and structure. Recent clinical interest in the phenomenon of spontaneous disappearance of gallstones has emphasized chemical or metabolic factors, to the exclusion of structural considerations. This report also shows that the internal dynamics of gas-fissuring may account for some cases of spontaneous disappearance of gallstones.



FIG. 1. The Mercedes-Benz symbol is distinguished by 3 encircled radii.

REPORT OF CASES

CASE 1. A 48 year old female presented with a 5 month history of intolerance to fatty foods and intermittent episodes of right upper quadrant pain. Physical and routine laboratory examinations were unremarkable. A preliminary roentgenogram of the right upper quadrant before oral cholecystography showed a single Mercedes-Benz sign (Fig. 2*A*), without

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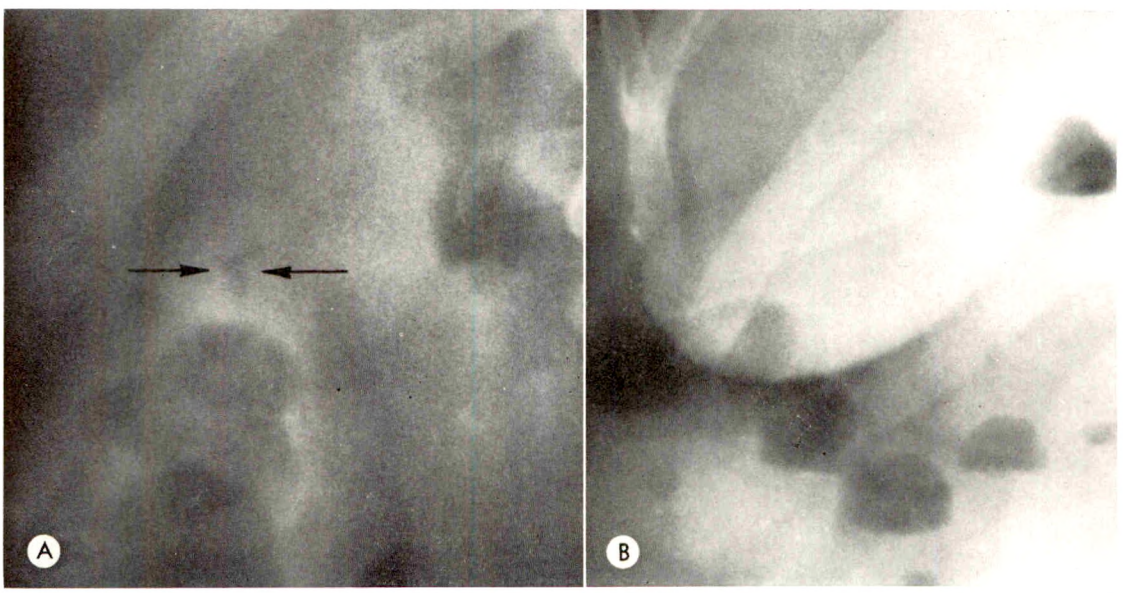


Fig. 2. Case 1. (*A*) Triradiate lucency representing gas fissuring within a nonopaque gallstone is shown (arrows) on plain film roentgenogram. Note its relationship to the hepatic flexure of the colon. Oral cholecystography at this time showed nonvisualization of the gallbladder. (*B*) Gallbladder series 22 months later demonstrates opacification of the organ, without evidence of calculi.

calcifications. There was nonvisualization of the gallbladder. The patient elected to defer surgery and remained asymptomatic. Reevaluation 22 months later with a gallbladder series showed a normally functioning organ without calculi (Fig. 2*B*). No stellate radiolucencies were present. Repeat examination with multiple pressure spot roentgenograms pre- and post-fat stimulation in the decubitus, erect and Trendelenburg positions failed to reveal any evidence of calculus.

Case 11. A 56 year old female was evaluated for an 8 month history of change in bowel habits. During the course of a barium enema study, a distinct Mercedes-Benz sign was observed in the right upper quadrant of the abdomen (Fig. 3, *A* and *B*). An upper gastrointestinal series was intrinsically negative but confirmed the presence of the finding. Oral cholecystography showed nonvisualization of the gallbladder. Intravenous cholangiography demonstrated a normal common bile duct but without opacification of the gallbladder. Cholecystectomy was performed, confirming the diagnosis of chronic cholecystitis with the presence of a single 1.5 cm. mixed facetted calculus. Its specific gravity was not determined but it was noted that the calculus floated in water.

PHYSIOCHEMICAL DYNAMICS OF CHOLELITHOGENESIS

Based on their composition, gallstones have classically been divided into 3 categories: (1) *pure* (*homogeneous*), containing virtually pure cholesterol or pigment; (2) *mixed* (*heterogeneous*), in which two or more components appear in all parts of the stones and there is considered to be no difference between the composition of the center and that of the periphery; and (3) *combination* stones in which the composition of the center differs from that of the periphery. By sophisticated techniques of histochemical analysis, Bogren[5] has recently shown that pigment (amorphous primary and secondary calcium bilirubinate) is the only constituent which appears in all biliary calculi and that it always appears to occur in their centers. These studies lead to the conclusion that pigment is, therefore, the crystallization-nucleus of biliary calculi, all of which are built up in the same way. After formation of the crystallization-nucleus, cholesterol monohydrate and other compounds are precipitated.

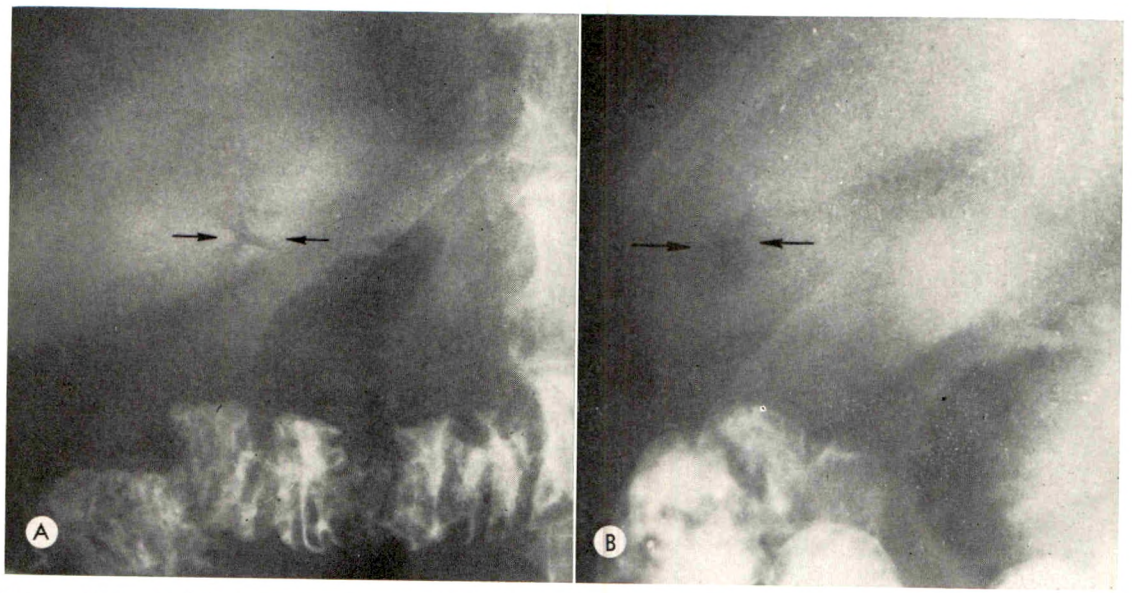


FIG. 3. Case II. (*A*) Frontal projection. (*B*) Oblique projection. Stellate radiolucent fissuring (arrows) with club-shaped terminal enlargements within a nonopaque gallstone. Note the maintenance of its relationship to the hepatic flexure of the colon.

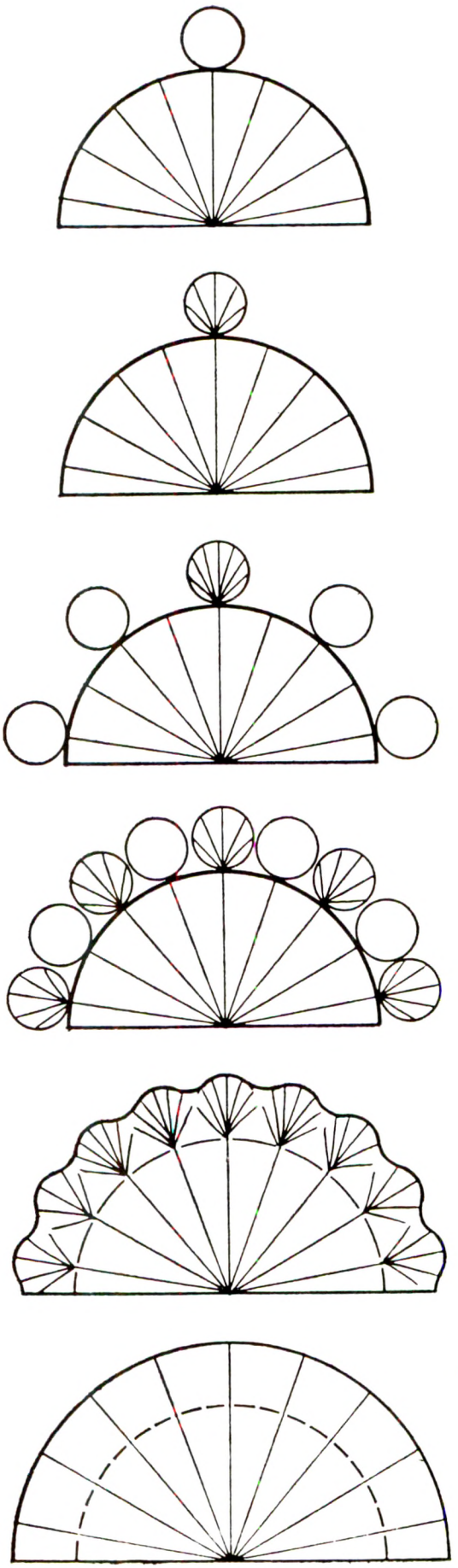
Bile is a supersaturated solution in which cholesterol is adsorbed on the ionic micelle and normally kept from precipitation by bile salts. Derangements in the "critical ratio" of cholesterol to bile salts (generally accepted as 1:13) favor the precipitation of cholesterol. While amorphous at first, this quickly crystallizes into a distinctive radial distribution from the nucleus of the calculus.

This pattern of a typical gallstone formation is in accord with well-established physiochemical principles. Schade[28] has shown the appearance of the radiating crystalline structure of cholesterol spheroliths in a colloid medium. These coalesce, a process that may take from 2 or 3 to several weeks. The spheroliths extend into and adsorb successive adhering spheroids (Fig. 4). The concentric incremental layers of a calculus, between which pigment and other compounds may be trapped, are clearly explained by these dynamics. Another physiochemical process, the "Leisegang" phenomenon,[21] in which substances are rhythmically precipitated about a nucleus in a gel has been invoked. Kleeberg[19] has reproduced many of the structural features of gallstones by this colloidal action. Its role in human cholelithogenesis, however, is uncertain, except perhaps in the presence of overtly infected protein-rich bile.

SIGNIFICANCE OF FISSURING OF GALLSTONES

Fissures within biliary calculi are surprisingly common. While recognized by pathologists for almost 200 years, their actual incidence had not been appreciated until the observations made by Hinkel[17] in 1954. By roentgenographing the unopened gallbladders containing calculi in a series of 100 consecutive cholecystectomies, fissures were found to be present in almost 50 per cent. Usually fluid occupies the fissures, but in 46 per cent the fissures contained some amount of gas. The fluid content, containing traces of sodium chloride, cholesterol and calcium, has been found to be as high as 0.5 cc.[23] Analysis of the gas, which may measure up to 60 mm.^{3}, has shown 6–7 per cent to be carbon dioxide, 0.5 per cent oxygen, and the remainder a noncombustible gas, probably nitrogen.[20]

Kommerell and Wolpers[20] felt that all



biliary calculi pass through a gas-generating phase as a result perhaps of organic decomposition. In the light of further studies and particularly advances in colloidal chemistry, this thesis, however, is no longer tenable. According to Schade,[28] with advancing age of concretions, naturally occurring internal shrinkage results in dendritic cracks extending peripherally, sometimes to the surface. The effect of this dehydration and colloid-crystal shrinkage is the production of "faults" of negative pressure which influence the inward diffusion of fluids or gases dissolved in the surrounding bile. Whether the fissures contain fluid or gas is probably dependent upon the size and negative pressure of the central schism, the extent of the dendritic fissures and the porosity of the calculus.[17]

The rate of precipitation and crystallization is a crucial factor in determining the number, size, shape and structure of the gallstones. This has been observed by many investigators.[5,10,17,19] Rapid precipitation is reflected in multiple, polyhedral and facetted nonopaque calculi of the "mixed" variety having a radial crystal arrangement and concentric colored rings. This type of gallstone constitutes the majority of biliary calculi and it is precisely within this type that fissuring is particularly common. Fissuring has not been observed *in vivo* in the "pure" cholesterol or pigment calculi[17] which are often single and are believed to form more slowly. It is thus apparent that fissure development may be more closely related to the rapid formation of the gallstone than its age.

Autofragmentation of gallstones is a phenomenon which has been recognized for many years. Bauer[3] outlined the lines of force in pyramidal and cube-shaped stones which determine the planes along which

FIG. 4. Formation of a gallstone by spheroid aggregation and change to a radiating crystalline structure.

splitting occurs. Torinoumi[33] first speculated that splitting along the fissures may lead to spontaneous rupture of a gallstone giving rise to a family of small stones. Figure 5 is a roentgenogram of a surgically removed gallstone, demonstrating central gas fissuring extending in a triradiate pattern toward the periphery. Progression of this process is probably the basic factor in spontaneous fragmentation.[17]

The phenomenon of *spontaneous disappearance of gallstones* is being increasingly recognized.[2,11,14] It is likely that chemical or metabolic factors may play a role in the dissolution of some gallstones, particularly the post partum cholesterol calculus. However, the probability of the asymptomatic passage of gallstones of the mixed, essentially nonopaque variety following their spontaneous fissure-fragmentation is clearly supported by multiple observations. Stones up to 5 mm. can traverse the cystic duct without impaction and often without symptoms, and stones of up to 7 mm. can pass through the sphincter of Oddi.[23] In a review of 5,037 surgically treated patients, Glenn[13] reported that stones in the common duct are almost invariably present in patients with small gallstones and a large cystic duct. Richards[27] reported a case in which numerous calcified stones were roentgenologically demonstrated in transit in the cystic duct, common bile duct and intestine. At operation 12 days later, no fistula was found and only 3 stones remained in the common bile duct. Schembra[29] has clearly demonstrated during cholecystography that moderate sized facetted calculi may pass painlessly out of the gallbladder, through the cystic duct and into the common bile duct. In 2 patients, Brodén[7] has roentgenologically documented the disappearance of gallstones containing central gas fissuring and attributed it to disintegration and passage of the concretions. We believe this is what occurred in our Case 1. In recent experiences with several cases of spontaneously disappearing gallstones, the interval between the roentgen examination clearly showing the calculi and surgical confirmation of their absence was as short as 16 days to 1 month. This conflicts with the assumption that the stones might have had time to dissolve and substantiates their likely ductal passage.[11,14]



Fig. 5. Postoperative roentgenogram. Note that the 3 stellate fissures are perpendicular to the midpoints of the sides of the facetted calculus.

The *specific gravity* of gas-containing gallstones, furthermore, appears to predispose to their ductal passage even before the stage of autofragmentation. The usual specific gravity of gallstones is 1.040–1.080,[1] whereas that of bile is generally below 1.040. Gallbladder bile, however, probably never becomes completely homogeneous but tends toward stratification.[8] This has been shown clearly *in vivo* by Tera[32] who documented that the specific gravity of the bile strata within a gallbladder varies between 1.0207 and 1.0358 (Fig. 6). The specific gravity is usually considerably less in pathologic gallbladders with stones. In point of fact, the "layering" phenomenon of multiple nonopaque gallstones floating in opacified bile reflects the equalization of the specific gravity of the calculi with that of a layer of the stratified bile (Fig. 7*A*). We have

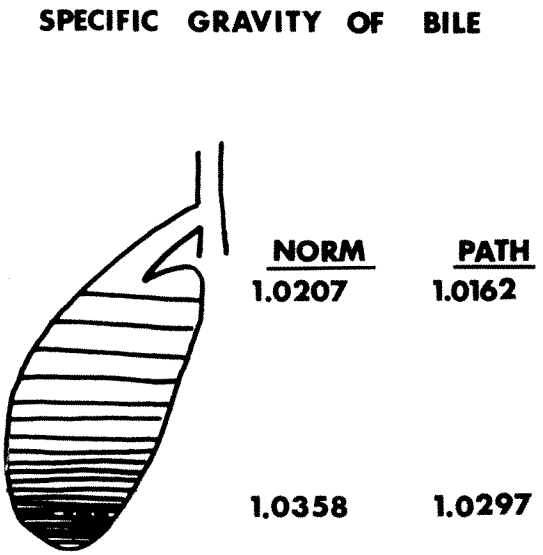


FIG. 6. Stratification of bile within the gallbladder. Note that the layers of least specific gravity reside near the neck and cystic duct.

further observed that when the gallbladder is emptying, the specific gravity of the bile changes, indicating that the less dense bile is discharged first. This is clearly revealed by the ascent of the layer of gallstones on postcontraction roentgenograms (Fig. 7*B*). Gas within fissured gallstones makes their specific gravity unusually low, in the range of 0.822[16] to 1.035.[1] When both fissured and nonfissured calculi are present, it can be demonstrated that all of the fissured stones float higher in the gallbladder than any of the nonfissured calculi.[1] With contraction of the gallbladder, it therefore seems likely that the light gas-containing gallstones may be easily transported with the less viscid bile through the ductal system.

ROENTGENOLOGIC FEATURES AND DIFFERENTIAL DIAGNOSIS

The plain film roentgenographic features of the Mercedes-Benz sign are distinctive. The stellate radiolucent fissures are most frequently widest centrally and radiate like points of a star. At times, club-shaped enlargements mark the terminal parts of the radii (Fig. 3*A*). Usually 3 symmetrically lucent fissures are identifiable, although 1 to 5 may occasionally be present. The radii are not necessarily equidistant, but tend to be of the same length. They are most often perpendicular to the mid-points of the sides, and as a rule they do not extend to the periphery, but occupy the inner two-thirds of the calculus. In this way, the contour and size of a nonopaque calculus can be reconstructed on the plain film roentgenogram. The calculi usually range in size between 0.3 and 1.5 cm.[17,22,31] They are typically multiple, with up to 20 reported,[1] and are invariably facetted.[9,12,17,18] The demonstration of only a single Mercedes-Benz sign in multiple projections is thus distinctly unusual (Cases I and II) and suggests the previous and perhaps asymptomatic passage of other fragmented calculi.

Rarely, some fluid-filled central fissures are radiopaque[20,25,26] due to their calcium content. A faint rim of calcification about the stone may be present,[7,15] but this is often not noticed until the more striking gas-containing fissures are observed.

The lucency of the fissures has persisted on roentgenograms for as long as 7 years. Absence of the gas on follow-up roentgenographic study may indicate either replacement by fluid or fragmentation and passage.

It is apparent from examination of postoperative specimens[1,7,9,17] that gas-filled fissures are a much more common phenomenon in gallstones than is indicated by clinical experience. Small indistinct fissures may be beyond the limits of routine roentgenographic resolution or may be mistaken for an artifact. Even large gas-filled fissures may no doubt escape detection when they have their main extension in a plane at right angles to the central ray. Figure 3*B* illustrates the loss of definition of the characteristic dendritic pattern when not viewed *en face*.

On oral cholecystography, the gallbladder may visualize,[1,7,9] but more often, as in our Case II, does not.[1,7,15] Opacification of the gallbladder directly shows the facetted contour of the calculi. Only rarely are other nonfissured nonopaque stones present.[1,7,15]

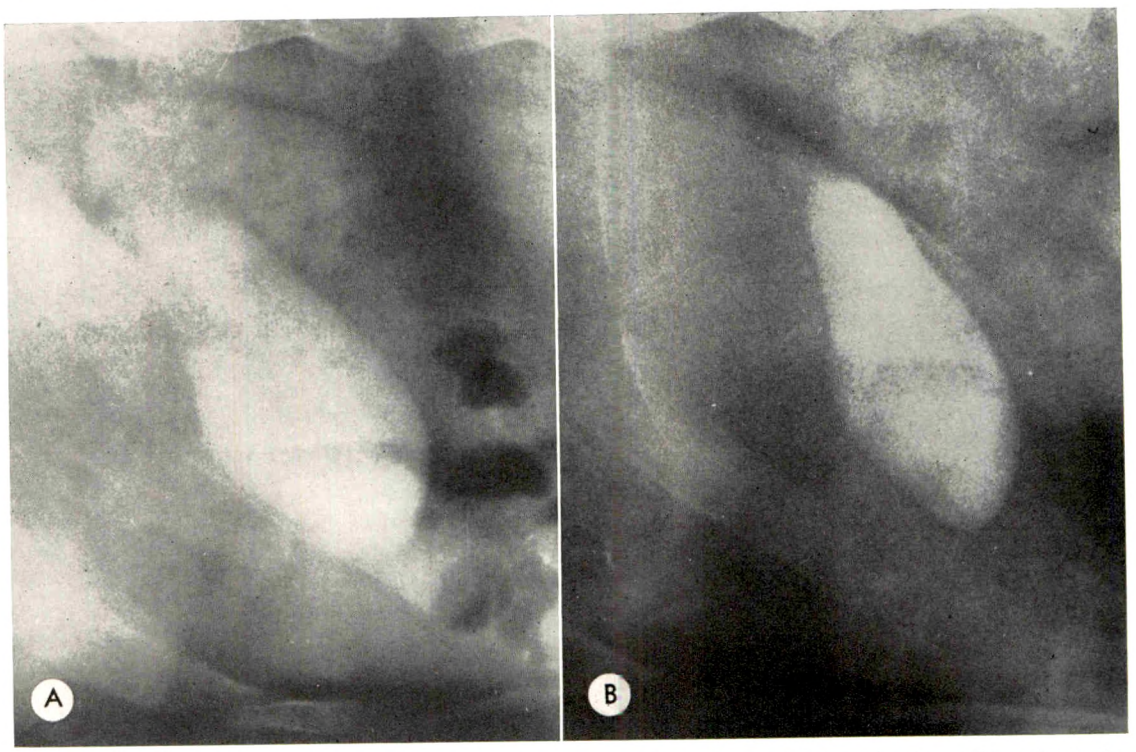


Fig. 7. (*A*) "Layering" phenomenon of multiple nonopaque calculi in decubitus projection. This reflects the multiple strata of bile of differing specific gravity. (*B*) Ascent of layer of calculi after contraction of the gallbladder indicates that the less viscid bile has been discharged.

The differential diagnosis of small discrete localized radiating radiolucencies in the right upper quadrant of the abdomen is limited. Gross collections of gas as may occur in a subhepatic or liver abscess, emphysematous cholecystitis, gas in the biliary, ductal or portal venous system, subhepatic appendix, an abscess in the right kidney or perirenal region and conceivably, gas in an abdominal wall abscess present no problem in localization and identification. Confirmation of the Mercedes-Benz sign on plain film roentgenograms is established by localizing it specifically to the gallbladder area by noting its relationship to the hepatic flexure of the colon and the duodenal bulb or second portion of the duodenum (Fig. 2*A*; and 3, *A* and *B*). In short, stocky individuals with a horizontal stomach and a posteriorly directed duodenal bulb, the *en face* visualization of the pyloric channel ("pyloric star") might simulate some of the features of the Mercedes-Benz sign, but is usually easily identified. The simple ingestion of water replaces the air and obscures the pyloric star. If uncertainty persists, an upper gastrointestinal series conclusively demonstrates the relationships.

SUMMARY

The Mercedes-Benz sign, a distinctive pattern of usually triradiate radiolucent branching, represents the common but often clinically overlooked phenomenon of gas-fissuring within gallstones.

Twenty-eight reports have been collected from the literature. The observation not only permits the plain film roentgenographic diagnosis of nonopaque calculi, but provides an insight into the physiochemical processes of cholelithogenesis.

Two cases are reported and illustrated, in 1 of which spontaneous disappearance of the gallstone occurred.

The correlation of gas-fissuring of gallstones to the dynamics of calculus formation and their autofragmentation and

spontaneous passage through the ductal system is discussed.

Propensity to spontaneous passage also appears related to the specific gravity of bile strata and the influence of contraction of the gallbladder.

Emphasis is placed on the roentgenologic features and differential diagnosis.

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ROENTGENOLOGIC FINDINGS IN RECURRENT PYOGENIC CHOLANGITIS*

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IN Asia the pattern of benign biliary tract disease differs from that reported from Western countries. The majority of patients suffer from a disease similar to the Western type of cholelithiasis, but a smaller group have a disease of a different pattern. Although individual reports differ in detail they are all strikingly similar in describing a type of cholangitis which is characterized by recurrent attacks of fever, chills and rigors, abdominal pain and jaundice and in which grossly dilated extra and intrahepatic ducts are found to contain soft pigment stones, biliary mud or pus. Culture of the bile shows a pyogenic organism which is almost invariably *E. coli*, and this organism is usually grown from blood culture in severe cases.

The disease is rare in Africa,[10] but large series have been reported from Hong Kong, Taiwan, Japan, Malaya, Singapore and Korea. The disease has also been found in emigrants from these countries in America, Britain, Australia and Ceylon.

The various authors have called the condition recurrent pyogenic cholangitis,[1,7] oriental cholangitis,[6] oriental cholangiohepatitis,[9] and biliary obstruction syndrome of the Chinese.[4]

In the acute or fulminating case, correct diagnosis is of great importance as emergency decompression of the biliary tree may be the only therapy to prevent a fatal outcome. In all cases a full assessment of the extent of the disease is necessary to plan the most effective form of surgery. In addition to biliary exploration and removal of stones, surgery may include hepatic lobectomy, biliary bypass or hepatodocholithotomy. It is in this assessment that radiology plays a major role.

Recurrent pyogenic cholangitis has definite roentgenologic features which are described in this paper.

MATERIAL AND METHOD

In the 5 year period since the opening of the University Hospital in 1967 there have been 243 patients with nonmalignant biliary disease, of whom 87 (36 per cent) were diagnosed on clinical grounds or by operative findings to have recurrent pyogenic cholangitis. The 87 patients consisted of 62 Chinese, 13 Malays, 11 Indians and 1 Eurasian. The average age was 50 years. There were 51 males and 36 females, making a male: female ratio of 1.4:1.

In all 87 patients a plain abdominal roentgenogram was taken; cholecystography was performed in 30 patients; intravenous cholangiography with tomography in 12 patients; operative cholangiography in 38 patients; T-tube cholangiography on the 10th postoperative day in 75 patients.

RESULTS

PLAIN ABDOMINAL ROENTGENOGRAPHY

The findings are given in Table 1. Aerobilia was seen in 3 patients, but in these patients there was no evidence of choledocho-duodenal fistula and the infecting organism was no different from that found in other cases.

TABLE I

PLAIN ABDOMINAL ROENTGENOGRAPHIC FINDINGS IN RECURRENT PYOGENIC CHOLANGITIS

Number	Gallstones		Aerobilia	
87	7	8%	3	3%

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TABLE II

NONOPERATIVE ROENTGENOLOGIC FINDINGS IN RECURRENT PYOGENIC CHOLANGITIS

Examination	Number	Non-function		Stones		Dilated Ducts		Normal	
Cholecystogram	30	24	80%	4	13%	0		2	7%
Intravenous cholangiogram	12	6	50%	2	17%	3	25%	1	8%

CHOLECYSTOGRAPHY AND INTRAVENOUS CHOLANGIOGRAPHY

The findings are given in Table II. These investigations gave limited information, as in 80 per cent of the cholecystograms the gallbladder failed to function and in 50 per cent of the intravenous cholangiograms the biliary tree was not visualized.

OPERATIVE AND T-TUBE CHOLANGIOGRAPHY

Strictures, stones and dilatation of the ducts are the characteristic features on cholangiography of recurrent pyogenic cholangitis and the findings are given in Tables III and IV. The term common duct has been used to include the common bile duct and common hepatic duct. The most frequently observed feature in the T-tube cholangiogram was a dilated common bile duct above the normal limit of 12 mm. with an average diameter of 21 mm. Dilatation and strictures of the left hepatic duct were more commonly seen than of the right hepatic duct. Stones were found more frequently in the common duct than in the hepatic ducts.

Most patients showed evidence of the disease in the whole of the biliary system, but in some the disease was restricted to the common duct and left hepatic duct or common duct alone. The localization of the disease is shown in Table V.

DISCUSSION

The first description of the disease was made by De Langen,[3] who observed that

TABLE III

OPERATIVE CHOLANGIOGRAPHIC FINDINGS IN 38 PATIENTS WITH RECURRENT PYOGENIC CHOLANGITIS

Site	Number Filled	Strictures		Stones		Dilatation	
Common duct	38	13	29%	8	21%	29	76%
Right hepatic duct	37	12	32%	1	3%	21	57%
Left hepatic duct	34	19	56%	1	3%	28	82%
Right hepatic radicles	30	5	17%	0		3	10%
Left hepatic radicles	18	6	33%	0		6	33%

TABLE IV

T-TUBE CHOLANGIOGRAPHIC FINDINGS IN 75 PATIENTS WITH RECURRENT PYOGENIC CHOLANGITIS

Site	Number Filled	Strictures		Stones		Dilatation	
Common duct	75	35	47%	17	23%	66	88%
Right hepatic duct	73	33	45%	6	8%	37	51%
Left hepatic duct	72	45	63%	10	13%	54	75%
Right hepatic radicles	72	30	42%	4	6%	12	17%
Left hepatic radicles	62	33	53%	4	7%	21	34%

in the natives of the Dutch East Indies, now Indonesia, pigment stones low in cholesterol were found in the hepatic ducts. Stock and Tinckler[9] from Hong Kong suggested that the disease was due to blockage of the bile ducts by infestation with the liver fluke *Clonorchis sinesis* complicated with an *E. coli* infection, but this cannot be the cause in Malaya as *Clonorchis* infestation does not occur due to the absence of a suitable snail as intermediate host.[5] Ong[7] thought that portal bacteremia played an important part and reproduced features of the disease experimentally in rabbits by tying the common bile duct and injecting *E. coli* into the portal vein. Harrison-Levy[4] suggested that there was a physiologic block at the lower end of the common bile duct resulting in dilatation of the biliary tree with subsequent stagnation with infection of the bile and stone formation. Wen and Lee[11] postulated that intrahepatic stones may be caused by metabolic derangement due to dietary deficiency.

The disease may be difficult to diagnose, particularly when there is primary cholangitis without the florid picture of strictures and stones being present, or where there is long standing calculous or other disease of the ducts with an element of secondary infection producing some features of recurrent pyogenic cholangitis. Cunningham and Yong[2] have suggested that the advanced disease may develop in these situations, but it may not be possible to identify which condition occurred initially.

The sex incidence of the Western type of biliary disease is 1 male:2 females, but this ratio is altered in recurrent pyogenic cholangitis with the disease occurring more frequently in males. The disease also occurs at a younger age and patients in their third and fourth decades may be seriously affected. The high number of Chinese in this series reflects the fact that the University Hospital serves a largely Chinese population. King[6] has shown in a large survey that Malays and Indians are more prone to biliary disease than Chinese.

Table V

Localization of Disease from the T-tube Cholangiogram in 75 Patients with Recurrent Pyogenic Cholangitis

Site	Per Cent
Common duct, right and left hepatic ducts	58
Common duct and left hepatic duct	19
Common duct	17
Left hepatic duct	4
Common duct and right hepatic duct	1
Right and left hepatic ducts	1

The commonest finding on the T-tube cholangiogram was a dilated common bile duct. However, contrast medium could be observed on fluoroscopy to flow into the duodenum and, as in nearly all these patients instruments were freely passed through the ampulla of Vater at the time of operation, it is unlikely that any obstruction was present. Wen and Lee[11] also found that the sphincter of Oddi was usually patulous with no evidence of obstruction at that site. This incompetence of the sphincter of Oddi may allow reflux of ingested air from the duodenum into the common bile duct and so accounts for the aerobilia (Fig. 1) which was seen in 3 patients (Table 1). In these patients there were no gas forming organisms infecting the biliary system and no evidence of a choledocho-duodenal fistula to cause the aerobilia.

As the primary site of the disease is in the hepatic and common bile ducts, cholecystography is of little help in establishing the diagnosis and in 80 per cent of the cholecystograms the gallbladder failed to function (Table II). In 50 per cent of the intravenous cholangiograms the biliary tree failed to opacify which is probably due to the ducts being so dilated that the contrast medium has become too dilute for visualization. However, in most cases only 20 ml. Biligrafin was used but with larger doses better roentgenograms might be obtained.

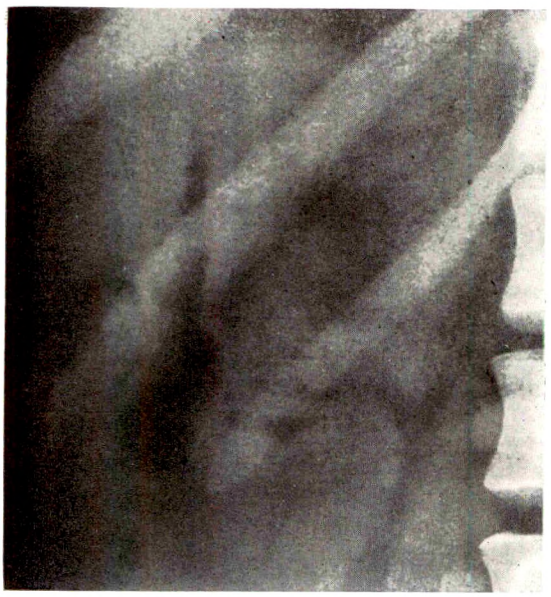


FIG. 1. Aerobilia. Plain abdominal roentgenogram of 25 year old Malay female with recurrent pyogenic cholangitis showing air in a dilated biliary system.

The diagnosis of recurrent pyogenic cholangitis is established at cholangiography when the findings in the biliary tree affecting both the intra and extrahepatic parts are strictures, stones and dilated ducts (Tables III and IV). Strictures and stones were better demonstrated on the postoperative T-tube cholangiogram than on the operative cholangiogram, where the film quality is poorer due to use of a portable roentgenographic unit and a stationary grid, absence of fluoroscopy and difficulty in positioning the patient to fill the left hepatic duct. Although only 38 patients had operative cholangiography, it is now standard practice to perform this procedure during all operations on the biliary tree. This is important as unsuspected localized disease may be demonstrated which may be severe enough to necessitate immediate resection. In milder cases the clinician will be alerted to undertake careful follow-up of the patient. The surgeon must ensure that the left hepatic duct is fully demonstrated. To this end the operating table may need to be rolled to the left and tilted head down, sufficient contrast material, which may be in excess of 60 ml., must be injected and digital compression on the common bile duct may help to fill the intrahepatic radicles. If the left hepatic duct cannot be filled by these maneuvers, it may be necessary to obtain a transhepatic cholangiogram by passing a catheter and injecting contrast material directly into the left hepatic duct.

Strictures, stones and dilatation occur more commonly in the left hepatic duct than the right (Table V), which may be due to poorer drainage of the left hepatic duct, as it arises more obliquely from the common hepatic duct. In patients with mild disease the left hepatic duct may show the changes of recurrent pyogenic cholangitis, whereas the right hepatic duct is much less affected or it may be normal (Fig. 2). In advanced cases the whole of the biliary tree is dilated, with multiple strictures, and may be filled with stones

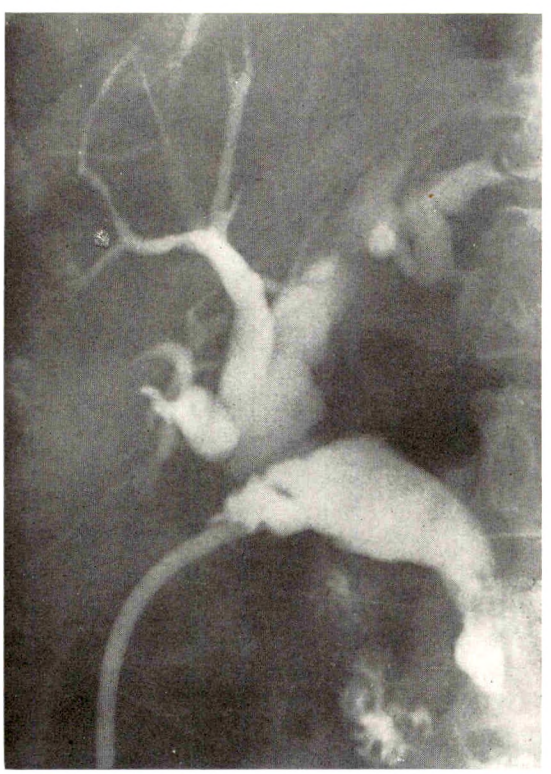


FIG. 2. Left hepatic duct more affected than the right. T-tube cholangiogram in a 58 year old Chinese woman showing strictures and dilatation of the left hepatic duct, whereas the right is much less affected. The common bile duct is also dilated.

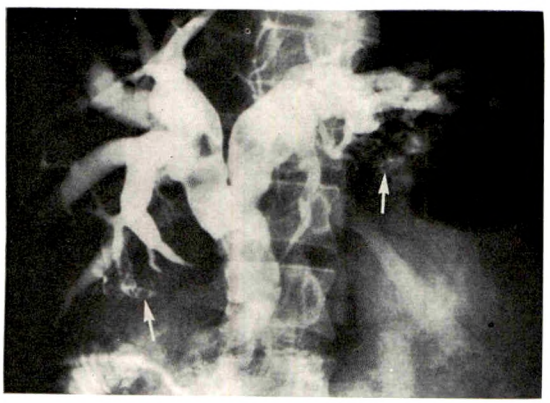


Fig. 3. Advanced disease. T-tube cholangiogram in a 22 year old Malay male showing the whole biliary system dilated with multiple strictures and filled with stones. The arrows indicate intrahepatic stones.

(Fig. 3). In 4 cases abscess formation was seen in the liver.

Strictures may occur anywhere in the biliary tree (Fig. 4), and on occasion may be associated with marked dilatation (Fig. 5). Stones can form and may be held up behind a stricture (Fig. 6).

The stones commonly seen in recurrent pyogenic cholangitis are usually multiple soft pigment stones with pigment flakes and aggregations of mud which may be found only in the biliary ducts and may not be present in the gallbladder (Fig. 7). Stones are often difficult to remove at operation, so retained stones may be seen on the T-tube cholangiogram. These stones are more difficult to demonstrate roentgenologically than the larger more discrete cholesterol stones seen in the Western type of cholelithiasis. Early films are exposed in T-tube cholangiography using a small amount of dilute contrast material. The very large volumes of contrast material which are sometimes necessary to fill the dilated biliary system may otherwise obscure the calculi. To fill the left hepatic duct, it may be necessary to inject the con-

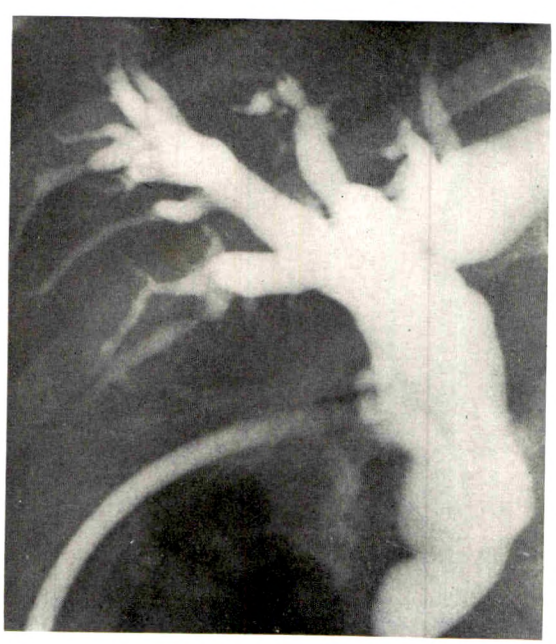


Fig. 4. Strictures. T-tube cholangiogram in a 52 year old Chinese female showing strictures in the right hepatic duct and hepatic radicles. The left hepatic duct and common bile duct are also dilated.

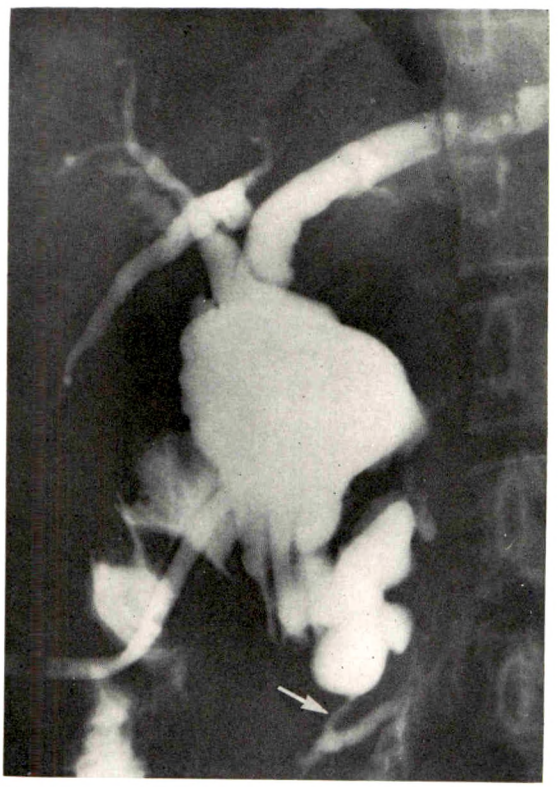


Fig. 5. Grossly dilated common bile duct. T-tube cholangiogram through a choledocho-jejunostomy in a 19 year old Chinese female showing multiple strictures in the common bile duct with a marked stricture at its lower end (arrow). Reflux into the pancreatic duct has occurred. The left hepatic duct is more dilated than the right.



FIG. 6. Intrahepatic stones. T-tube cholangiogram in a 28 year old Malay male showing a stricture at the origin of the right hepatic duct (arrow) and stone formation distal to it.

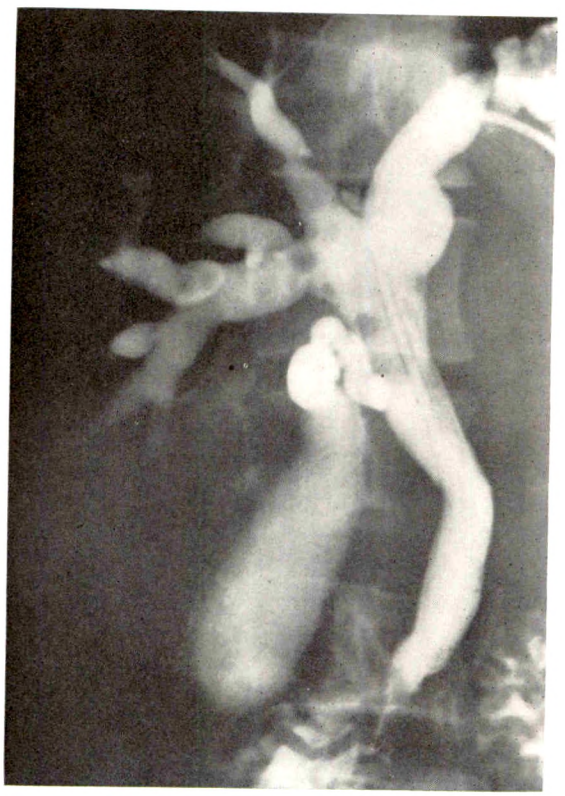


FIG. 7. Normal gallbladder. Postoperative transhepatic cholangiogram in a 32 year old Chinese male showing stones in the right and common hepatic ducts, but none in the gallbladder.

trast material with the patient in a steep left oblique or left lateral position. A delayed roentgenogram will show retention of contrast medium in the biliary system and, if the common duct is dilated, it is important to observe on the fluoroscopic screen whether there is free flow of contrast material into the duodenum.

CONCLUSION

The diagnosis of recurrent pyogenic cholangitis may be suspected on the plain abdominal roentgenogram by the presence of aerobilia.

Cholecystography is of little help in establishing the diagnosis.

The characteristic changes seen on cholangiography, which are due to infection of the biliary system, are strictures, stones and dilatation of the hepatic and common bile ducts with the left hepatic duct more affected than the right.

SUMMARY

The roentgenologic features in 87 cases of recurrent pyogenic cholangitis are described.

The characteristic findings on cholangiography are strictures, stones and dilatation of the hepatic and common bile ducts.

The importance of establishing the diagnosis is stressed and the special techniques in operative and T-tube cholangiography to give the maximum information are outlined.

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POSITION OF THE DUODENAL BULB AND LIVER SIZE*

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RADIOLOGISTS have long been intrigued by the possibility of determining liver size by radiographic methods. Various techniques have been tried, ranging from subjective estimates based on intuitive evaluation of the plain film abdominal roentgenogram to sophisticated mathematical formulas derived either from film measurements[4,12,20,21,23] or vascular injections[3,7,12] (arterial or venous) or diagnostic pneumoperitoneum,[9,12] positive contrast peritoneography,[6] or isotope scanning.[17,19] When thorotrast was in vogue, the liver could be seen[12] because the radiopaque and radioactive colloid was concentrated in the reticuloendothelial cells of the organ.

Some of these methods have been more successful than others, but all have their drawbacks. Angiography and peritoneography would seem to be the most promising for accuracy, but they cannot be done simply and in volume, or they entail some morbidity and should probably be reserved for specific indications. Liver scanning is fairly accurate but it is an expensive study. Thorotrast, because of its long effective half life is not, and should not be used but there are occasional patients still alive who have the material irreversibly attached to their livers. Roentgenograms of the abdomens of these patients are quite striking (Fig. 1, *A* and *B*).

Observations made from the plain film roentgenogram have to take into account that the liver is a multifaceted organ which can be spatially oriented in the body in an almost infinite number of ways. Often that portion visible on the anteroposterior roentgenogram thought to represent the inferior liver edge actually lies posteriorly in the abdomen, whereas that part the clinician would palpate is anterior and presents a thin tapering edge to the roentgen ray beam (Fig. 1, *A* and *B*). This, we believe, is the reason for the disparity that exists between clinical estimation of hepatomegaly and plain film roentgenography. It also offers one explanation of the apparent "large liver" described by the radiologist which cannot be confirmed by palpation, and conversely it simultaneously explains the large liver edge which may be "flipped" by the clinician, but is not seen on the roentgenogram.

Any study correlating palpation and roentgenograms which fails to take this spatial orientation into account cannot be valid. It could be likened to the fable of the blind men examining the elephant. The paw and the tail indeed represent parts of the elephant but certainly do not indicate the size of his ears or trunk.

Whatever objective parameter one utilizes to estimate liver volume, enlargement is best determined by correlating size with patient surface area.[2,10] Unlike our pediatrician colleagues, however, most radiologists have not been indoctrinated in the use of nomograms for converting height and weight into square meters.

Displacement of adjacent viscera by an enlarged mass of liver has been used and probably comprises the major part of the Gestalt or pattern approach to evaluating hepatomegaly.[8,13,14,16,18] We do not intend to denigrate this approach by any means. In fact, it is often quite effective. However, while neighboring organ displacement by increasing liver volume has long been appreciated as a sign of enlargement, little has been written about the converse; *i.e.*, displacement of adjacent viscera by con-

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tracting liver mass.[5,14,15,18] Displacement of the duodenal cap in both of these directions comprises the basis of the present study.

The liver bears a constant relationship to the duodenum, and a suspensory ligament, the hepatoduodenal ligament, ties these structures into an anatomically integrated unit. Therefore, enlargement of the liver would be expected to displace the duodenal bulb down and away from its usual position, and shrinkage of the liver should retract the bulb upward. The following study was performed to test this hypothesis.

MATERIAL AND METHOD

We reviewed prone Bucky roentgenograms from the upper gastrointestinal series of 151 adult patients, 103 of whom had some objective measurement of liver size recorded in their charts. Autopsy weight, liver scan, splenoportogram, or surgery was accepted as a reasonable objective parameter. Enlargement of the liver by clinical palpation was accepted, if 2 or more senior physicians had unequivocally agreed that hepatomegaly of significant degree was present. This latter is perhaps the least objective criterion, but in the hands of an astute clinician, 4 finger-breadths enlargement has been shown to be reliable at least 85 per cent of the time.[22]

Gastrointestinal series in 47 randomly selected adult patients were also evaluated and were considered to be part of the control group; 32 other patients with normal liver size by objective criteria comprise the remainder of the control group. Since surface area was not available for our patients, absolute liver weights were used. A liver less than 1,200 grams was considered small, 1,200 to 2,000 grams was deemed within normal limits, and a liver over 2,000 grams was considered enlarged.[11]

RESULTS

We found that in 74/79 patients (93 per cent) (Fig. 2, *A* and *B*) in the control group, the duodenal cap lay: (a) at or below the twelfth rib on the right; (b) to the right of the midline; and (c) above the superior border of L3. In only 1 of the 79 was the bulb above the twelfth rib, and in this case the patient was a huge individual whose liver weighed 1,630 grams, but it was cirrhotic. His spleen weighed 940 grams. Four cases were below the superior border of L3 or to the left of the midline. For 2 we have no explanation as they were from the randomly selected group of patients. On the other hand, the other 2 patients' livers were in the 1,600 to 2,000 gram range, bordering on enlargement, in patients of average body build.

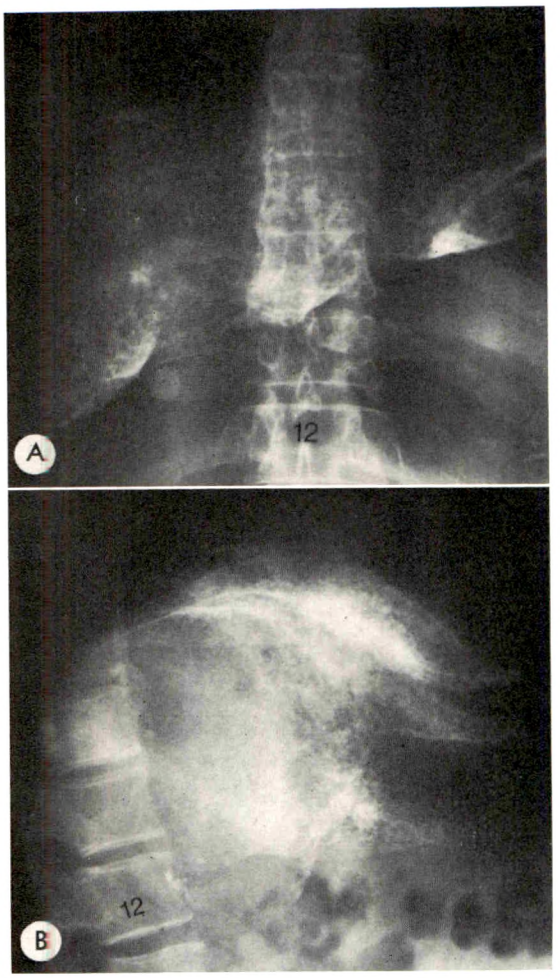


Fig. 1. (*A* and *B*) Abdominal roentgenograms with hepatic retention of Thorotrast. Note that the lowest part of the liver is posteriorly located. The anterior edge is high and presents a thin surface to the roentgen-ray beam. The spleen is well seen.

In 27 patients whose liver was small by

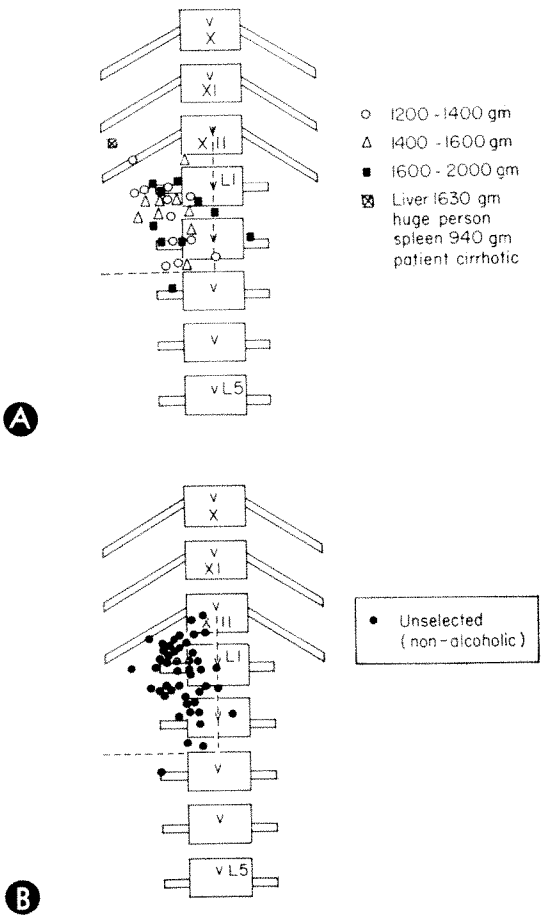


FIG. 2. Location of the duodenal cap in the control series. (*A*) Location of the cap in relation to normal size liver in 32 patients. (*B*) Location of the cap in 47 upper gastrointestinal examinations of randomly selected, nonalcoholic, noncirrhotic patients.

objective standards (Fig. 3), the duodenal bulb lay above the twelfth rib in 13 instances. Two patients with demonstrably small livers were well below the lower defined limits. Both of these exceptions demonstrate important pitfalls to be avoided. One patient whose bulb was at the inferior border of L_4 at the midline, strikingly demonstrates how important it is to correlate liver mass with body size. This particular patient weighed only 94 pounds, yet her liver at autopsy weighed 1,115 grams, small by absolute criteria, but large relative to total body mass. The other patient whose bulb was low-lying had marked

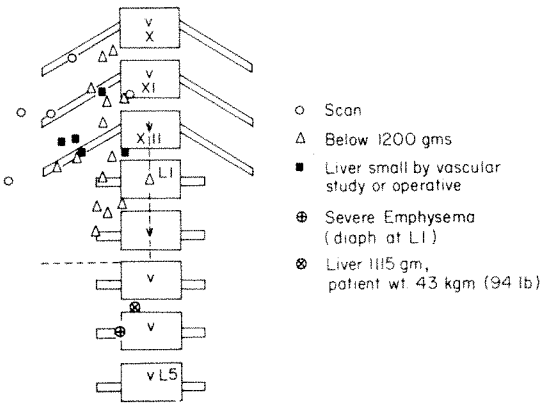


FIG. 3. Location of the duodenal cap in 27 patients whose livers were considered small by objective criteria.

pulmonary emphysema with flattened, almost inverted, diaphragms.

The cap of another patient with a minuscule liver that weighed only 750 grams lay at the midline, overlying the midportion of the body of L1 (Fig. 5*A*). She had massive ascites which undoubtedly insinuated itself between the liver and the flank and displaced the whole aggregate medially and downward (Fig. 5*B*).

Among the 44 patients with a large liver (Fig. 4) we found the duodenal bulb displaced below the superior border of L_3 and/or across the midline in 15. Interestingly, one cap was located above the level of the twelfth rib. At first blush, this would

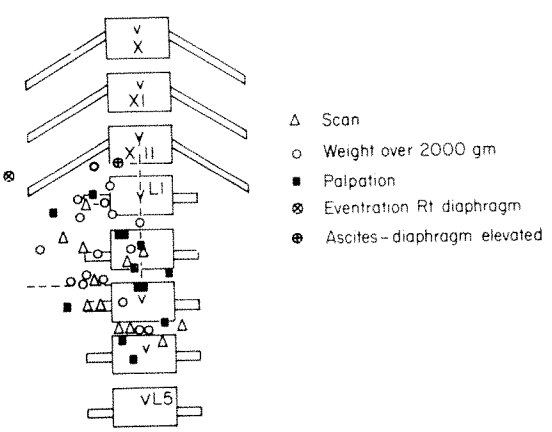


FIG. 4. Location of the duodenal cap in 44 patients whose livers were considered large by objective criteria.

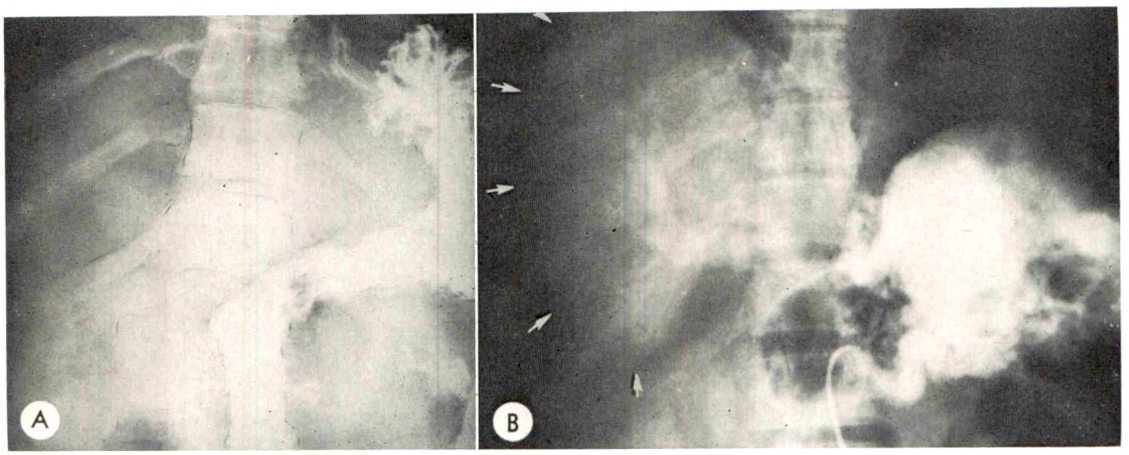


Fig. 5. (*A*) The cap is to the left of midline at L1 in this patient with a 750 gram liver (retouched). (*B*) Splenoportogram shows a shrunken liver (arrows) displaced to the left by massive ascites.

seem to be a marked paradox. A moment's reflection, however, would suggest that anything that elevates the diaphragm should have this effect. Indeed, this patient had marked diaphragmatic eventration.

Several other cases demonstrate progressive shift in duodenal cap position with alteration in liver size. For example, 1 had nutritional cirrhosis with a progressively shrinking liver. The bulb in 1962 was at the base of the right transverse process of L1; by 1965 the bulb had migrated above the twelfth rib.

Figure 6, *A* and *B* is an excellent graphic demonstration of the close relationship between the bulb and the liver. The cap is located above the twelfth rib in the gastrointestinal study. Liver scan shows a very small liver and huge spleen. Furthermore, the scan clearly depicts a defect into which the duodenal bulb snugly fits. The patient had cirrhosis of the liver.

Several other examples in our series demonstrate progressive displacement in the opposite direction. In 1 it is noted that, in 1963, the bulb was at the level of the midline of L1. Between 1963 and 1969 the bulb shifted down from that position to the superior border of L3. At that time the

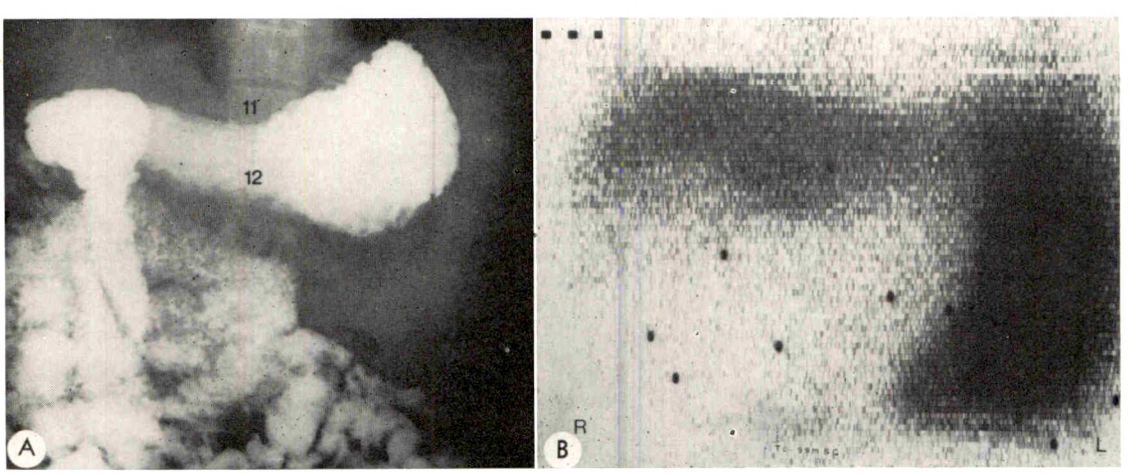


Fig. 6. (*A*) Marked elevation of the duodenal cap is dramatic in this patient whose liver is small and spleen is big. (*B*) The scan shows a small liver with the defect of the cap on its inferior surface.

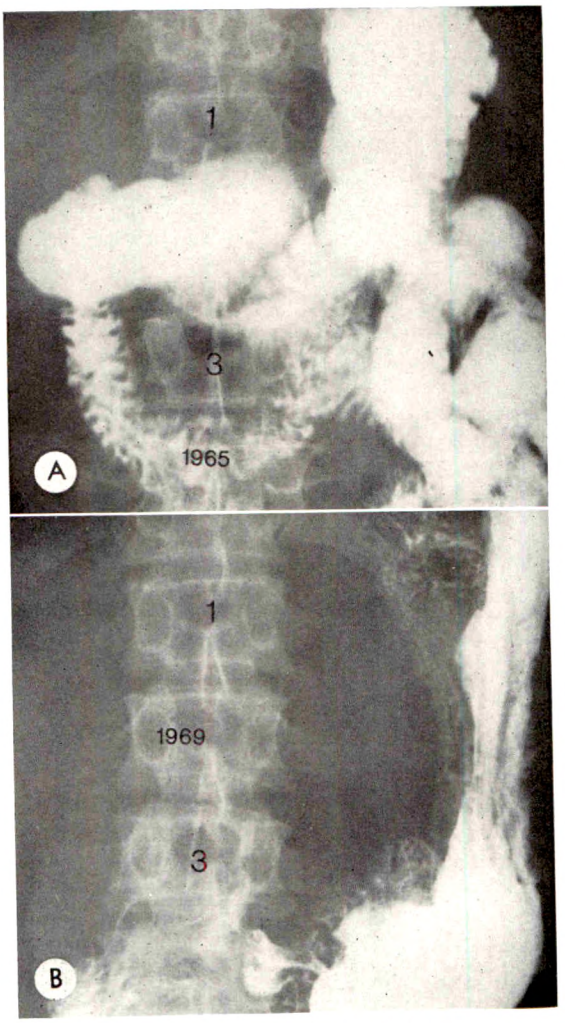


FIG. 7. (*A* and *B*) Downward shift of bulb and stomach is seen here in 2 gastrointestinal examinations made 4 years apart. Liver metastasis from bronchogenic carcinoma was the cause.

liver was clinically palpable 4 fingerbreadths below the right costal margin, and was firm and nontender. Multiple small defects were described in the liver scan and "filling defects" were noted in the fundus of the stomach. The patient was lost to follow-up and the final diagnosis is not known, but hepatomegaly is unquestioned, probably due to metastasis.

Another example is illustrated in Figure 7, *A* and *B*. Striking downward migration of the bulb from the inferior border of L1 laterally, to the superior aspect of L4 medially occurred between 1965 and 1969. Hepatic enlargement was due to metastases from bronchogenic carcinoma.

Figure 8, *A* and *B*, demonstrates fully the relation of the duodenum to the hepatic and gastroduodenal artery and the liver. The patient, a 54 year old woman, had abdominal discomfort and right upper quadrant abdominal mass. The upper gastrointestinal study showed displacement of the duodenum, with the duodenal bulb at the level of the lower edge of L3 just to the right of the midline. The right hemidiaphragm was elevated. A large area of

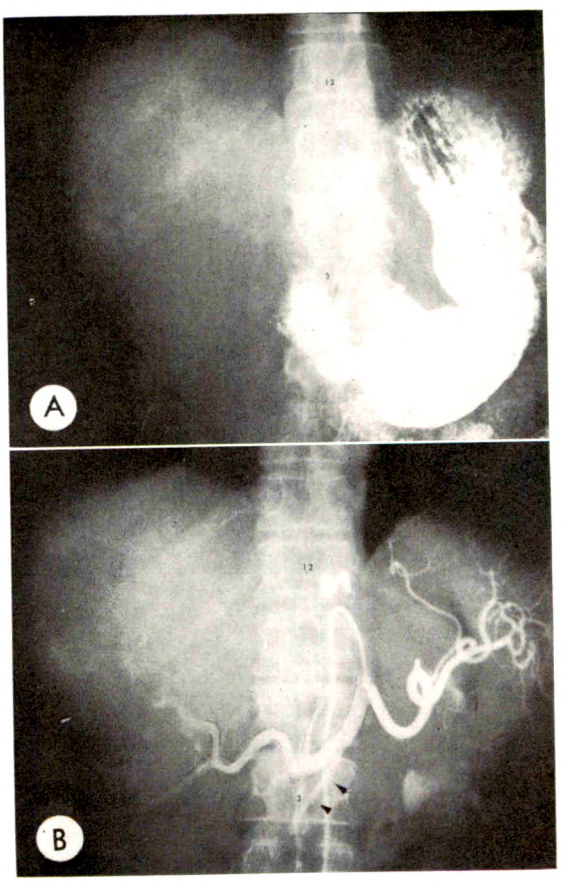


FIG. 8. Metastatic colloid carcinoma to the liver. Primary site was colon. (*A*) Upper gastrointestinal study shows marked displacement of the duodenal bulb downward and to the left. Large area of amorphous calcification in the right lobe of the liver. (*B*) Selective celiac angiography, arterial phase (retouched), showing displacement of the hepatic artery downward and to the left. The gastroduodenal artery is displaced to the left of midline (arrowheads).

amorphous calcification was seen in the right lobe of the liver. Liver scan with Tc^{99m} sulfur colloid verified hepatomegaly with a large area of absent uptake in the right lobe, corresponding to the area of amorphous calcification seen on roentgenograms. Selective celiac angiography showed downward and leftward displacement of the hepatic artery. The origin of the gastroduodenal artery was displaced to the left of the midline and downward. The venous phase of the study showed similar displacement of the portal vein and its tributaries.

At operation, the liver was seen to be huge and contained a large metastatic deposit of colloid carcinoma in its right lobe. The primary site was the colon.

DISCUSSION

Elevation of the bulb above the level of the twelfth rib in the absence of a high diaphragm was consistently found in patients who had a small liver. If one also excludes a mass pushing the gastrointestinal tract from below, this cephalic position of the bulb strongly suggests a small liver. Normal bulb position does not exclude shrunken liver.

As Whalen *et al.*[24,25] have pointed out in their discussions of vector forces operative in intraabdominal viscus displacement, one can predict with fair success the originating locus of a mass by the direction of adjacent visceral shift. We believe that our study demonstrates that, when hepatic enlargement displaces the duodenal bulb, it frequently does so in an inferior and medial direction. Displacement of the bulb out of the limits defined and in the direction indicated therefore suggests to us a large liver. Obviously, other appropriately placed extrahepatic masses or fluid could do this. Absence of displacement in no way excludes hepatomegaly. Paradoxically, the duodenal bulb will be above the twelfth rib in occasional patients with a large liver. Eventration of the diaphragm, diaphragmatic paralysis, or herniation is usually the cause.

It is not our intent to claim the discovery of a new and infallible sign for liver enlargement or shrinkage. We do believe, however, that a "rough and ready" estimation of liver size can be made in any gastrointestinal series if: (1) the bulb is displaced beyond the limits we have defined—the greater the displacement the more likely it is to be reliable; (2) there is no abnormality of diaphragm position; (3) the size of the patient is taken into account; and (4) other causes of displacement are excluded.

SUMMARY

The barium filled duodenal bulb on the prone Bucky roentgenogram is most frequently located at a level below the twelfth rib, to the right of the midline and above the upper border of L3.

Displacement above this area strongly suggests a small liver, whereas displacement below and to the left suggests hepatomegaly.

Exceptions are discussed.

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DISPLACEMENT OF DUODENUM BY AN ENLARGED LIVER*

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ROENTGEN findings of an enlarged liver in upper gastrointestinal series include: mass in the right upper quadrant of the abdomen; displacement of the stomach to the left and posteriorly; and downward displacement of the hepatic flexure of the colon.[1–6] Displacement of the duodenum by hepatomegaly is not a usual finding, but must also be included as a diagnostic sign.

It has been stated that the duodenum should not be significantly displaced by hepatomegaly.[2–4]

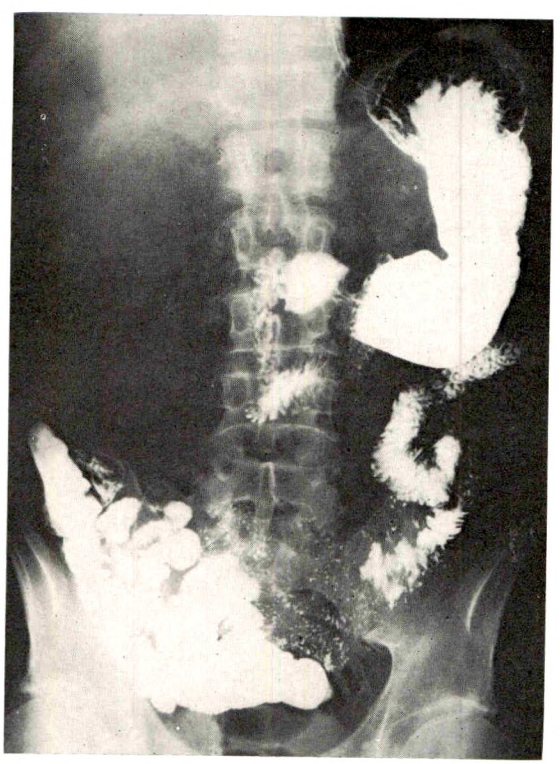


FIG. 1. Case 1. A 66 year old white male with widespread metastatic carcinoma of the oropharynx and palpable liver. An upper gastrointestinal series shows a marked left side displacement of the descending portion of the duodenum by an enlarged liver.

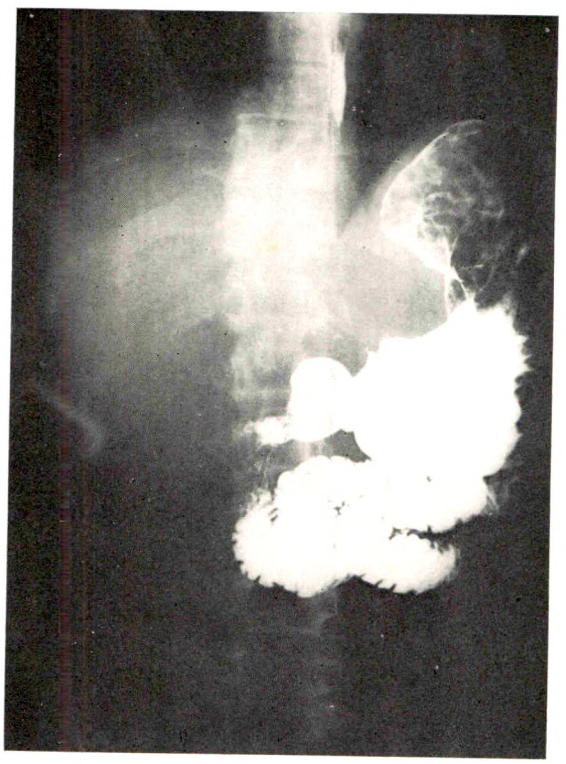


FIG. 2. Case 11. A 60 year old white male with known idiopathic hemochromatosis. Widespread hepatoma was found at autopsy. An upper gastrointestinal series demonstrates a marked left side displacement of the duodenum by an enlarged liver.

Whalen *et al.*[5,6] point out that the duodenum may be displaced by an enlarged caudate lobe of the liver.

MATERIAL

We have encountered 7 cases of varying degrees of left side displacement of the duodenum by an enlarged liver in the past several months (Fig. 1 through 7) and feel

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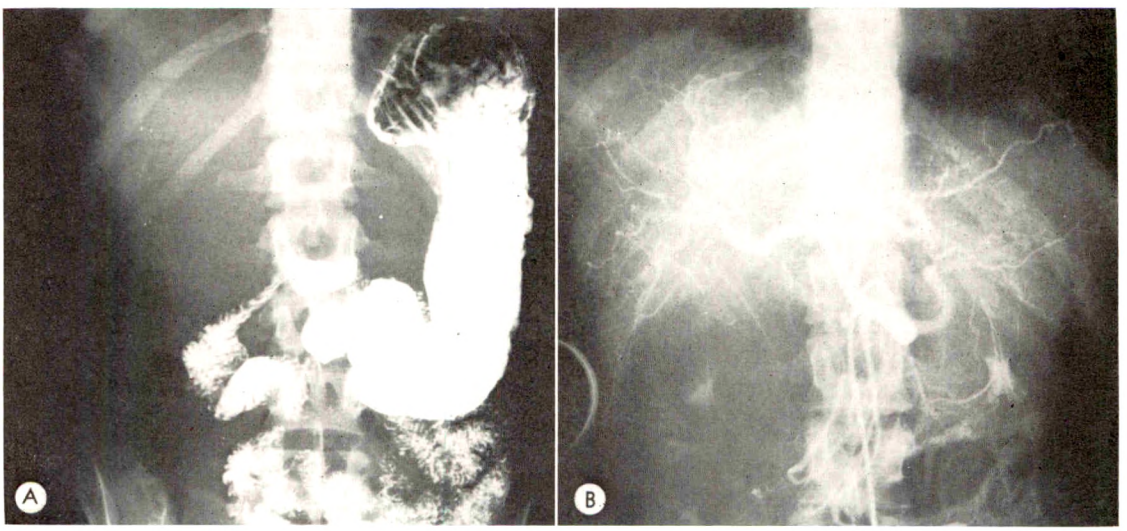


FIG. 3. Case III. (*A* and *B*) A 24 year old black female with complaints of 14 pounds weight loss and abdominal pain. Abdominal angiogram shows a large avascular mass in the right lobe of the liver (*B*).

that this, in fact, is not an infrequent finding as has been thought in the past.

DISCUSSION

Displacement of the gastrointestinal tract by an enlarged liver or mass in the liver was well summarized by Nice *et al.*[3]

The roentgenographic findings include:

A. Mass in the right lobe of the liver
   1. Displacement of the stomach to the left
   2. Depression and posterior displacement of the hepatic flexure of the colon
   3. Depression of the transverse colon
   4. No displacement of the second portion of the duodenum

B. Mass in the left lobe of the liver
   1. Marked left side displacement of the stomach
   2. Downward displacement of the splenic flexure
   3. Anterior displacement of the transverse colon
   4. No significant displacement of the second portion of the duodenum.

It has been the concept[2–4] that the duodenum, a retroperitoneal organ, should not be significantly displaced by an intraperitoneal mass such as an enlarged liver. However, it is not an infrequent finding that an intraperitoneal mass or organ enlargement will displace a retroperitoneal

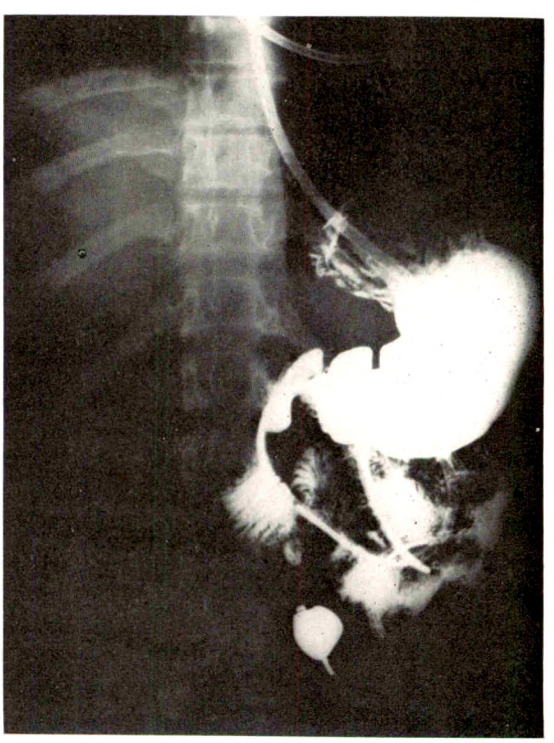


FIG. 4. Case IV. A 26 year old black male with right upper quadrant pain, tenderness and fever; 1,000 cc. pus was drained.

organ or *vice versa*. Downward displacement of the kidney by an enlarged spleen[4–6] is an example. Displacement of the ureters by an ovarian cyst and displacement of the small bowel or colon by a renal mass are others.

The caudate lobe of the liver is located immediately to the right of the decending portion of the duodenum. Enlargement of this portion of the liver or generalized hepatic enlargement will displace the duodenum, and the displacement is primarily to the left. No anterior or posterior displacement could be anticipated.

Other causes of the left side displacement of the duodenum include: enlarged gallbladder; enlargement or mass of the right kidney; mass of the right adrenal gland; and retroperitoneal tumor such as sarcoma.

All 7 cases we have encountered (Fig. 1 through 7) showed varying degrees of left side displacement of the second portion of the duodenum by an enlarged liver. There

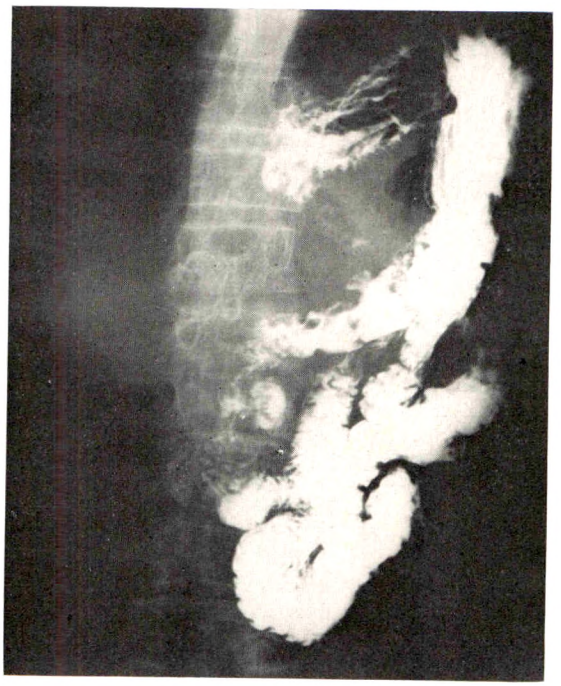


Fig. 6. Case vi. A 65 year old male with a long history of alcoholism and a palpable liver.

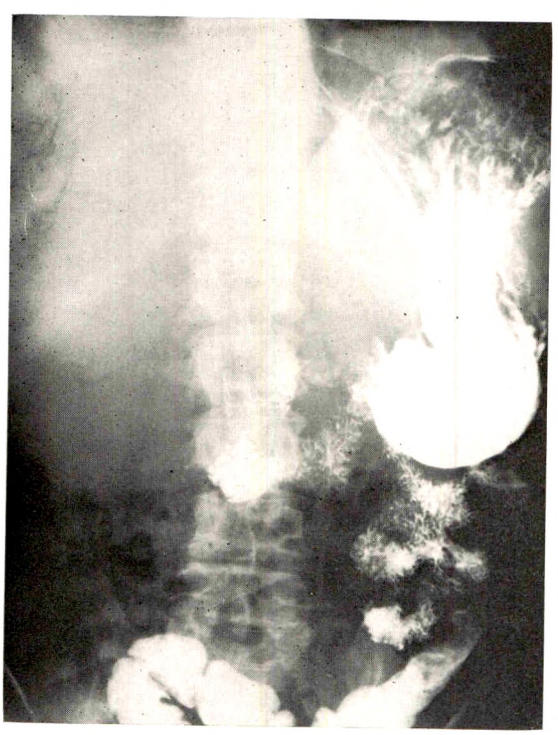


Fig. 5. Case v. Metastatic carcinoma of the breast with a liver palpable 10 cm. below the right costal margin.

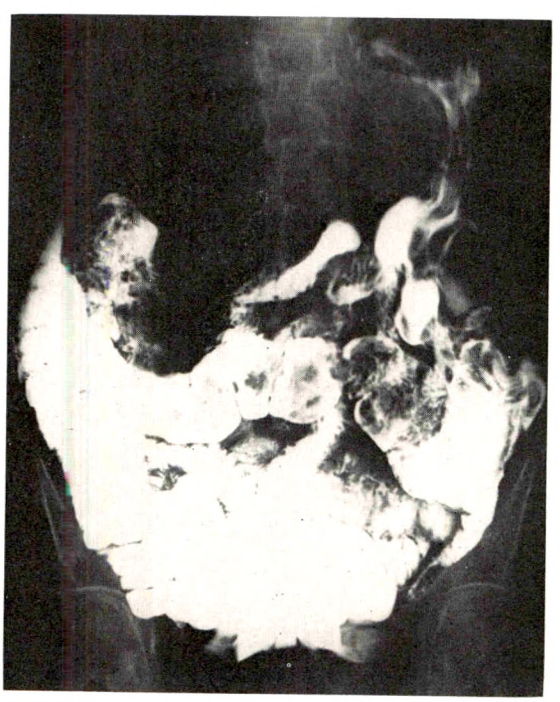


Fig. 7. Case vii. A 58 year old white male with metastatic adenocarcinoma of the rectum and a palpable liver.

was no displacement either posteriorly or anteriorly. This finding is not as infrequent as has been thought in the past.

SUMMARY

Displacement of the retroperitoneal organ by intraperitoneal mass is not an infrequent finding.

Seven cases of displacement of the descending portion of the duodenum by an enlarged liver are reported and discussed.

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UNILOBAR VENO-OCCLUSIVE DISEASE OF THE LIVER*

ANGIOGRAPHIC DEMONSTRATION OF INTRAHEPATIC COMPETITION SIMULATING HEPATOMA

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ANTEMORTEM roentgenologic documentation of hepatic veno-occlusive disease, *i.e.*, Budd-Chiari syndrome, can be difficult. The hepatic veins are also inaccessible for exploration at surgery. Until 1968, there were approximately 500 cases[13] reported in the literature, the majority of which were diagnosed at autopsy. Alterations in hepatic hemodynamics do occur and can present abnormal patterns on arteriography,[2,12] vena caval and hepatic phlebography,[2,13] splenoportography,[2,13] radionuclide scanning,[2] and parenchymal hepatography.[2,10,13] The combination of these modalities, and particularly intraparenchymal hepatography, can be used to arrive at the diagnosis in most cases.[13]

The diagnosis becomes increasingly difficult when all the hepatic veins are not involved. Asymmetric or incomplete occlusion allows intrahepatic portal vein to portal vein shunts as well as hepatic vein to portal vein shunts to occur.[11] When asymmetric venous occlusion occurs, a state of intrahepatic competition can arise. This produces areas of liver atrophy and hypertrophy which are distinctly different from the homogeneous congestive hepatomegaly that is usually described in cases of Budd-Chiari syndrome.[14,15,17] This competitive phenomenon is well known to those engaged in liver transplantation,[16,19,20] but has only been observed twice in human autopsy cases.[6,9]

The following case illustrates the roentgenographic changes that occur as a result of this physiologic situation.

REPORT OF A CASE

A 55 year old white female was admitted on July 12, 1972 for evaluation of polycythemia and ascites. She had been in good health until her admission in March, 1971 for removal of an adenoma of the thyroid gland. In September, 1971 she complained of headaches and dizziness and her family doctor told her she had polycythemia. She was treated with anticoagulants but later seemed to improve and the anticoagulants were discontinued. In June, 1972 she again noted fatigue and dizziness and was referred to a hematologist who performed a phlebotomy. Shortly afterwards she noticed an increase in abdominal girth and swelling of her legs. Thiazide diuretics were given without success. She gave no previous history of alcoholism, hepatitis, blood transfusions, exposure to toxic chemicals or drugs.

Physical examination revealed a blood pressure of 120/80 mm. Hg, pulse of 110/min., and regular respiration of 24/min. The patient appeared flushed. The peripheral lymph nodes were not palpable. The heart was not enlarged, and there were no murmurs. The neck veins were not distended. There were bibasilar rales of the chest. The abdomen was distended with ascites but without definite masses or tenderness. There was 3+ pretibial edema. A gynecologic examination was normal.

Laboratory data revealed a hemoglobin of 16.4 gm. per cent, hematocrit of 52 per cent, 370,000 platelets and white blood cell count of 9,200 per cu. mm. An SMA 12 revealed a total bilirubin of 1.8 mg. per cent, alkaline phosphatase of 250 units, LDH of 307 and SGOT of 222. Protein electrophoresis was within normal limits except for a low albumin. Urinalysis was normal except for a 1+ albumin. Australia antigen was negative. Alpha-1 fetoproteins were not detected. Leukocyte alkaline phosphatase was 106 units (normal 10–70). Paracentesis yielded 1,400 cc. of ascitic fluid which was clear and yellow. The protein was 2,160 mg. per cent with 97 white blood cells and 106 red blood cells. Papanicolaou smear and cell block were

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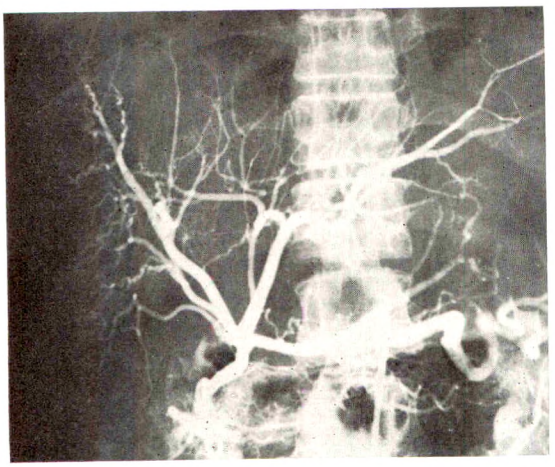


FIG. 1. An early arterial phase of celiac arteriography shows marked tortuosity of the right hepatic artery branches in a shrunken right lobe of liver. The left hepatic artery is large with stretched branches throughout an enlarged left lobe.

negative for malignant cells. Bone marrow examination revealed an active marrow. Following roentgenologic work-up, the patient was surgically explored.

Radiologic Findings. Roentgenologic studies included a normal upper gastrointestinal series and barium enema examination.

Visceral angiography demonstrated a hypervascular, shrunken right lobe of the liver with markedly tortuous arteries and a suggestion of neovascularity (Fig. 1; and 2). The left lobe was enlarged with stretching and draping of the arteries. The hepatographic phase showed a diffuse mottled appearance. The augmented venous phase, after 50 mg. of tolazoline was injected into the superior mesenteric artery, showed hepatofugal flow and filling of esophageal varices (Fig. 3). The right portal vein had a discrete cut-off, typical of an anatomic obstruction (Fig. 4). The spleen was enlarged with areas of lucency suggestive of infarcts.

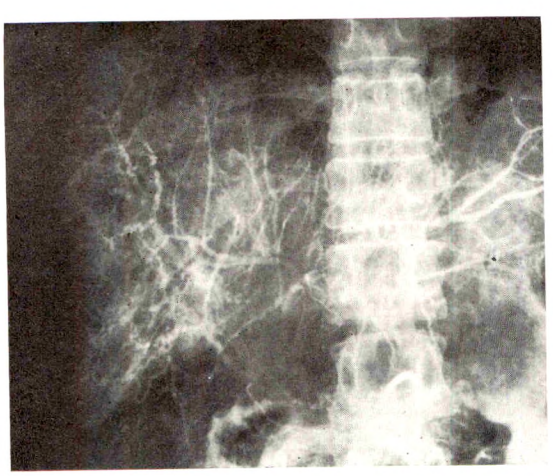


FIG. 2. A later study shows marked hypervascularity in the right lobe and a large amount of ascites. This hypervascularity is caused by superimposition of corkscrew-shaped hepatic arteries and sinusoidal puddling which give the appearance of neovascularity.

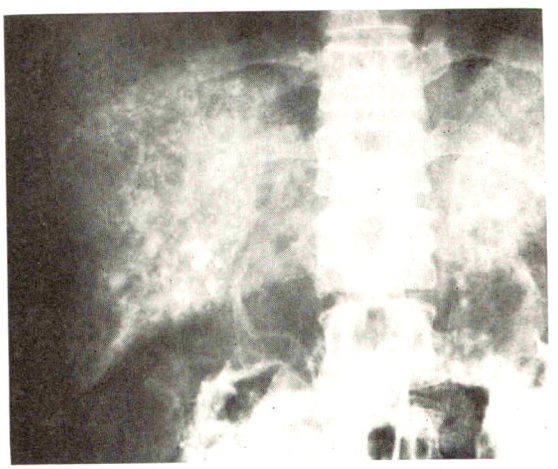


FIG. 3. A later venous phase demonstrates sinusoidal puddling in the right lobe only. This is indicative of hepatic venous outflow obstruction.

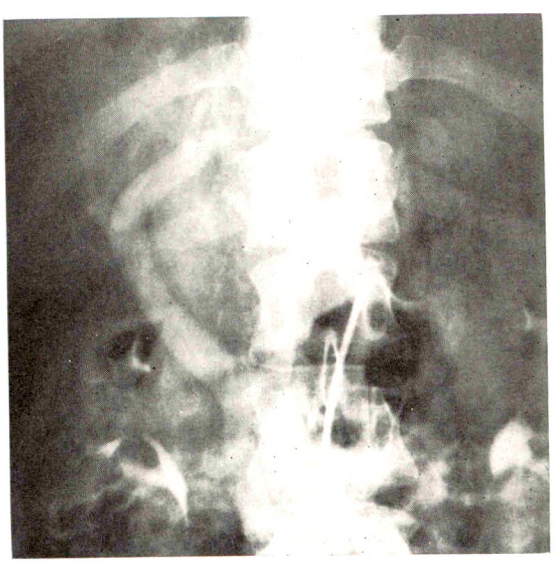


FIG. 4. The venous phase of a superior mesenteric artery injection shows a normal portal vein and an apparent discrete occlusion of the right portal branch.

Venacavagraphy demonstrated a normal inferior vena cava. Reflux into the hepatic veins did not occur, and the hepatic veins could not be entered selectively from the vena cava.

A liver scan using technetium 99m sulfur colloid was interpreted as showing a decreased uptake in the entire right lobe compatible with a mass lesion. The spleen was enlarged with multiple filling defects.

The combination of these findings suggested occlusion of the hepatic and right portal veins and possibly a hepatoma of the right lobe. An intraparenchymal hepatogram and a liver biopsy were suggested. Since the patient was to have a portacaval shunt as treatment for the ascites, these studies were to be done at the time of the exploration.

Surgical Findings. At exploration there was yellow, clear ascitic fluid. The right lobe of the liver was contracted to about one-quarter normal size and was firm and smooth. The left lobe felt spongy and was slightly enlarged.

A superior mesenteric vein catheter measured an elevated portal pressure of 470 mm. of saline. Injection of contrast material into this catheter showed hepatofugal flow and gastric varices (Fig. 5). The same discrete cut-off of the right portal vein was seen.

Following this, a needle was placed into the parenchyma of the right lobe of the liver. Twenty cubic centimeters of conray 60 were injected, which collected in the sinusoids and then flowed retrograde to fill the right portal branches, the main right portal vein and finally the left portal system (Fig. 6). This documented intrahepatic portal shunting from right-to-left and demonstrated a functional rather than an anatomic occlusion of the right portal vein. A similar injection into the left lobe showed no intrahepatic shunting.

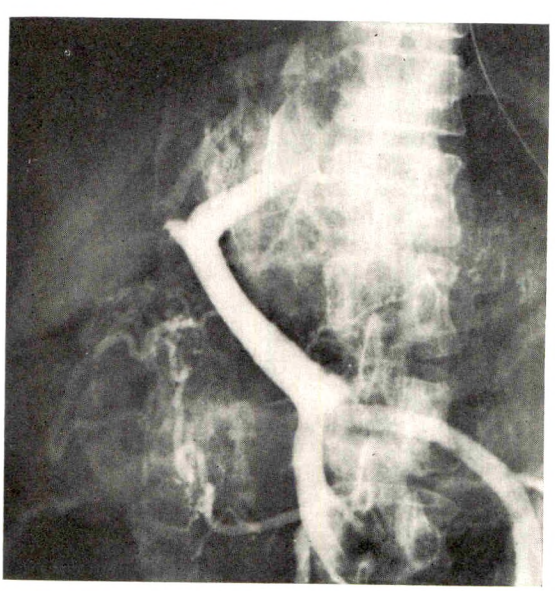


Fig. 5. An operative direct superior mesenteric venogram confirms the impression of right portal vein occlusion and antegrade flow in the left portal vein. Gastric varices are also demonstrated.

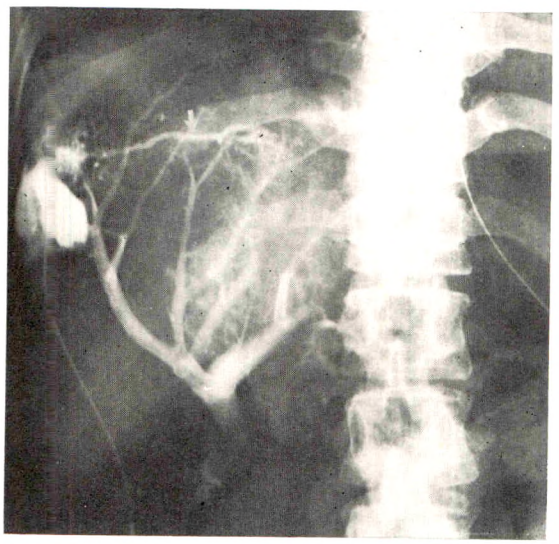


Fig. 6. An hepatic parenchymagram was obtained by the direct injection of contrast material into the liver. Preferential retrograde flow from the sinusoids to a clearly patent right portal vein branch is demonstrated. Blood shunts from the right portal vein to the left portal vein and thence to the systemic circulation are seen. Antegrade filling of a single hepatic vein (arrow) which then drains back into the right portal system is also present.

A biopsy from the right lobe was interpreted as showing infracapsular fibrosis. The centrilobular veins were congested with mild liver cell necrosis. The left lobe biopsy showed more central congestion but less fibrosis. These findings, particularly of the left lobe, were thought to be typical for hepatic vein occlusion. There was no histologic evidence of a tumor.

A portacaval shunt was constructed between the side of the portal vein and the side of the inferior vena cava with correction of the portal hypertension.

Postoperatively, she has done well with only mild derangement of liver function. The ascites

has completely disappeared. Presently she is on no therapy for polycythemia.

DISCUSSION

Hepatic venous outflow obstruction can be the result of right heart failure, constrictive pericarditis, obstruction of the inferior vena cava or occlusion of the hepatic veins.[2,10] The venous occlusion may be due to clot, tumor,[2,11] or a congenital membrane[7] found in the intrahepatic portion of the vena cava. Recently, outflow occlusion has been reported in patients receiving oral contraceptives.[3,5] Primary or secondary polycythemia has been found in about one-third of the cases.[2] The majority of cases, however, are thought to be due to primary thrombosis or endophlebitis.[11] When the syndrome is suspected clinically, an attempt should be made to document the abnormality in order to plan the therapy. Local tumor invasion or distant tumors producing erythropoietin[1,11] should obviously be excluded before specific therapy is undertaken. Portacaval shunting has been shown to control the ascites in some patients.[4,21]

The syndrome may develop acutely or subacutely, depending upon the degree and rapidity of the hepatic venous occlusion.[11] Sudden, complete occlusion is usually manifested by shock and liver failure.[6] Retrograde thrombosis may extend to the portal and mesenteric veins, producing portal obstruction and bowel infarction. Less acute or slowly progressive occlusion produces intractable ascites.[6,11] One should suspect this syndrome in any patient who presents with refractory ascites and near normal liver function.[11,13] In a series of cases reported in 1945, the chronic cases lived from a few weeks to 6 months.[6]

Although most reported cases refer to congestive hepatomegaly as a result of the outflow obstruction, Kelsey and Comfort[6] suggested that liver atrophy could occur in long term cases. One of the autopsy cases they reported demonstrated atrophy of the left lobe and a "compensatory hypertrophy" of an unaffected caudate lobe. This was similar to a pathologic description of a case given by Murray and Kimmel.[9] The explanation for the atrophy was thought to be due to cirrhosis, and the hypertrophy was referred to as compensatory.

Rous and Larimore[14,15] in 1920 showed that when the hepatic duct or portal vein to one lobe of the liver was ligated, the lobe would atrophy. It was Schalm *et al.*[16,17] who popularized and extended these observations. They represented the liver as being in a homeostatic balance between bile flow, portal inflow and venous outflow. They postulated that the lobe with more of these functional possibilities would hypertrophy, while the part with a functional disadvantage would atrophy. This balanced atrophy and hypertrophy would result in maintenance of a constant liver mass. The same phenomenon has been shown to occur in heterotopic liver transplants if the host liver is left *in situ*.

This concept of "functional" competition has been challenged by Starzl and his coworkers.[8,18] They noted that there was a qualitative as well as a quantitative effect of the splanchnic venous blood on the liver. Using the portacaval transposition described by Child, they delivered the same quantity of venous blood to the liver after transposing these vessels. Although the portal venous flow eventually reached the liver through re-circulation, deglycogenation of the liver did occur, suggesting that there were subtle metabolic changes even though hepatic integrity was preserved. To determine the qualitative effects of venous inflow they devised a "split transposition" experiment.[8] The right or left portal vein was attached to the suprarenal inferior vena cava and the other portal branch was left attached to the portal vein. Therefore, one lobe received blood from the inferior vena cava and the other lobe received portal blood. The total venous inflow was the same in both and the oxygen content was similar. The lobe receiving portal venous blood thrived, while the lobe receiving vena cava blood atrophied. They concluded that survival favors the

lobe receiving a "hepatotrophic substance," found only in portal vein blood. Van DerHeyde[19,20] was able to show, however, that portal blood deprivation alone was not responsible for the atrophy, and proved experimentally the presence of a true competitive phenomenon. He took 2 groups of dogs and grafted a liver heterotopically, leaving the host liver *in situ*. When the host liver was left with adequate bile drainage and good portal flow, the auxiliary liver became shrunken. If, on the other hand, the host liver was deprived of bile flow and portal blood, the auxiliary liver would be normal or hypertrophied. The deduction then is that there is a true competition between host and auxiliary livers and between lobes of the same liver.

When incomplete hepatic venous occlusion is present, the hypertrophic and atrophic areas can be histologically similar.[6,9] There is centrilobular congestion and necrosis in both hypertrophic and atrophic areas and a sharp border between the congested area and the surviving cells. Replacement fibrosis is seen in the atrophic areas. In the regions of hypertrophy, the cells are increased in number and size.

We believe that this case demonstrates the phenomenon of intrahepatic competition between the compromised right lobe with its occluded hepatic veins and functionally occluded portal veins and the left lobe of the liver with its more normal portal blood supply and hepatic venous drainage. The resulting roentgenographic appearance superficially mimics a hepatoma with occlusion of the right portal vein, ascites, portal hypertension, increased and distorted vasculature of the right lobe of liver and a large cold area in the right lobe on technetium scans. The shrunken right lobe also closely simulates the angiographic features of cirrhosis with tortuosity of hepatic arteries due to loss of normal parenchyma.

The only truly diagnostic study is the hepatic parenchymatogram. This procedure confirmed the functional nature of the apparent right portal vein occlusion and established the hepatic hemodynamics that are diagnostic of hepatic vein thrombosis.

SUMMARY

Unilobar hepatic vein thrombosis can result in interlobar competition with ultimate atrophy of the occluded lobe and hypertrophy of the less affected lobe.

The angiographic appearance of this condition mimics hepatoma and cirrhosis in the atrophic lobe and could lead to diagnostic inaccuracy.

Injection of contrast material directly into the hepatic parenchyma is the technique of choice to establish the true state of the intrahepatic circulation.

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ANGIOGRAPHY IN BLUNT HEPATIC TRAUMA*

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BLUNT hepatic trauma resulting from an external blow or sudden compression is one of the most serious forms of abdominal injury. The mortality rates for these patients have varied in reported series from approximately 30 per cent[2,11,16] to over 70 per cent.[4] The principal cause of death is hemorrhage. Symptoms generally commence within hours or days following the incident, but a latent period of months or even years is not uncommon.[11]

Although the surgical literature abounds with discussions of the diagnosis and treatment of blunt hepatic trauma, one finds relatively little pertaining to the use of angiography. Several authors have reported the angiographic findings in small series,[1,3,4,13] while others have reported upon individual cases.[5,7–10] These papers and our own experience indicate that angiography often can play a crucial role in proper surgical management. Nevertheless, this procedure has been almost totally ignored in several extensive, recently published discussions of liver trauma.[2,11,16]

The purpose of this paper is to summarize the angiographic findings in various types of liver injury and to emphasize the great importance of this modality in the evaluation of these patients.

TYPES OF LIVER INJURIES AND THEIR ANGIOGRAPHIC MANIFESTATIONS

Liver injury resulting from blunt trauma can take a variety of forms. Some of the most important are: (1) subcapsular hematoma; (2) internal parenchymal rupture with hematoma formation; (3) parenchymal contusion; (4) laceration or avulsion of vessels; (5) arteriovenous fistula; (6) pseudoaneurysm or cavity formation with resultant hemobilia; (7) formation of a traumatic "bile cyst;" and (8) inferior vena caval obstruction.

1. *Subcapsular hematoma.* This is generally the least serious of the major liver injuries and sometimes can be treated conservatively. It is due to a superficial rent on the surface of the liver and may develop substantial expansile pressure if it remains confined beneath the capsule. The hematoma is an avascular mass which displaces the liver away from the borders of the abdominal cavity and produces an extrinsic compression deformity or concave outer border during the hepatogram phase of the angiogram. This lesion is demonstrated in Figure 1, *A* and *B*.

2. *Parenchymal rupture with hematoma formation.* Internal rupture of the liver is usually also accompanied by hematoma formation, and it is the latter which is chiefly responsible for the angiographic abnormalities. The hematoma appears as an avascular mass within the parenchyma. Hepatic arteries and portal veins are displaced, elongated and stretched about the periphery of the mass. During the hepatogram phase of the study, an irregular radiolucent defect is present. Collections of extravasated bile or areas of septic necrosis are somewhat less common complications of liver trauma which may have a similar angiographic appearance.

3. *Parenchymal contusion.* Parenchymal contusion may occur without rupture or may be present at rupture margins. In either event, angiography reveals patchy hypervascularity and irregular accumulation of contrast medium within the hepatic

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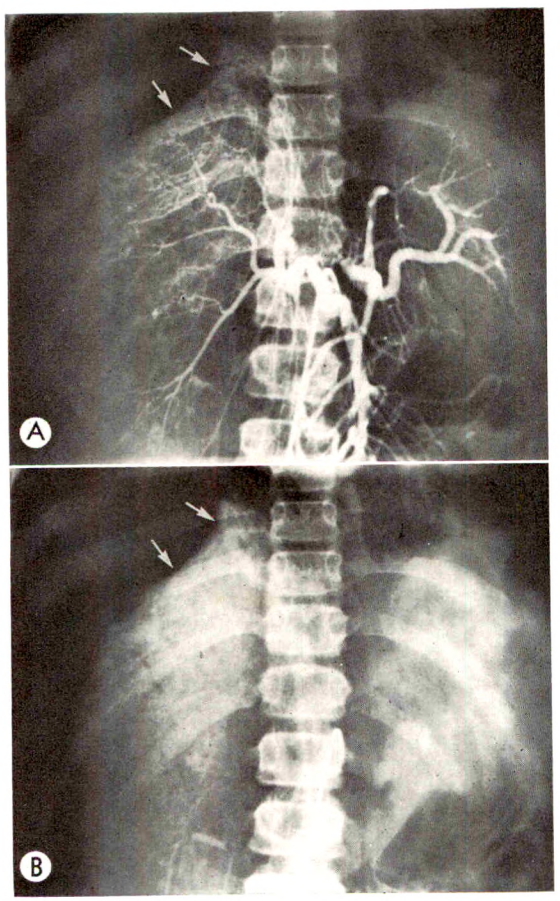


FIG. 1. (*A*) Arterial and (*B*) hepatogram phases of a superior mesenteric arteriography in which the hepatic artery arises from the superior mesenteric artery. A large subcapsular hematoma of the liver is present following blunt abdominal trauma. The pressure of the blood confined beneath the liver capsule has displaced the entire liver medially and produced parenchymal compression and a concave border along the superior aspect (white arrows).

tissue.[3] Figure 2, *A–C*, is an example of both central hepatic rupture with hematoma formation, and surrounding parenchymal contusion. Angiographic demonstration of parenchymal contusion necessitates careful surgical inspection of the area to assess its viability. If circulation is compromised, the liver parenchyma will appear cyanotic. If total devascularization has occurred, the affected area will be yellowish-gray in color.[12] A cardinal rule of surgical treatment of liver injury is complete removal of this devitalized tissue.

4. *Laceration or avulsion of vessels.* Lacerations of hepatic artery branches will result in extravasation of contrast material during angiography,[3,4] just as occurs in cases of gastrointestinal tract bleeding. During a somewhat later phase, a pseudoaneurysm may form (Fig. 2, *A–C*; 4, *A* and *B*; and 5, *A–C*) and serve as a source of subsequent hemorrhage into the hepatic parenchyma or the biliary tree. Occlusion of arteries can occasionally be seen;[1,4] this is probably the result of spontaneous thrombosis of a lacerated vessel. One of the most serious of all hepatic injuries is avulsion of the hepatic veins from the inferior vena cava.[15] These patients are generally bleeding so rapidly and massively when they are admitted to the hospital that there is no time for angiography to be performed. We are not aware of any cases in which venous bleeding has been demonstrated by inferior venacavagraphy or hepatic venography.

5. *Arteriovenous fistula.* Hepatic artery-portal vein shunts develop quite rapidly, having been demonstrated only a few hours following blunt liver trauma.[1,4] They can result from either laceration of relatively large vessels or parenchymal contusion. Spontaneous closure of such lesions has been reported.[1] Figure 3 is an example of post-traumatic hepatic artery-portal vein fistula. Hepatic artery-hepatic vein fistulae are seen with much less frequency. This is undoubtedly due to the fact that small hepatic artery and portal vein branches are very closely approximated in the periportal connective tissue, but are separated from the hepatic veins by the radius of the liver lobule.

6. *Pseudoaneurysm or cavity formation with hemobilia.* This complication may develop within days or months following an episode of liver trauma. Hemobilia is sometimes accompanied by colicky right upper quadrant pain and transient jaundice. Hepatic angiography is the only method of preoperatively localizing the source of bleeding. In many instances a sac-like collection of contrast material is

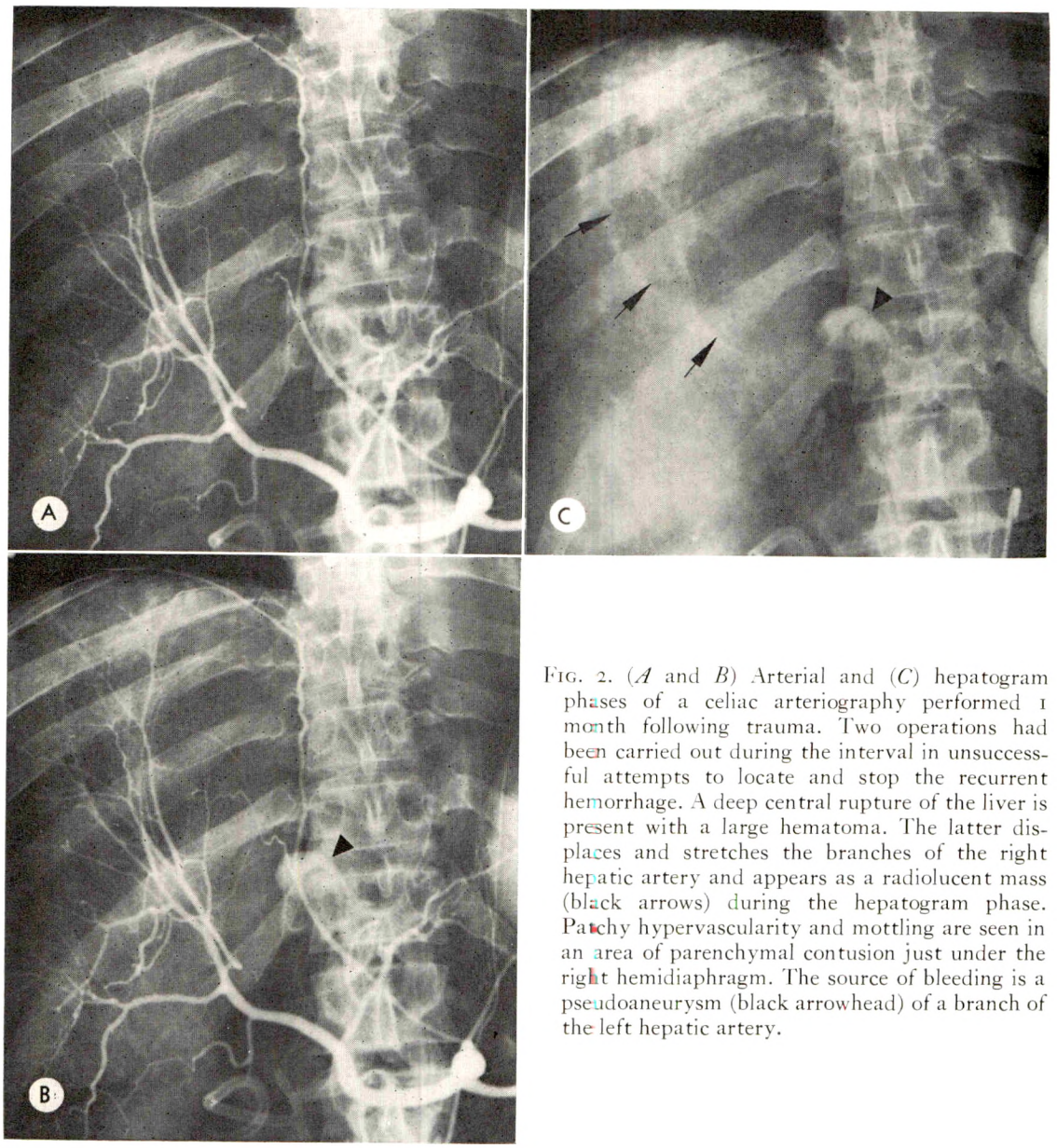


Fig. 2. (*A* and *B*) Arterial and (*C*) hepatogram phases of a celiac arteriography performed 1 month following trauma. Two operations had been carried out during the interval in unsuccessful attempts to locate and stop the recurrent hemorrhage. A deep central rupture of the liver is present with a large hematoma. The latter displaces and stretches the branches of the right hepatic artery and appears as a radiolucent mass (black arrows) during the hepatogram phase. Patchy hypervascularity and mottling are seen in an area of parenchymal contusion just under the right hemidiaphragm. The source of bleeding is a pseudoaneurysm (black arrowhead) of a branch of the left hepatic artery.

seen filling rapidly from one of the hepatic artery branches (Fig. 4, *A* and *B*). This represents either a pseudoaneurysm[1,10] or a cavity which results from necrosis or infection of devitalized tissue and erodes into an adjacent artery.[7,8,9] Ultimately these lesions, which cannot be differentiated angiographically, decompress themselves into a nearby bile duct and gastrointestinal bleeding results. Less frequently, the angiogram of hemobilia patients reveals hepatic artery-portal vein fistulae.[4] In such cases, the exact pathogenesis of the gastrointestinal bleeding is unclear. It has also been pointed out[16] that stress peptic ulceration is a relatively common complication of hepatic injury. Therefore, if gastrointestinal bleeding occurs and no intrahepatic source is found at angiography, the arteries supplying the stomach and duodenum must also be adequately studied.

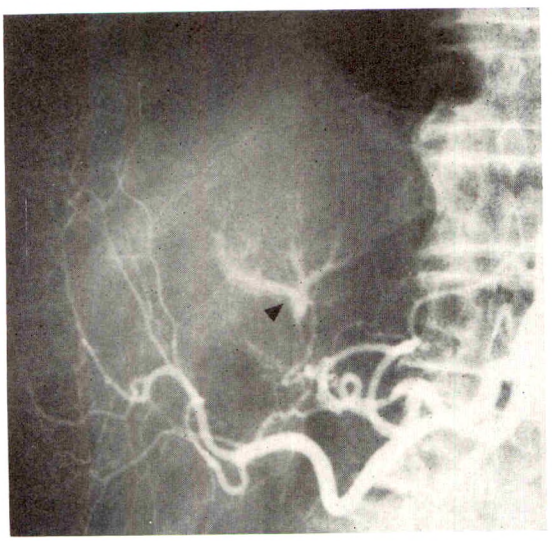


FIG. 3. Post-traumatic fistula between branches of the left hepatic artery and intrahepatic branches of the portal vein (black arrowhead). The surrounding area is avascular, probably indicating the presence of a hematoma. The patient was treated conservatively.

7. *Formation of a traumatic "bile cyst."* Traumatic interruption of a bile duct is generally demonstrated by operative cholangiography.[12] Although it produces no immediate specific arteriographic changes, it may lead to the formation of a so-called traumatic bile cyst—the accumulation of a large confined collection of bile within the substance of the liver. This lesion (Fig. 5, *A–C*) resembles a hematoma angiographically, although the latter is more common. The cystic, bile-containing mass displaces vessels and appears as an avascular defect during the hepatogram phase.

8. *Inferior vena caval obstruction.* This is a potentially serious late complication which resulted in the sudden death of the patient shown in Figure 2, *A–C*. His original liver injury had occurred 1 month earlier and, during the interval, 2 operations had been performed at another hospital in an unsuccessful attempt to locate and stop recurrent intra-abdominal bleeding. On the day of admission to the New York Hospital, emergency arteriography was performed and the patient was taken to the operating room immediately thereafter. Inferior venacavagraphy was not performed. At surgery, an apparently successful repair of a large rupture and pseudoaneurysm was carried out, but the patient abruptly expired 10 hours later. Postmortem examination revealed extensive thrombosis of the hepatic veins and suprahepatic inferior vena cava with multiple pulmonary emboli as the cause of death. The vena caval involvement could probably have been diagnosed preoperatively if inferior

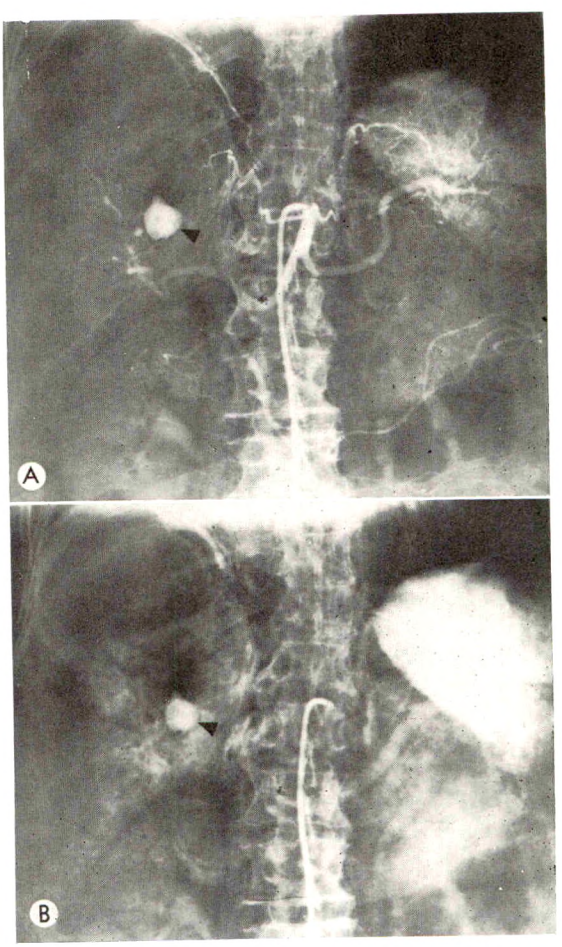


FIG. 4. (*A* and *B*) Early and late phase films from a celiac arteriography. The patient had sustained blunt trauma with fractured right posterior ribs 10 days earlier. On admission, she had a low hematocrit and tarry stools. A large pseudoaneurysm (black arrowhead) of one of the hepatic artery branches is present and is the most likely source of gastrointestinal bleeding. The large irregular avascular area above it was found to be an infected hematoma.

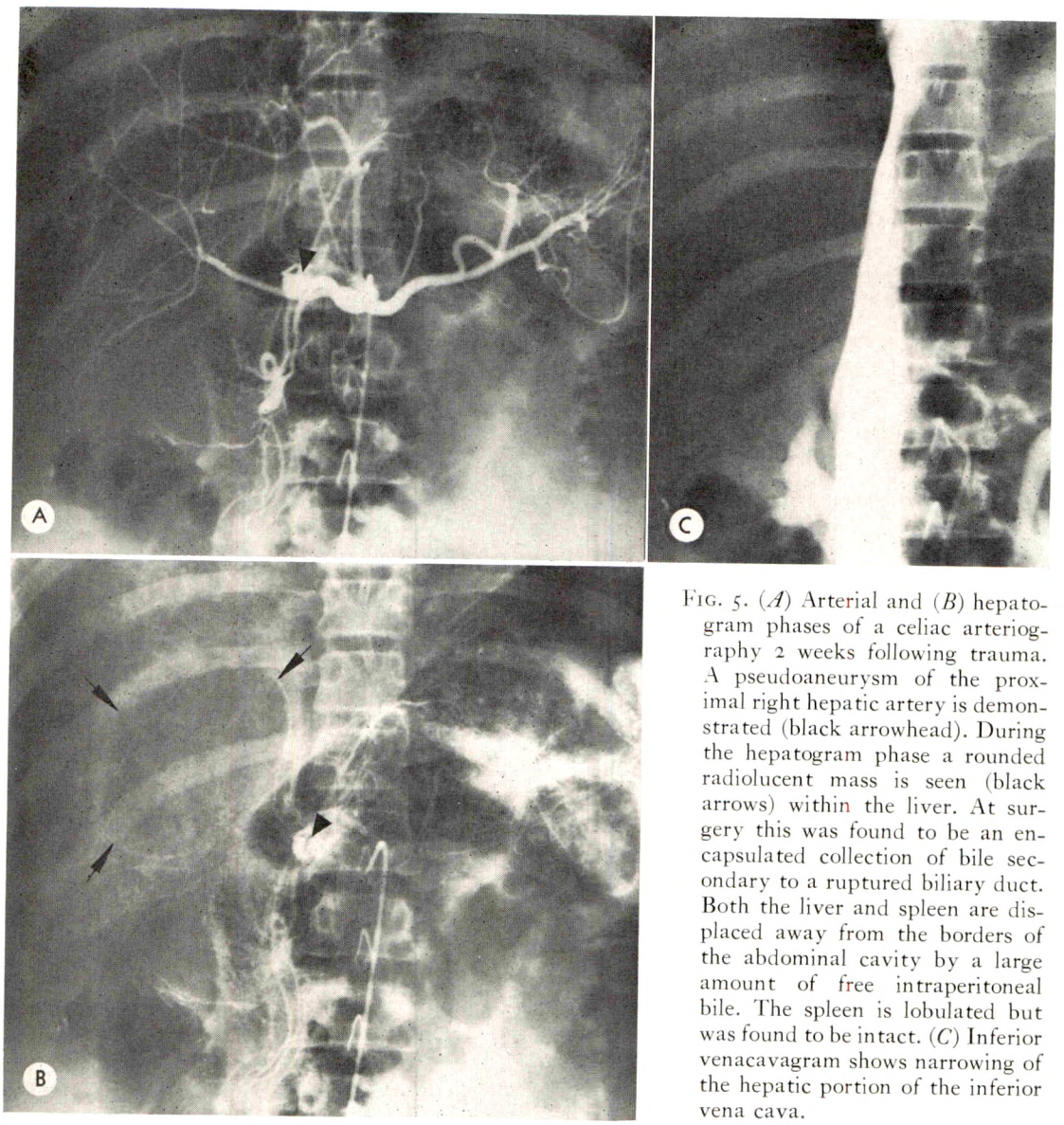


FIG. 5. (*A*) Arterial and (*B*) hepatogram phases of a celiac arteriography 2 weeks following trauma. A pseudoaneurysm of the proximal right hepatic artery is demonstrated (black arrowhead). During the hepatogram phase a rounded radiolucent mass is seen (black arrows) within the liver. At surgery this was found to be an encapsulated collection of bile secondary to a ruptured biliary duct. Both the liver and spleen are displaced away from the borders of the abdominal cavity by a large amount of free intraperitoneal bile. The spleen is lobulated but was found to be intact. (*C*) Inferior venacavagram shows narrowing of the hepatic portion of the inferior vena cava.

venacavagraphy had been performed. Nonthrombotic obstruction of the inferior vena cava has also been reported,[11] caused by a massive intrahepatic hematoma and leading to acute renal failure.

As a result of these cases, we now perform an inferior venacavagraphy on every patient who is referred for hepatic angiography following blunt hepatic trauma.

TREATMENT

Operative repair of the traumatized liver is a complex subject which has been extensively discussed in the surgical literature.[2,6,11,16] We will only briefly review the salient aspects here.

The surgeon's first task is location and control of the source of bleeding. For superficial injuries, hemostasis and external drainage may be all that is necessary. Deeper ruptures must be carefully approximated, with sutures to obliterate potential dead space. Devitalized tissue must be debrided. If damage is diffuse and extensive, major hepatic resection may be required. Human patients have survived re-

moval of up to 80 per cent of the liver. Once repair of the actual hepatic injury has been carried out, proper drainage of the wound and the perihepatic area is mandatory. T-tube drainage of the biliary tree has been advocated by some surgeons for all central or hilar injuries, but this point is still controversial.[16]

The indications for conservative, nonoperative treatment are not clearly defined, but it would seem that a significant number of patients with subcapsular hematomas, localized intraparenchymal hematomas or uncomplicated arteriovenous fistulae can be managed in this manner.[1,4]

DISCUSSION

It is apparent that blunt abdominal trauma can produce a variety of complex hepatic injuries. Surgical treatment is often urgent and difficult, even under the best of circumstances. Reoperation, complication and mortality rates remain unacceptably high. Angiography has proven to be a highly reliable method of defining the type, location and extent of most liver injuries, except in cases of venous avulsion or acute interruption of biliary ducts. The procedure can be carried out in a relatively short period of time while the operating room is being prepared and blood typed and crossmatched. We feel that hepatic arteriography and inferior venacavagraphy should be carried out in almost every patient suspected of hepatic injury or who requires reoperation following earlier inadequate surgery. The only contraindication is the existence of such rapid hemorrhage as to make further delay of surgery life-threatening.

SUMMARY

Abdominal blows or sudden external compression may result in a variety of complex and dangerous liver injuries.

The nature of these injuries is discussed, their angiographic appearance and the principles of surgical treatment are described.

Angiography is an extremely valuable adjunct in the management of such patients, in that it defines the type, location and extent of most of the lesions prior to operation.

It is to be hoped that wider application of this procedure, which is apparently not being utilized to its fullest potential in some centers, will result in significant improvement in the high complication and mortality rates associated with blunt hepatic trauma.

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HEPATIC ARTERIAL ESCAPE FROM VASOPRESSIN-INDUCED VASOCONSTRICTION: AN ANGIOGRAPHIC INVESTIGATION*

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DURING the intraarterial administration of vasopressin in the treatment of upper gastrointestinal hemorrhage,[4] we have noted that the hepatic arterial system appears to escape the vasoconstriction induced in the other branches of the celiac axis. In the first 2 instances in which we made this observation, vasopressin was being administered into the common hepatic arteries of patients who were proved angiographically to be bleeding from branches of the gastroduodenal artery. Despite vasoconstriction of the gastroduodenal artery and its branches and cessation of hemorrhage, the hepatic arterial system was not constricted. We assumed in both instances that the catheter was fortuitously positioned so that the vasopressin was preferentially streaming into the gastroduodenal artery, thus avoiding the hepatic circulation. Later we observed that no hepatic arterial constriction occurred in a patient in whom a catheter had been displaced from its original position in the gastroduodenal artery into the right hepatic artery, and realized that the hepatic arterial system may respond differently from the other splanchnic arteries. Furthermore, we have noted no functional or histologic hepatic abnormalities that could be attributed to vasopressin-induced constriction of the hepatic arteries, even after prolonged perfusions of the celiac axis in cirrhotic patients.

Fear of the consequences of hepatic arterial vasoconstriction has led some investigators to avoid this form of therapy or to use other vasoconstrictive agents.[8] The physiologic and therapeutic implications of these observations stimulated us to review objectively the effects of vasopressin on the size of splanchnic arteries infused during the treatment of upper gastrointestinal bleeding.

The present study reports measurements of angiographic arterial diameters before and after the administration of therapeutic doses of vasopressin.

MATERIAL AND METHOD

The angiographic studies of all patients who had received intraarterial vasopressin in the management of upper gastrointestinal bleeding, at this hospital, were analyzed. All examinations of patients in whom angiographic examinations were performed before and immediately after vasopressin infusions, and in whom both examinations were performed under identical conditions, were included. Fifteen patients who had received celiac axis angiography and 8 with superior mesenteric arteriography were selected for study. Angiography was performed using percutaneous green Kifa catheters through which 70 per cent hypaque was injected at 15 ml. per sec. The volume of infusion, pressure of injection and timing of filming were constant for each patient before and after vasopressin.

All markings on films were obliterated and they were identified only by code number. The sites for measurement were marked on paired pre- and post-vasopressin angiograms. Arterial diameters were measured using vernier calipers and read to the nearest 0.1 mm. All arteries that were satisfactory for measurement on both pre-

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TABLE I

EFFECT OF VASOPRESSIN ON ARTERIAL DIAMETER

Artery	No. of Patients	No. of Measurements	Change in Arterial Diameter
			Mean ± S.E.M. (per cent of prevasopressin diameter)
Common hepatic	12	15	− 3.1 ± 3.4*
Hepatic	14	85	+ 7.7 ± 2.2
Gastroduodenal	12	45	−29.2 ± 3.7‡
Left gastric	7	10	− 9.0 ± 4.1†
Splenic	9	37	−14.0 ± 2.9‡
Superior mesenteric	8	100	−11.5 ± 2.0‡

* Not significant statistically.

† $p<0.025$, ‡ $p<0.001$ } statistical difference (unpaired t test) from hepatic artery.

and post-vasopressin films were marked for measurement. Arterial diameters were measured before and after each branching. No branches less than 1.0 mm. in diameter were considered measurable. All films were measured twice by the same observer. First, paired sets of films, unidentified as "pre"- or "post-vasopressin" films, were measured. Subsequently, the films were re-randomized and re-numbered and re-measured individually. The 2 measurements for each artery on each film were then averaged. If the 2 measurements differed by more than 5 per cent from the mean, a third measurement was made and the aberrant measurement discarded. The mean post-vasopressin measurement for each artery was expressed as percentage of the pre-vasopressin diameter.

The effect of vasopressin on each arterial system was assessed by determining the mean change in diameter for all arteries (Table I). In addition, the response of each arterial system to vasopressin for each patient was determined by assessing the predominant effects on arterial diameter, *i.e.*, increased, unchanged or decreased (Table II). Finally, the effect of vasopressin in relation to arterial size was evaluated by determining for each arterial system the mean change in diameter for arteries of all sizes. Changes in arterial blood flow were calculated from measured changes in arterial diameters based on Poiseuille's law (Table III).

RESULTS

RELIABILITY OF MEASUREMENTS

In 305 pairs of observations the mean difference between readings was 0.24 mm.

TABLE II

EFFECT OF VASOPRESSIN ON ARTERIAL DIAMETER

Artery	Total No. of Measurements	Effect on Arterial Diameter: No. of Measurements			Total No. of Patients	Effect on Arterial Diameter: No. of Patients		
		Increased	Unchanged	Decreased		Increased	Unchanged	Decreased
Common hepatic	15	3	7	5	12	2	6	4
Hepatic	85	36	31	18	14	9	1	4
Gastroduodenal	45	4	4	37	12	0	1	11
Left gastric	10	1	3	6	7	1	2	4
Splenic	37	1	11	25	9	0	1	8
Superior mesenteric	100	15	19	66	8	1	0	7

TABLE III

EFFECT OF CHANGES IN ARTERIAL DIAMETER ON CALCULATED ARTERIAL BLOOD FLOW

Artery	Pre-Vasopressin Diameter in Per Cent	Post-Vasopressin Diameter in Per Cent	Post-Vasopressin Blood Flow in Per Cent
Hepatic	100	107.6	134
Gastroduodenal	100	70.8	25
Left gastric	100	91.0	69
Splenic	100	86.0	55
Superior mesenteric	100	88.5	61

and ranged from 0.0 to 1.3 mm. The distribution of these differences is shown in Figure 1. The great majority of differences were 0.3 mm. or less. The paired readings differed by a mean of 4.9 ± 0.2 per cent of the initial reading. In estimating changes differences greater than 5 per cent were considered significant.

Common hepatic artery. In 12 patients common hepatic arteries ranged from 6.4 to 11.0 mm. in diameter prior to vasopressin therapy. The mean diameter after vasopressin based on 15 measurements was 97 per cent of the mean pre-vasopressin diameter (Table I). After vasopressin the arterial diameter decreased in 4 patients (33 per cent), increased in 2 (17 per cent), but in 6 (50 per cent) remained unchanged (Table II).

Hepatic artery. In 14 patients hepatic arteries and their branches ranged from 1.2 to 9.1 mm. in diameter. The mean diameter of 85 measurements after vasopressin was 107 per cent of the pre-vasopressin level (Table I). After vasopressin 9 patients (64 per cent) showed an increment and 1 showed no change. In 4 the hepatic arteries were constricted (28 per cent). Thirty-six of the 85 individual measurements (42 per cent) increased after vasopressin, while 31 (36 per cent) remained unchanged and 18 (21 per cent) decreased significantly (Table II) (Fig. 2).

Gastroduodenal artery. In 12 patients the diameters of the gastroduodenal arteries ranged from 1.4 to 5.2 mm. The mean diameter of the gastroduodenal arterial branches was 71 per cent of pre-vasopressin diameters, a decrease of 29 per cent (Table I). After vasopressin 11 of the 12 patients showed a significant mean decrease in diameter (92 per cent) and 1 remained unchanged. Thirty-seven of the 45 individual measurements were significantly decreased (82 per cent) (Table II) (Fig. 2, *A* and *B*).

Left gastric artery. In 7 patients the diameters of the left gastric artery ranged from 2.0 to 8.4 mm. The mean diameter of the left gastric artery after vasopressin was 9 per cent smaller than before vasopressin (Table I). After vasopressin 4 of the 7 patients (57 per cent) were significantly decreased, 2 were unchanged and 1 increased (Table II). Six of the 10 measurements were decreased (60 per cent); only 1 was increased (Table II).

Splenic artery. In 9 patients the diameters of the splenic artery and its branches ranged from 2.0 to 11.4 mm. The mean splenic arterial diameter after vasopressin was 86 per cent of its pre-vasopressin size (Table I). After vasopressin 8 of the 9 pa-

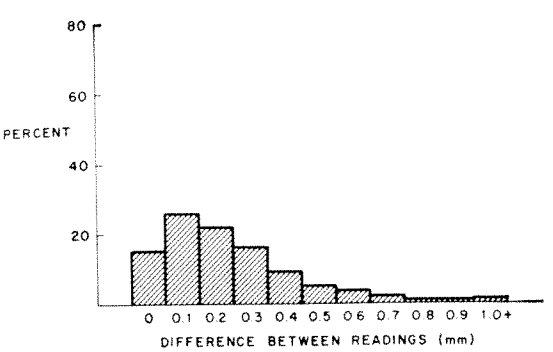


FIG. 1. Consistency of serial measurements of arterial diameter. The distribution of the differences between 305 pairs of measurements is expressed in terms of percentage. More than 81 per cent of the pairs agreed within 0.3 mm.

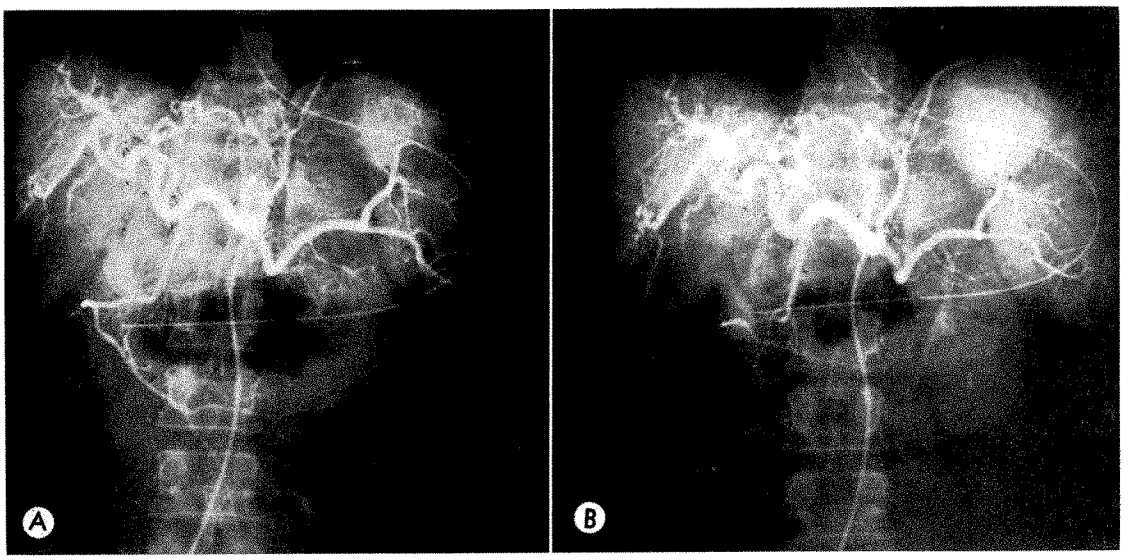


Fig. 2. Celiac axis angiograms (*A*) before and (*B*) after vasopressin infusion illustrate a typical response. After vasopressin, generalized vasoconstriction is noted except for the hepatic artery and its divisions, which are visibly dilated. Arterial diameters were measured at the sites indicated by the black marks on the films.

tients (89 per cent) showed a significant decrease in diameter and 1 remained unchanged. Twenty-five of the 37 individual measurements (68 per cent) were significantly decreased and 11 (30 per cent) were unchanged. Only 1 increased (Fig. 2).

Superior mesenteric artery (SMA). In 8 patients the diameter of the SMA and its branches ranged from 1.2 to 10.6 mm. The mean diameter of all post-vasopressin SMA measurements was 88 per cent of the mean pre-vasopressin value, a mean decrease of 12 per cent (Table 1). After vasopressin 7 of the 8 (88 per cent) showed a significant decrease in diameter; 1 showed an increase. Sixty-six of the 100 individual measurements (66 per cent) were decreased, 19 (19 per cent) were unchanged and 15 (15 per cent) were increased (Fig. 3, *A* and *B*).

Association of Arterial Size to Responses to Vasopressin

Small arteries (<6 mm. in diameter) appeared to be very responsive and large arteries (>6 mm.) appeared to be relatively unresponsive to the intraarterial administration of vasopressin. The common hepatic arteries, which were >6 mm. in diameter in all patients studied, showed few significant changes (Fig. 4). Similarly, hepatic arteries of this size uncommonly showed changes. Hepatic arteries with diameters <6 mm., however, showed a moderate degree of dilatation. Superior mesenteric arteries up to 7 mm. in diameter, and splenic arteries up to 9 mm. in diameter showed constriction after vasopressin. Gastroduodenal and left gastric arteries, which were usually <6 mm. in diameter, all responded with vasoconstriction. Although the smallest arteries tended to show the greatest constriction, the number of data were insignificant to establish this relationship statistically.

Effect of Changes in Arterial Diameter on Blood Flow

According to Poiseuille's law the factor which most affects volume flow is the radius of the vessel since it is raised to the fourth power in the formula. Assuming factors such as perfusion pressure, viscosity and the length of the artery to remain constant, the mean changes in diameter of the various arteries permit calculation of the blood flow through these arteries

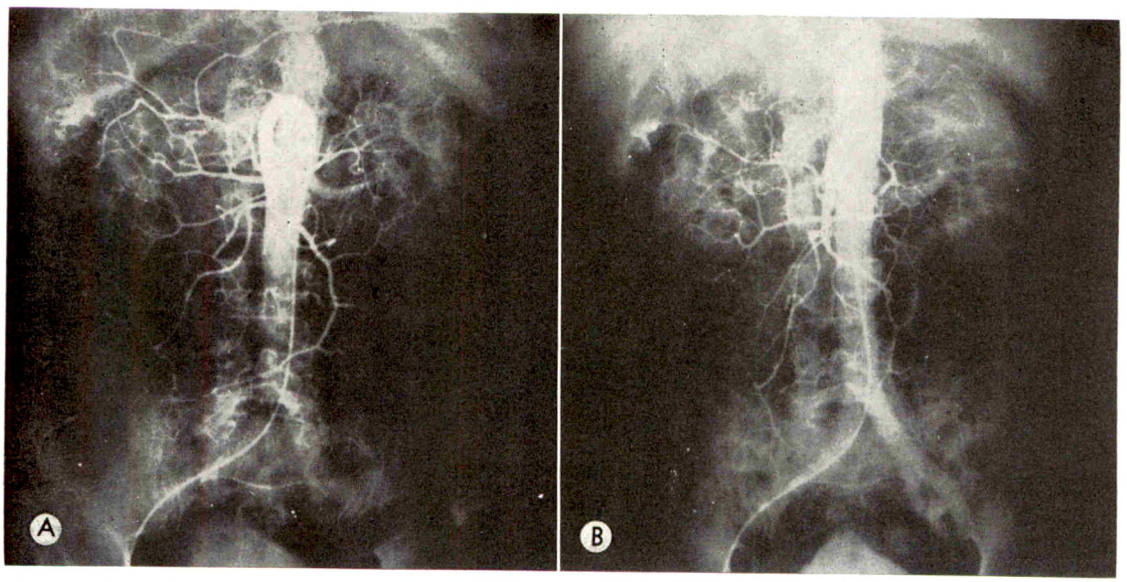


FIG. 3. (*A*) Superior mesenteric arteriogram before vasopressin therapy shows an active bleeding site (upper left) from a diverticulum of the hepatic flexure of the colon. (*B*) Typical response of the superior mesenteric artery to vasopressin (0.2 units per min. for 20 min.). Note the marked decrease in extravasation of contrast material and the decrease in diameter of all branches. The smallest peripheral branches of the superior mesenteric artery have almost disappeared. Reflux of contrast material into the aorta represents increased resistance in the superior mesenteric artery system.

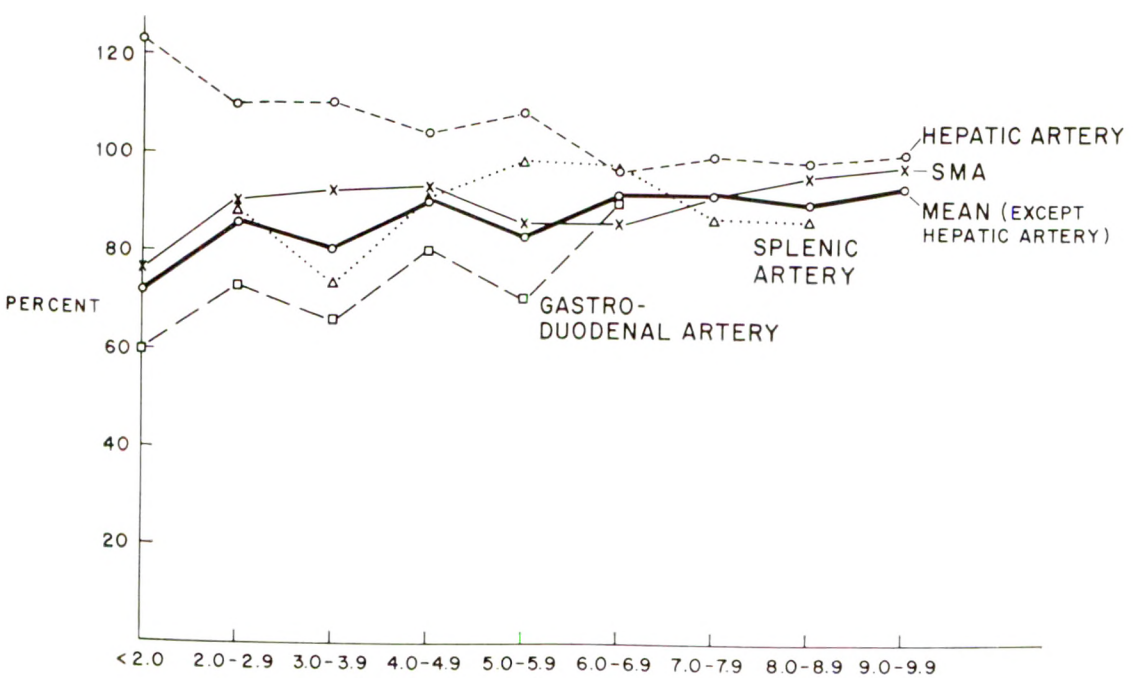


FIG. 4. Relation between arterial size and response to vasopressin. Small branches of the hepatic arterial system showed greater dilatation than the large branches. Hepatic arteries >6 mm. in diameter did not change appreciably. Small radicals of the gastroduodenal, splenic and superior mesenteric arteries (SMA) showed greater vasoconstriction than larger branches. The mean values of these 3 arteries (heavy line) show that, although all sized branches constricted, the smaller branches were more responsive.

(Table III). The small mean increment in diameter of the hepatic arteries of 7.7 per cent resulted in an increment of 34 per cent in blood flow. Gastroduodenal arterial blood flow, which showed the greatest decrease in mean diameter (29.2 per cent) decreased to 25 per cent of pre-vasopressin blood flow.

DISCUSSION

Selective intraarterial vasoconstrictive treatment of gastrointestinal hemorrhage is an exciting and dramatic form of therapy which appears to have great promise.[1,2,4,8] Although most investigators have used vasopressin, some have used other vasoconstrictive agents and/or abbreviated periods of infusion in part, at least, to avoid the dangers of hepatic ischemia.[2]

The present study demonstrates clearly that, although intraarterial vasopressin routinely causes vasoconstriction of the gastroduodenal, left gastric, splenic and superior mesenteric arteries, the hepatic artery usually escapes this effect. In fact, after vasopressin has been infused for at least 15 minutes, most hepatic arteries are slightly *dilated*.

The escape of the hepatic arterial system from the vasoconstrictive effects of vasopressin is not a new observation. Several authors have shown in dogs that continuous intravenous infusions of vasopressin initially induce a decrease in hepatic blood flow, but within a few minutes hepatic blood flow begins to return to normal in contrast to portal blood flow which remains decreased throughout the infusion.[3,5–7] In related studies performed at this institution it has been shown in dogs that infusions of vasopressin into the celiac axis induce an immediate 40–50 per cent decrease in hepatic and splenic arterial blood flow.[10] The splenic arterial blood flow remains depressed, however, while hepatic arterial flow rapidly increases until it is considerably *above* pre-infusion levels. Brant and his associates[2] commented that they too have noted a transient decrease in hepatic arterial blood flow followed by increased hepatic arterial flow during celiac infusions of vasopressin.

Presumably this increase in hepatic arterial blood flow is a compensatory vasodilatory response to decreased portal venous pressure and portal blood flow. Schenck and his associates[9] have shown in man that occlusion of the portal vein results in a large, prompt increase in hepatic arterial flow. Zimmon and Kessler[11] have reported that hepatic arterial flow appears to be autoregulated and responds rapidly to increments and decrements in portal venous blood flow.

In the present study large arteries showed little or no change in diameter, while small arteries appeared progressively more responsive as their size diminished. No measurements were made of the arterioles, which were too small for accurate measurement and which are even more responsive to both neural and humoral stimuli. It was noted that arteries <1 mm. in diameter often became invisible during vasopressin administration, as larger arteries in the same system constricted.

Small changes in arterial diameter cause disproportionately larger changes in the volume of blood flow (Table III). Theoretically, a 50 per cent decrease in arterial diameter will cause a 94 per cent reduction in blood flow through that artery. The largest mean change that we observed was a decrease of 29 per cent in the diameter of the gastroduodenal artery, which resulted in a 75 per cent reduction of gastroduodenal arterial flow. Assuming even more effective vasoconstriction at the arteriolar level, vasopressin infusions may actually achieve even greater reductions in blood flow than our measurements suggest.

The correlation between our angiographic calculations of vasopressin-induced changes of blood flow in man and the direct measurements of blood flow in dogs undergoing vasopressin infusions into the celiac axis are impressive. We calculated a mean increase of 34 per cent in hepatic arterial blood flow in our patients, while a mean increment of 36 per cent was found in

the dogs.[10] We calculated a mean reduction in splenic arterial blood flow of 55 per cent in the patients; a mean decrease of 50 per cent was found in the dogs. Whether these similarities are coincidental or of physiologic significance is not known.

Occasionally paradoxical responses were observed. In 1 patient who received an infusion of vasopressin into the SMA all 10 measurements made showed the arterial diameters to be 10 to 37 per cent *greater* than before vasopressin. Review of the patient's record written at the time of treatment, revealed a note which stated that the branches of the SMA had promptly constricted after vasopressin administration. Whether this represents an aberrant response of the SMA to vasopressin or an error in film labeling can not be resolved at this time. If these paradoxical data do represent such an error, the vasoconstrictive effects of vasopressin will have been even more consistent and the mean decrease in diameter of the SMA even greater than we reported.

CONCLUSION

Differential responses of different arterial systems to different humoral and neural stimuli are well established. The escape of the hepatic arterial system from the sustained vasopressin-induced vasoconstriction that characterizes the rest of the celiac arterial branches probably represents a compensatory response to the decrease in portal blood flow. When portal blood flow to the liver is decreased, the hepatic arterial blood flow increases to maintain hepatic blood flow at a liver-sustaining level.[9,11] That this regulatory adjustment is teleologically important is demonstrated by the fact that the hepatic artery escapes the potent vasoconstrictive effects of vasopressin and actually *dilates* despite the continuous administration of this constrictor substance. The control mechanisms for the regulation of hepatic blood flow are critical to understanding the derangements of portal hypertension, and vasopressin may provide one route for exploring this unknown area.

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DELAYED DUCTAL DRAINAGE: AN ENDOSCOPIC PANCREATOGRAPHIC SIGN OF CARCINOMA OF THE HEAD OF THE PANCREAS*

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ENDOSCOPIC pancreato-cholangiography is being used to diagnose pancreatic and biliary diseases.[2–4, 9–11, 15]

The purpose of this paper is to describe a roentgenographic sign thought to be diagnostic of carcinoma of the head of the pancreas, *i.e.*, a dilated *and* obstructed pancreatic duct with delayed drainage of contrast medium.

METHOD

The premedication and technique of retrograde pancreato-cholangiography are similar to those described by Vennes and Silvis.[15] A JF-B Olympus Duodenoscope with a CLE Olympus light source was used for all of the examinations. Glucagon (0.5–1.0 mg.) injected intravenously or atropine (0.4 mg.) injected intravenously at the time of endoscopy causes sufficient duodenal relaxation for the procedure to be carried out.

The cannulation procedure is performed with intermittent fluoroscopic monitoring and simultaneous video taping. The availability of immediate video replay provides repeated assessment of gross pancreatic duct opacification and at the same time reduces radiation exposure to all concerned.

When it has been established fluoroscopically that the pancreatic duct and/or biliary tract is adequately filled, the patient is placed in a left lateral decubitus position with the cannula maintained in the papilla of Vater. The endoscope is then removed and roentgenograms are obtained in the left lateral decubitus, left posterior oblique and the anteroposterior positions consecutively. A "floating-top" fluoroscopic table facilitates rapid patient positioning. The films are viewed immediately and additional roentgenograms are obtained if necessary to better demonstrate specific segments of the pancreatic duct. Following this film series, the patient is placed in a *right* lateral decubitus position to promote drainage of the pancreatic duct. Ten and 20 minute delayed studies are obtained with the patient returning to the anteroposterior position during film exposure. If complete pancreatic duct drainage is demonstrated on the initial roentgenograms, the study is terminated. In the event that contrast medium remains in the pancreatic duct, 30 minute to 1 hour interval studies are obtained for several hours to determine drainage rate.

REPORT OF CASES

CASE I. C.B. was an 83 year old man with obstructive jaundice (bilirubin 6.0 mg. per cent) and weight loss. An upper gastrointestinal barium examination was normal. Retrograde pancreatography demonstrated a dilated and obstructed pancreatic duct that did not drain significantly until 60 minutes after filling (Fig. 2). Surgery was refused. The patient's condition deteriorated clinically with anorexia and weakness. Death occurred in 2 weeks with a temperature of 39° C. the last 36 hours.

Autopsy demonstrated carcinoma of the head of the pancreas involving the main pancreatic duct. There was no tumor extension beyond the pancreas, but an abscess was found in the head of the pancreas with purulent material in the main pancreatic duct.

CASE II. C.N. was a 79 year old male nursing home resident who was noted to be jaundiced (bilirubin 5.8 mg. per cent). His general physical condition was good except for confusion attributed to cerebral arteriosclerosis. Retro-

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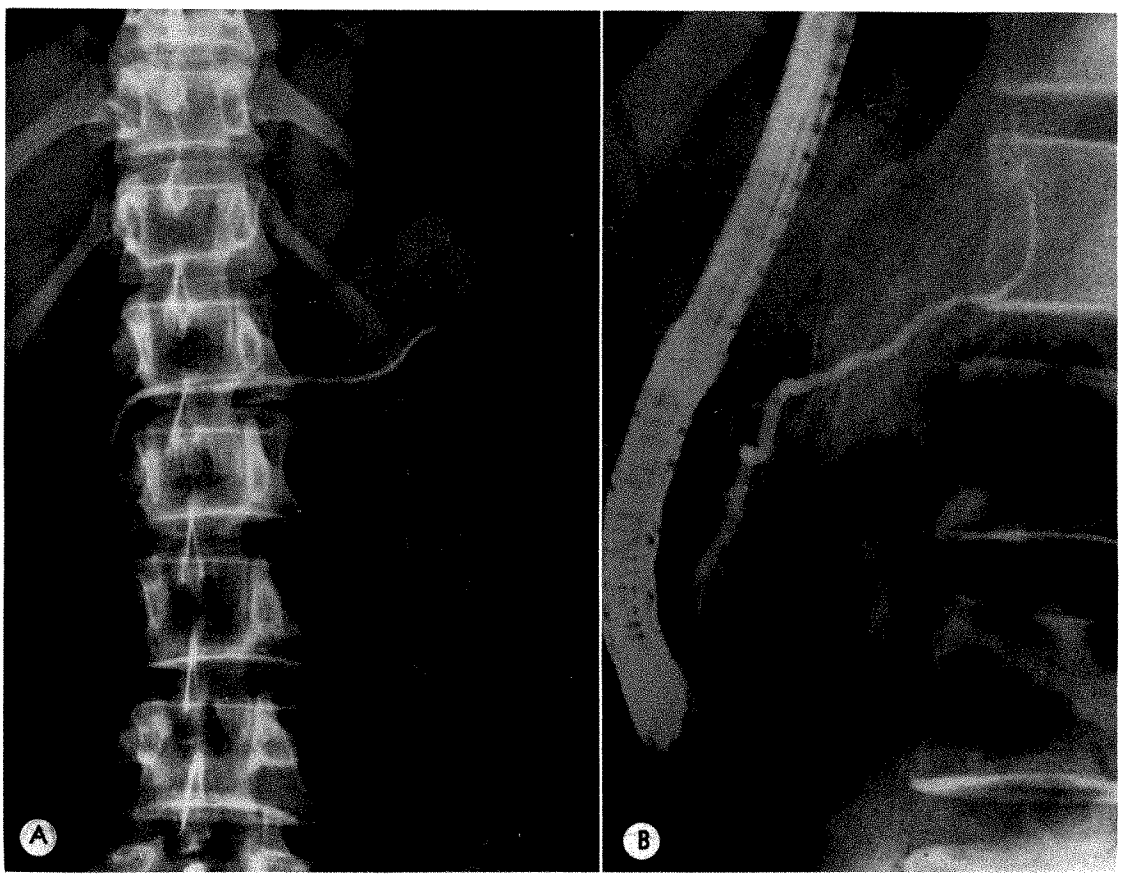


FIG. 1. (*A*) Anteroposterior and (*B*) lateral projections of the normal pancreatic duct. Small spherical lucencies within the duct proved to be gas bubbles.

grade pancreatography revealed a dilated main pancreatic duct that did not drain in a normal fashion (Fig. 3). Subsequent barium examination of the upper gastrointestinal tract demonstrated minimal enlargement of the medial side of the duodenal loop without mucosal abnormality. In view of the patient's age and mental status, the family and his physicians decided against laparotomy. Three weeks later the patient expired.

Autopsy revealed a very small, well-localized tumor of the pancreatic duct in the head of the pancreas. The location of the ductal carcinoma was such that it obstructed the duct of Wirsung and the dilated common bile duct at their junction just proximal to the papilla. The papilla was not involved. The cancer was resectable and very likely a curable lesion. There were no changes of chronic pancreatitis throughout the gland parenchyma or duct system. The right coronary artery was completely occluded, but there was no evidence of a myocardial infarction.

RESULTS

Sixteen patients with carcinoma of the pancreas have been examined endoscopically. Fifteen of these patients had serum bilirubin elevations greater than 5 mg. per cent and therefore intravenous cholangiography was not attempted. In 5 patients retrograde pancreatograms could not be obtained. In 4 of these 5 patients, total obstruction of the papilla prevented retrograde contrast medium injection despite apparent cannulation. The other patient had tumor invasion into the duodenum.

Three patients had partial retrograde filling of the pancreatic duct system along with opacification of the biliary tree. In 1 of these patients an obstruction was dem-

onstrated in the pancreatic duct in the mid-body of the pancreas. Widely metastatic carcinoma was found at laparotomy. Two patients with extensive tumors arising from the head of the pancreas demonstrated filling of only a short segment of the terminal pancreatic duct and complete opacification of the biliary tract with marked dilatation of all biliary radicles.

One patient had obstruction of retrograde flow into both the pancreatic and biliary system, except for the distal 1 cm. of the pancreatic duct. A 3 cm. tumor occluded the pancreatic duct in the head of the organ and was completely removed at surgery. There were no metastases.

The remaining 7 patients with carcinoma of the head of the pancreas demonstrated a well-filled, dilated pancreatic duct which failed to empty on the 20 minute delayed roentgenograms (Fig. 4–6). The protocol to enhance drainage of the pancreatic duct as is described in Method was followed in each case.

We encountered 3 endoscopic-retrograde patterns in our 16 malignant tumors: (A) no retrograde filling of either biliary or pancreatic ductal system due to extensive tumor blocking the papilla (5 patients); (B) partial filling of the pancreatic duct distally and some filling of the biliary ducts

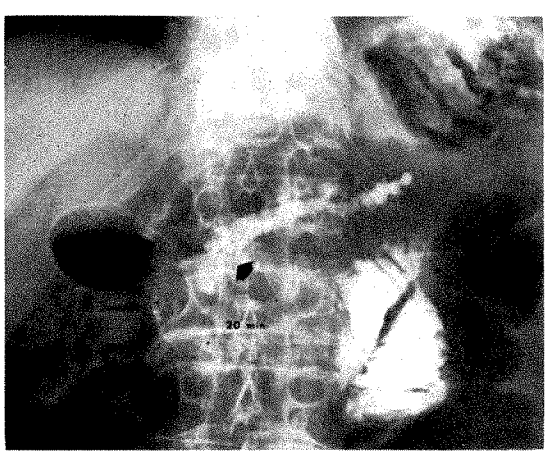


Fig. 2. Case 1. Twenty minute delayed film following retrograde pancreatography. The arrow indicates the opacified pancreatic duct in the region of the head of the pancreas. The opacified segment of the duct is seen to be markedly dilated with alveolarization in the distal body and tail of the pancreas. The tumor was located between the proximal end of the opacified pancreatic duct and the medial margin of the gas shadow of the loop of the duodenum.

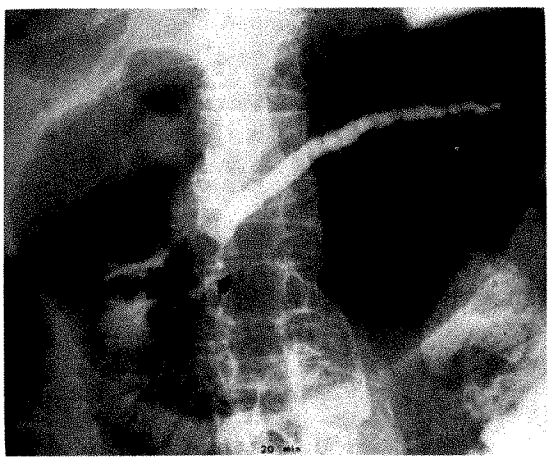


Fig. 3. Case 11. Twenty minute delayed film following retrograde pancreatography. The arrow indicates the tumor location which has infiltrated and narrowed the proximal main pancreatic duct causing the distal obstruction.



Fig. 4. Twenty minute delayed film following retrograde pancreatography. The curved arrow demonstrates the slightly dilated, non-draining obstructed main pancreatic duct. The small arrows delineate the margins of the tumor in the head of the pancreas. The "puddling" of contrast medium within the head of the pancreas is felt to represent dilated ducts secondary to a partially obstructed accessory ductal system.

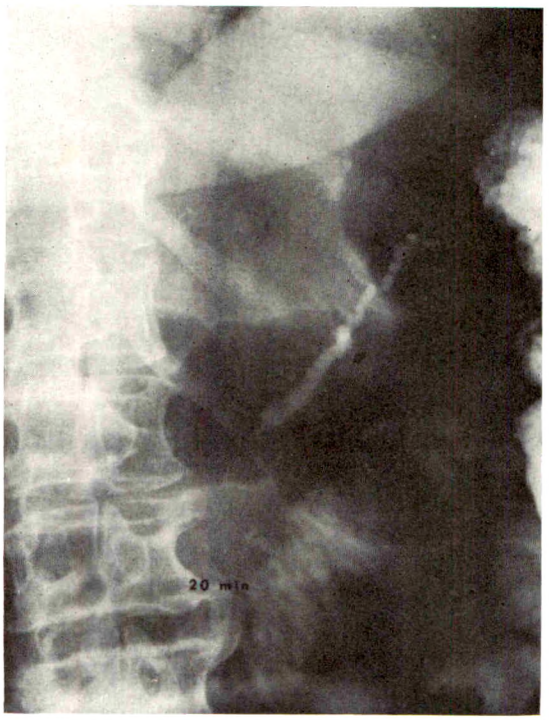


FIG. 5. Twenty minute delayed film following retrograde pancreatography. Left posterior oblique projection. The arrows indicate the dilated, serpiginous, obstructed main pancreatic duct.

(4 patients); (C) complete filling of the pancreatic duct revealing a dilated and poorly draining duct system (7 patients).

The patients in Group C show anatomic roentgenologic characteristics indistinguishable from chronic pancreatitis, *i.e.*, a dilated, irregular pancreatic duct. Twenty such cases with chronic pancreatitis have been examined by endoscopic pancreatography. Figure 7, *A* and *B*, demonstrates the typical dilated duct which may be seen in a patient with carcinoma or chronic pancreatitis. The differentiation can be made, however, on the basis of the complete drainage seen on the 20 minute delayed study. This case was documented surgically at which time the pseudocyst that is evident in Figure 7*A* was drained.

All of the carcinomas in this series have been confirmed histologically, either by biopsy or autopsy. Changes of chronic pancreatitis were also found in 1 of the cancer patients. In 6 of the patients with retrograde filling (Group C), no extrapancreatic tumor extension was present. Two of these 6 patients had well-localized, small, resectable malignancies. This indicates that a positive finding of obstruction does not necessarily indicate inoperability.

The procedure was clinically well tolerated in all instances. However, pancreatic abscesses which were discovered at autopsy in 2 patients may have been related to the cannulation.

DISCUSSION

Many authors have discussed the differentiation of chronic pancreatitis from carcinoma by means of the pancreatogram.[5–8,11–15] The general appearance of both conditions is that of a dilated, irregular-shaped duct. In some instances the presence of calcifications and/or pseudocyst formation favors pancreatitis. Preoperative assessment of the drainage rate of the contrast medium from the pancreatic duct system to distinguish malignant from benign diseases has not been previously reported.

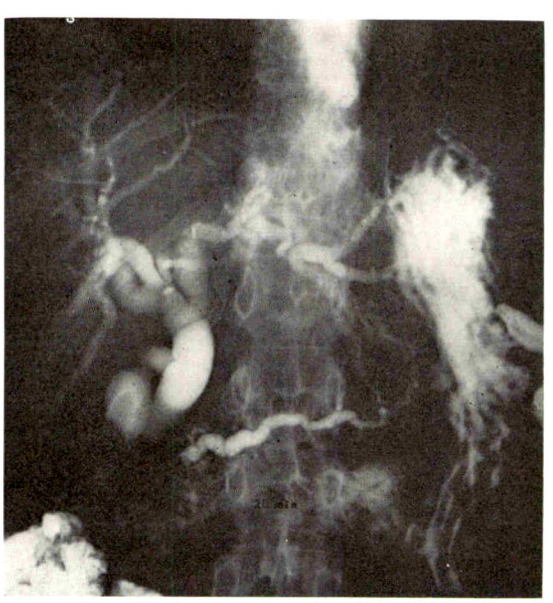


FIG. 6. Twenty minute delayed film following retrograde endoscopic filling of both the pancreatic and biliary systems. The arrows indicate the dilated, obstructed main pancreatic duct.

Using operative pancreatography, Artigas and La Calle[1] and Howard and associates[7,8] based the diagnosis of chronic pancreatitis on the rapid drainage of contrast medium into the duodenum from a dilated and often distorted pancreatic duct system. This contrasted with the complete obstruction (delayed drainage) noted in carcinoma of the pancreas. They were unable to positionally drain their patients, however, because of the operative technique being employed. Details regarding the duration of obstruction, *i.e.*, drainage rate, in their patients were not mentioned. Kasugai *et al.*[9] have noted that in some elderly patients the normal pancreatic duct retains the contrast medium as long as 10 minutes after retrograde filling. They made no mention of the drainage characteristics in 9 cases of carcinoma.[10] Ogoshi *et al.*[12] recently described 21 patients with carcinoma of the pancreas studied by endoscopic pancreatography. Ten of these patients had a stenotic main pancreatic duct with distal dilatation similar to those demonstrated in Figures 4–6. Drainage techniques with delayed roentgenograms were not used to distinguish carcinoma from chronic pancreatitis. They had previously mentioned a "rat-gnawn" appearance as being typical of chronic pancreatitis.[11] Our experience and that of Okuda *et al.*[13] in commenting on endoscopic pancreatography suggest that improvement in its diagnostic usefulness might depend upon such factors as the speed at which the contrast medium is cleared from the pancreatic duct. This sign is what we now report as being characteristic for carcinoma of the head of the pancreas. The delayed ductal drainage is presumably on the basis of tumor obstruction within the pancreatic duct. In no instance of normal or noncalculous benign disease have we seen significant opacification persisting on the 20 minute delayed film (Fig. 7, *A* and *B*).

In 2 patients abscesses have been noted in the pancreas at autopsy. This may be related to obstruction of normal drainage. Our 2 cases with an abscess occurred in patients having the most prolonged retention of contrast medium we have observed. While antibiotics may be helpful in preventing this complication, it is likely that surgical decompression of an obstructed, non-draining duct would be necessary to prevent sepsis.

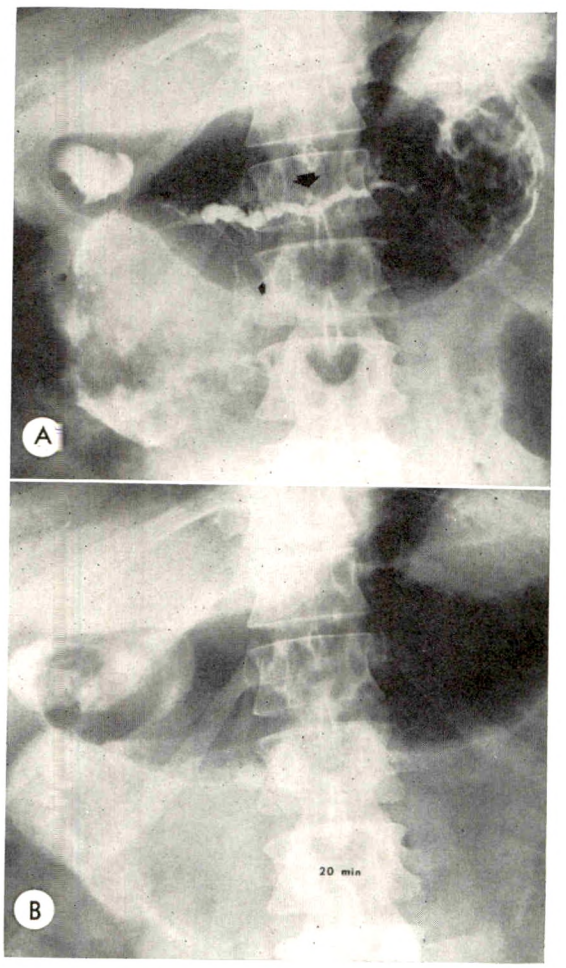


Fig. 7. (*A*) Chronic pancreatitis with pseudocyst. Immediate post-retrograde pancreatography film with the largest arrow demonstrating a dilated, serpiginous main pancreatic duct with some accessory duct filling. Smaller arrows outline the margin of a mass (pseudocyst) through which the course of the main pancreatic duct can be identified. (*B*) Twenty minute delayed film demonstrating complete drainage of the main pancreatic duct.

We believe that endoscopic pancreatography has great potential in the differential diagnosis of diseases of the pancreas. The finding of delayed ductal drainage has been

associated with 7 cases of carcinoma of the head of the pancreas and no benign lesions. Accuracy in "ruling-out" carcinoma remains to be determined.

SUMMARY

Sixteen patients with carcinoma of the pancreas have been examined by duodenoscopy and varying degrees of retrograde pancreatography. A standardized roentgenographic technique consisting of immediate filming after injection and 20 minute follow-up studies was used to assess contrast medium retention. On the basis of this standardized methodology, failure of the pancreatic duct to drain on a 20 minute delayed film appears to be indicative of carcinoma of the head of the pancreas.

Benign noncalculous chronic pancreatitis may have a pancreatographic appearance anatomically similar to carcinoma, but is distinguished by its non-obstructed draining characteristics. All of the patients with delayed ductal drainage had confirmed adenocarcinoma of the head of the pancreas. Two of these lesions were small, resectable carcinomas.

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CYSTADENOMA OF THE PANCREAS*

A REAPPRAISAL OF ANGIOGRAPHIC FINDINGS

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PANCREATIC cystadenomas are rare primary neoplasms. Only 251 cases had been reported by 1971 when Lewis and Dormandy[14] reviewed the literature. The classical angiographic description includes: (1) displacement of major vessels; (2) the presence of large feeding arteries; and (3) a "tumor" blush sometimes associated with pooling of contrast.[1,3,4,7,10,14,16,22,23] The report by Abrams *et al.*[1] of 3 cases of benign cystadenomas studied angiographically included 1 which was relatively avascular. This is the only previously reported case the present authors have been able to find of a pancreatic cystadenoma which did not have a hypervascular pattern.

Recently, however, we have seen 3 cases which were hypovascular and, therefore, difficult to differentiate by arteriography from a pseudocyst of the pancreas.

REPORT OF CASES

CASE I. (Fig. 1, *A–C.*) This 29 year old female complained of constant midepigastric pain of 2 weeks' duration. The pain radiated to the back and left upper quadrant and was associated with nausea and vomiting. She had experienced similar symptoms for a short time approximately 6 months earlier. There was no history of abdominal trauma, pancreatitis or alcoholism. On admission her temperature was 102.2°F. She was obese, but an upper abdominal mass could be palpated. Pertinent laboratory data included normal white blood cell count, hemoglobin, serum amylase, SGOT, bilirubin, glucose and blood urea nitrogen. The serum alkaline phosphatase and LDH were slightly elevated.

An upper gastrointestinal examination demonstrated a large retrogastric mass displacing the stomach anteriorly and the ligament of Treitz inferiorly. There were no demonstrable varices. Intravenous urography and oral cholecystography were normal. Selective celiac and superior mesenteric arteriography demonstrated a large avascular mass in the region of the body and tail of the pancreas. The splenic vein was occluded and there were large omental venous collaterals.

At surgery a large, well-encapsulated, cystic mass was found and totally removed. This mass, which filled the lesser sac and was attached to the pancreas by only a pedicle, contained several hundred cc. of thick mucoid material and was composed of multiple cystic cavities. Histologic sections demonstrated the well-differentiated, uniform cuboidal epithelium typical of benign pancreatic cystadenoma.

CASE II. (Fig. 2, *A–D.*) An abdominal "lump" was noted by this 40 year old white female 3 months before admission. She denied other symptoms and there was no history of abdominal trauma, pancreatitis or alcoholism. A slightly tender, large left upper quadrant mass was palpable. Except for a slightly elevated sedimentation rate of 25, all blood chemistries were normal, as were the hemoglobin and white blood cell count.

An upper gastrointestinal examination revealed a very large left upper quadrant mass displacing the stomach anteriorly and slightly to the right. The ligament of Treitz was depressed. Varices were not demonstrated.

Selective celiac and superior mesenteric arteriography were performed and revealed marked displacement of both the superior mesenteric artery and normal pancreatic tissue to the right. The splenic artery and spleen were elevated. No vessels were seen within the mass. Large splenic collateral veins were opacified indicating splenic vein obstruction.

A tense encapsulated mass (18×12×13 cm.) was surgically removed from the tail of the pancreas. The splenic vein was obstructed by pressure from the mass, but it was not thrombosed. The mass contained 1,500 cc. of brownish fluid. Although it was grossly unilocular, histologic sections revealed multiple loculated cysts lined with columnar epithelium. There were no

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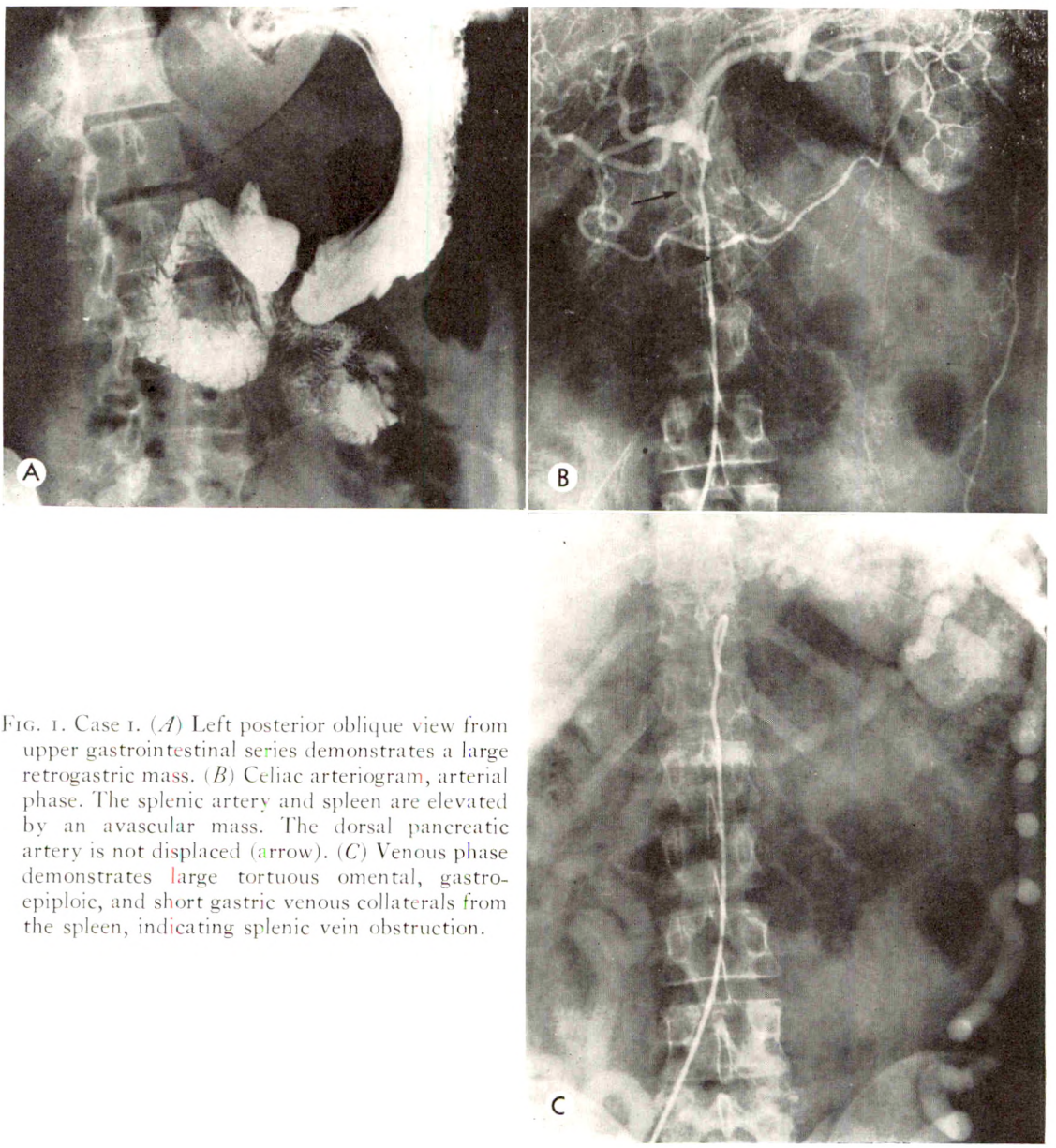


FIG. 1. Case I. (*A*) Left posterior oblique view from upper gastrointestinal series demonstrates a large retrogastric mass. (*B*) Celiac arteriogram, arterial phase. The splenic artery and spleen are elevated by an avascular mass. The dorsal pancreatic artery is not displaced (arrow). (*C*) Venous phase demonstrates large tortuous omental, gastroepiploic, and short gastric venous collaterals from the spleen, indicating splenic vein obstruction.

elements suggesting malignancy and pancreatic cystadenoma was the pathologic diagnosis.

CASE III. (Fig. 3, *A* and *B*.) One month of increasingly severe left upper quadrant pain was the admitting complaint of this 17 year old black female. She denied other symptoms and had no history of pancreatitis, abdominal trauma or alcoholism. Physical examination revealed a firm tender mass in the left upper abdomen, initially thought to be spleen. Hematologic and serum studies were all within normal limits.

The stomach was displaced medially and anteriorly on an upper gastrointestinal examination and an excretory urogram demonstrated depression of the left kidney.

Arteriography revealed elevation of the spleen and the splenic artery and vein. The mass contained a few small vessels and there was no early venous filling. A 12 cm. spherical mass was removed from the body and tail of the pancreas and was histologically typical of pancreatic cystadenoma.

DISCUSSION

Pancreatic cystadenomas comprise approximately 10 per cent of all pancreatic

cysts, and occur 9 times more often in females than in males. They most often occur during the fourth to sixth decades, although cases have been reported from adolescence to old age.[1,2,5,6,12,14,15] Three quarters of patients are less than 60 years old. Therefore, most patients are younger than the usual case of pancreatic carcinoma, but are similar in age to patients with pancreatic pseudocyst.

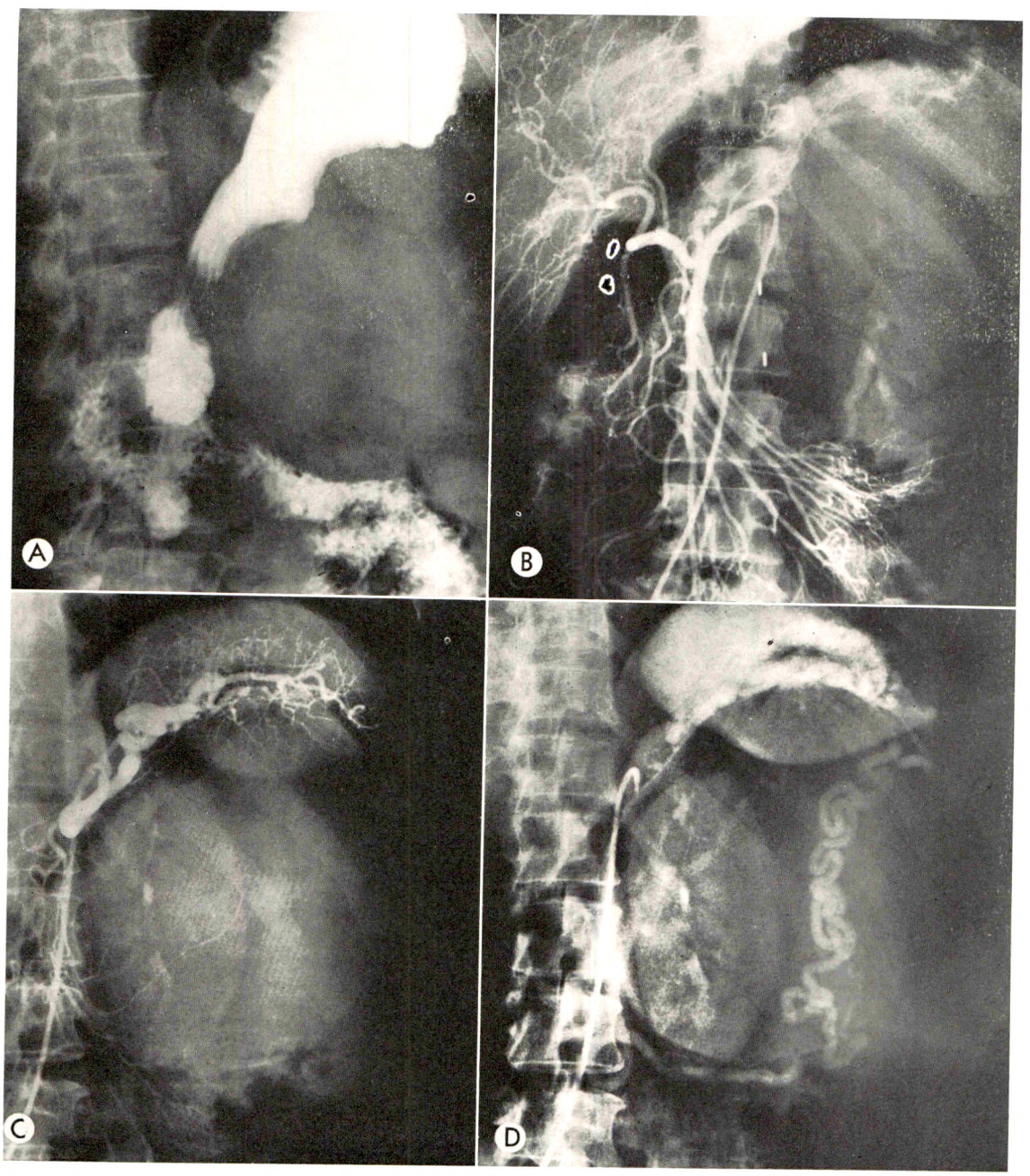


Fig. 2. Case 11. (*A*) Left posterior oblique view from upper gastrointestinal series demonstrates the stomach to be anteriorly and superiorly displaced by a large mass. (*B*) Superior mesenteric arteriogram demonstrates displacement of the superior mesenteric artery and its jejunal branches by a left upper quadrant avascular mass. The hepatic artery is normal. (*C*) Splenic injection also failed to reveal vascularity within the mass. Pancreatic vessels are displaced medially and the splenic artery and spleen are elevated. (*D*) Splenic vein obstruction is indicated by large retroperitoneal splenic venous collaterals.

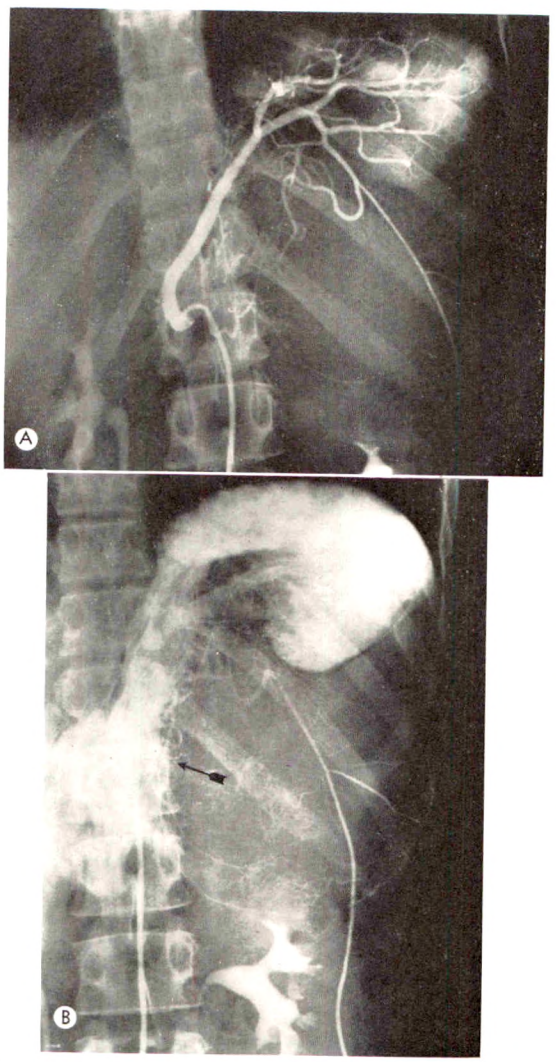


FIG. 3. Case III. Selective splenic arteriogram. (*A*) The splenic artery and spleen are elevated and the left kidney is depressed by a large left upper quadrant mass. (*B*) A later roentgenogram demonstrates minimal vascularity of the mass. Pancreatic tissue (arrow) is medially displaced. The splenic vein is patent and draped over the mass.

A palpable abdominal mass is present in approximately 80 per cent of the cases.[2] More than half of the patients complain of pain and/or dyspepsia. Symptoms may be present for many months, indicating the slow growth of the tumor. Jaundice is rare, as is gastrointestinal bleeding, which may be from gastric varices secondary to splenic vein obstruction or duodenal ulceration.[15,19] A history of abdominal trauma, alcoholism or pancreatitis is not usually obtainable.[2,8]

Diabetes is present among 10–20 per cent of these patients, but an explanation for this association is still forthcoming. The amount of endocrine tissue destroyed or compressed by the lesion is not extensive enough to explain the diabetes. The pancreatic exocrine function is not compromised.[1,5,6,14,21,23]

Soloway,[21] in his excellent review of 30 cases, reported a high incidence of associated metabolic and endocrine abnormalities. These include not only diabetes, but also obesity, sterility and infertility, hypertension, and thyroid dysfunction. Furthermore, he noted an increased incidence of benign and malignant neoplasms in other organs. The most common is breast carcinoma. There is as yet no clear explanation for these associations.

Biochemical studies are characteristically unrevealing. Serum amylase and lipase are usually normal, although these enzymes may be present within the cyst fluid.[2,11] Bilirubin and alkaline phosphatase levels are also usually normal, unless there is common duct obstruction, which occurs infrequently.

The majority of pancreatic cystadenomas and cystadenocarcinomas occur in the body or tail of the pancreas, although they may occur in the pancreatic head.[1,2,8,14] The average size is approximately 10 cm., but they range from incidental microscopic findings at necropsy to massive lesions as in Case II.

Calcification of the neoplasm is a diagnostically useful roentgenographic finding, but it is uncommon. Approximately 18 per cent of these lesions demonstrate stromal calcification pathologically, but in only 10 per cent or less is stromal or capsular calcification visible roentgenographically.[1,5,12,15] Haukohl and Melamed[12] described 2 cases of roentgenographically evident calcification and discussed the different patterns that are seen in lesions of the body and tail *versus* those of the head of the pancreas.

There is general agreement that total surgical removal of pancreatic cystadenomas is indicated. Marsupialization with internal drainage is inadequate both because of the multiple cavities which are not drained by this procedure and because of the malignant potential of these lesions. Partial excision is also inadequate because of the possibility of fistulae formation.[5,14]

The typical gross appearance of a pancreatic cystadenoma is that of a well-encapsulated, multilobulated, multicystic mass.[1,5,6,14,22] Those arising on the surface of the pancreas generally have fewer cysts than those arising within the gland substance.[14] It is not unusual for the cystadenoma to be connected to the pancreas by only a small pedicle.[6]

The nature of the cyst fluid varies in consistency from serous to mucoid, and in color from light yellow to red or dark brown, if there has been recent or remote hemorrhage.[15] The lining of the cyst wall is cuboidal or columnar epithelium. Most authorities believe that the presence of gross papillary infoldings of the epithelium indicates malignant potential of pancreatic cystadenomas. In some cases microscopic examination reveals an area of cystadenocarcinoma within an otherwise benign cystadenoma, causing speculation that cystadenocarcinomas arise within the benign tumor rather than *de novo*.[5,11,14] Some authors believe that the presence of gross papillary infoldings of the epithelium, even without cellular abnormalities, is sufficient for the diagnosis of malignancy.[6] Pancreatic cystadenomas are twice as common as cystadenocarcinomas.

Roentgenographic evaluation of these lesions begins with the abdominal plain roentgenogram. This may demonstrate an upper abdominal mass and, as previously noted, may reveal calcification. Barium studies are also useful in demonstrating and defining a mass in the region of the pancreas. There may be compression and displacement of the stomach, duodenum, transverse colon and splenic flexure. Gastric varices occasionally are seen when there is splenic vein obstruction. Intravenous urography may reveal depression of the left kidney. Unfortunately, none of these findings are specific; rather, they simply indicate the presence of a mass originating in the pancreas.

The typical hypervascular arteriographic findings of pancreatic cystadenoma have been described. However, as indicated by our 3 cases, such a pattern is not necessarily present. Two cases demonstrated no vessels within the tumor and 1 was hypovascular. The presence of a pancreatic mass was arteriographically demonstrated by displacement of contiguous major vessels and organs. Splenic vein obstruction was present in 2 cases and was indicated by large retroperitoneal and omental venous collaterals.

Angiographic demonstration of a large avascular pancreatic mass has been thought to be virtually diagnostic of a pancreatic pseudocyst.[9,18] The absence of vascularity of such a mass has heretofore been considered a feature which excludes pancreatic cystadenoma. The cases reported here indicate that a cystadenoma may not be excluded on the basis of a hypovascular arteriographic pattern. Arteriography can be used to differentiate between cystadenoma and pseudocyst only when the mass has the hypervascular pattern seen in cystadenoma.

When arteriography defines a large spherical avascular or hypovascular pancreatic mass, differentiation between a pseudocyst and a cystadenoma will rest on clinical grounds.

A history of previous pancreatitis, abdominal trauma or alcoholism strongly favors pseudocyst.[2,8,20] A male patient is much more likely to have a pseudocyst. Conversely, in a young or middle aged female without any conditions predisposing to pseudocysts, cystadenoma is a likely diagnosis in spite of the avascular nature of a pancreatic mass.

The explanation for the hypovascular pattern in the present 3 cases is not known. The cystadenoma in Case II was virtually

unilocular and therefore contained fewer septa than usual, which may account for the decreased vascularity. However, the lesions in Cases I and III were typically multilocular, and they too were without the usual hypervascular pattern. It is possible that hypovascularity is not unusual in cystadenoma, but rather that such lesions have been mistakenly diagnosed as pseudocysts and internally drained rather than excised and histologically studied.

SUMMARY

Three cases of markedly hypovascular cystadenomas of the pancreas are reported. This appearance has previously been thought to be extremely rare. Angiographically these cases were indistinguishable from pancreatic pseudocysts.

The clinical features are helpful in the differential diagnosis of large hypovascular pancreatic masses. A middle aged female without a history of pancreatitis, alcoholism or abdominal trauma, with a large hypovascular mass in the pancreas is likely to have cystadenoma.

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ANGIOGRAPHIC DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF ISLET-CELL TUMORS*

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TEL AVIV AND HADERAH, ISRAEL

SINCE the first angiographic demonstration of an islet-cell tumor by Olle Olsson in 1963[12] the number of reported cases has increased annually. Approximately 100 cases have been reported so far.[1,2,5,7,8] However, the question of the diagnostic accuracy of this method is still unsettled, because many of the authors only reported individual cases or a series of positive cases, without referring to false positive and false negative findings, or to angiographic appearances simulating or resembling islet-cell tumors.

Most series attempting to define the diagnostic accuracy of angiography in islet-cell tumors refer only to surgically or pathologically proven cases. In these series the rate of positive angiographic diagnosis is 20–90 per cent.[1–3] There is no series reporting the total experience of angiography in the diagnosis of islet-cell tumors.

The present report includes all cases referred for angiography with the clinical suspicion of islet-cell tumor whether or not such a tumor was eventually confirmed. We have also included cases in which angiography was performed for any reason and angiographic features closely simulating an islet-cell tumor were noted.

MATERIAL AND METHOD

This series includes 20 patients studied within the period 1966–1971 at the Chaim Sheba Medical Center. The patients were divided into 4 separate groups (Table I): Group A included all patients with surgically verified islet-cell tumors; Group B were those patients with a false positive diagnosis; Group C included all patients with angiographic findings simulating or resembling islet-cell tumors, in whom a correct angiographic diagnosis was made; and Group D included patients suspected initially of having an islet-cell tumor with negative angiography and a negative final clinical diagnosis.

TABLE I

NUMBER OF PATIENTS IN EACH GROUP UNDERGOING ANGIOGRAPHY

Group	Definition of Group (see text)	Number of Patients
A	Surgically verified	7
B	False positive	4
C	Simulating i.c.t.	4
D	Negative	5
Total	All cases	20

i.c.t. = islet-cell tumor.

All patients had selective celiac and superior mesenteric angiography with separate injections. Supraselective injections, gastric insufflation and subtraction were used in some cases. The description of the angiographic features is referred to in terms of arterial, capillary and portovenous phases rather than in seconds. We prefer this term of reference because the time of appearance of the capillary and portovenous phases is a function of the location of the catheter, the quantity of contrast material injected, the individual pattern of vascularization and the size of the spleen, which vary from patient to patient.

The indications for arteriography in our patients are given in Table II.

RESULTS

GROUP A

Of 7 patients in this group there were 4 (Cases 1–4) of benign beta-cell tumor, 1

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TABLE II

INDICATIONS FOR AND RESULTS OF ANGIOGRAPHY IN THE DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF ISLET-CELL TUMORS

Case No.	Group (see Table I)	Indications for Angiography	Results of Angiography	Final Diagnosis
1	A	Hypoglycemia	Positive	Beta-cell adenoma at surgery
2	A	Hypoglycemia	Positive	Beta-cell adenoma at surgery
3	A	Hypoglycemia	Positive	Beta-cell adenoma at surgery
4	A	Hypoglycemia	Suspected i.c.t.	Beta-cell adenoma at surgery
5	B	Hypoglycemia	False positive	Aberrant spleen at surgery
6	C	Hypoglycemia	Simulating i.c.t.	Multiple hemangiomata of pancreas at surgery
7	C	Hypoglycemia	Simulating i.c.t. (1)	No surgery
8	C	Hypoglycemia	Simulating i.c.t. (2)	No surgery
9	D	Hypoglycemia	Negative	No surgery (diabinese coma)
10	D	Hypoglycemia	Negative	No surgery
11	D	Hypoglycemia	Negative	No surgery
12	D	Hypoglycemia	Negative	No surgery
13	D	Hypoglycemia	Negative	Surgery—no tumor found
14	A	Zollinger-Ellison syndrome suspected	False negative	4 mm. non-beta-cell adenoma at surgery
15	B	Zollinger-Ellison syndrome suspected	False positive	Duodenal ulcer penetrating into pancreas
16	B	Zollinger-Ellison syndrome suspected	False positive	Duodenal ulcer penetrating into pancreas
17	A	Left upper quadrant tumor	Positive	Giant benign nonfunctioning alpha-cell tumor at surgery
18	A	Right upper quadrant tumor	Positive	Malignant nonfunctioning islet-cell tumor at surgery
19	B	Abdominal pain, hepatomegaly	False positive	Hemangioma of pancreas at surgery
20	C	Carcinoma of stomach, metastases	Simulating i.c.t.	Metastatic tumor in pancreas at surgery

i.c.t. = islet-cell tumor.
† Left adrenal and splenic vein on end (see text).
† Overstaining due to supraselective injection (see text).

(Case 14) of benign non-beta-cell tumor, 1 (Case 17) of benign nonfunctioning alpha-cell tumor and 1 (Case 18) of malignant nonfunctioning undefined islet-cell tumor.

Of the 4 beta-cell tumors 3, localized in the tail of the pancreas measuring 1.5–2 cm., were correctly diagnosed by angiography (Fig. 1; and 2). A fourth tumor, localized in the head of the pancreas measuring 4 mm. in diameter, was suspected on angiography but not positively diagnosed.

All 3 cases positively diagnosed showed a network of small vessels in the early arterial phase (Fig. 1*A*). This developed in the capillary phase into a round well defined dense shadow (Fig. 1*B*; and 2*A*). In 2 cases this shadow disappeared at the end of the portovenous phase, while in 1 it persisted beyond the end of the portovenous phase (Fig. 1*D*). Displacement of the pancreatic artery was not seen and venous drainage from the tumor was not demonstrated.

The fourth insulinoma (Case 4) was not positively diagnosed due to its small diameter. For the same reason the 4 mm. diameter non-beta-cell tumor localized in the head of the pancreas (Case 14) was not diagnosed angiographically, this being the only false negative case. Both nonfunctioning tumors were very large and both were positively diagnosed by angiography

islet-cell tumors: during the celiac injection the catheter was dislodged and part of the opaque medium entered the phrenic artery. An oval shadow appeared in the capillary phase persisting into the late portovenous phase (Fig. 9, *B–D*). In spite of the similarity to an islet-cell tumor, this was interpreted as representing superimposition of the stained left adrenal on the tail of the pancreas, because it was not seen on the frames exposed before the catheter was dislodged (Fig. 9*A*). A second round shadow more to the left, persisting late into the portovenous phase (Fig. 9, *B–D*) also simulated an islet-cell tumor. However, comparison of various stages within the venous phase revealed that this represented part of the splenic vein seen on end. The diagnosis of islet-cell tumor was ruled out angiographically. Further clinical testing

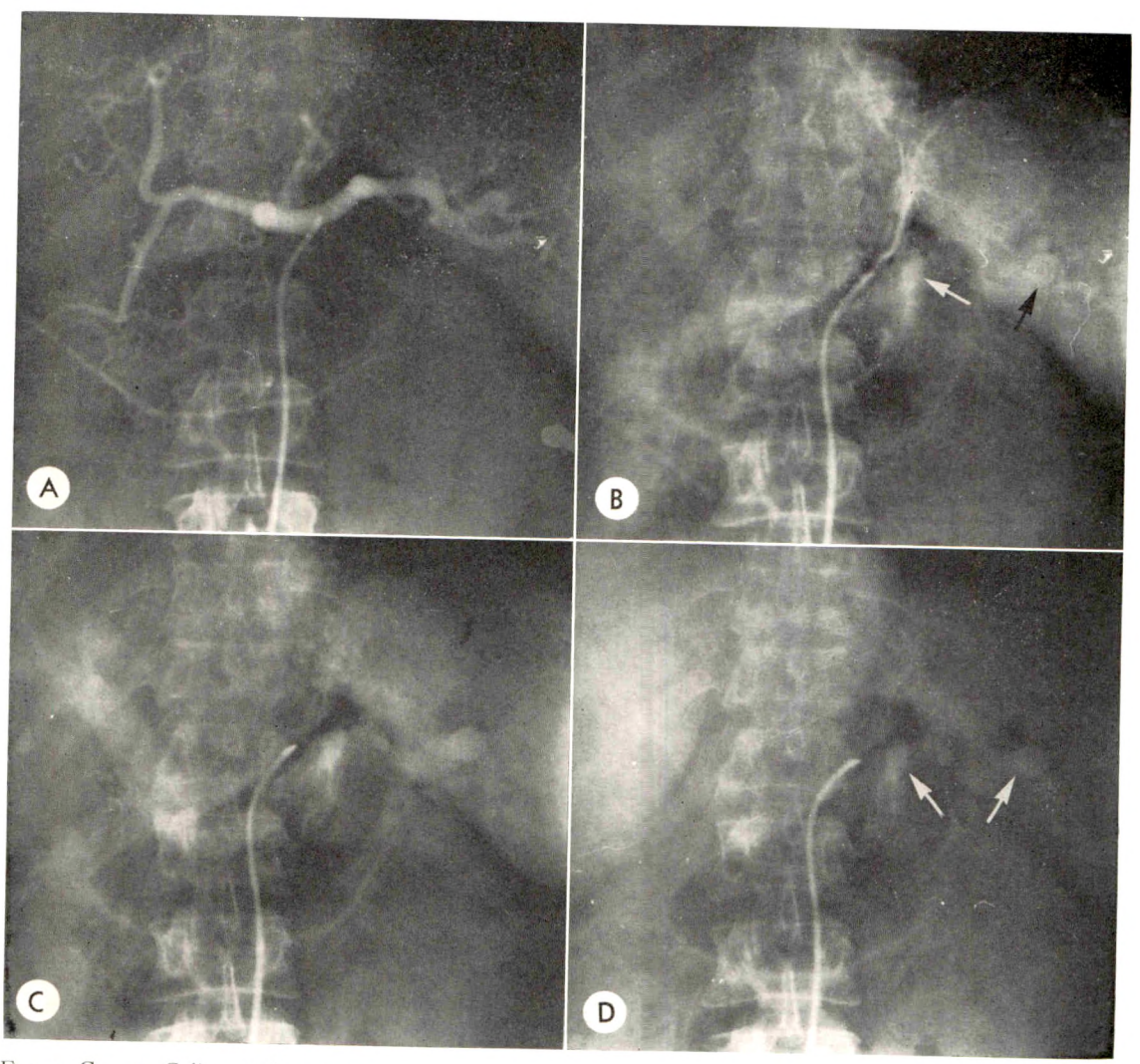


Fig. 9. Case 7. Celiac angiograms.

(*A*) Early arterial phase. (*B*) Late arterial phase showing the dislodged tip of the catheter in the phrenic artery. The adrenal gland is opacified (white arrow) and projected over the pancreatic area. A second round lesion (black arrow) is seen at the hilar area of the spleen. (*C*) Portovenous phase. The adrenal gland is still densely opacified. The round shadow at the hilar area of the spleen is caused by the orthograde course of the splenic vein. (*D*) Late portovenous phase. Both shadows (arrows), the adrenal gland and the splenic vein, are still opacified.

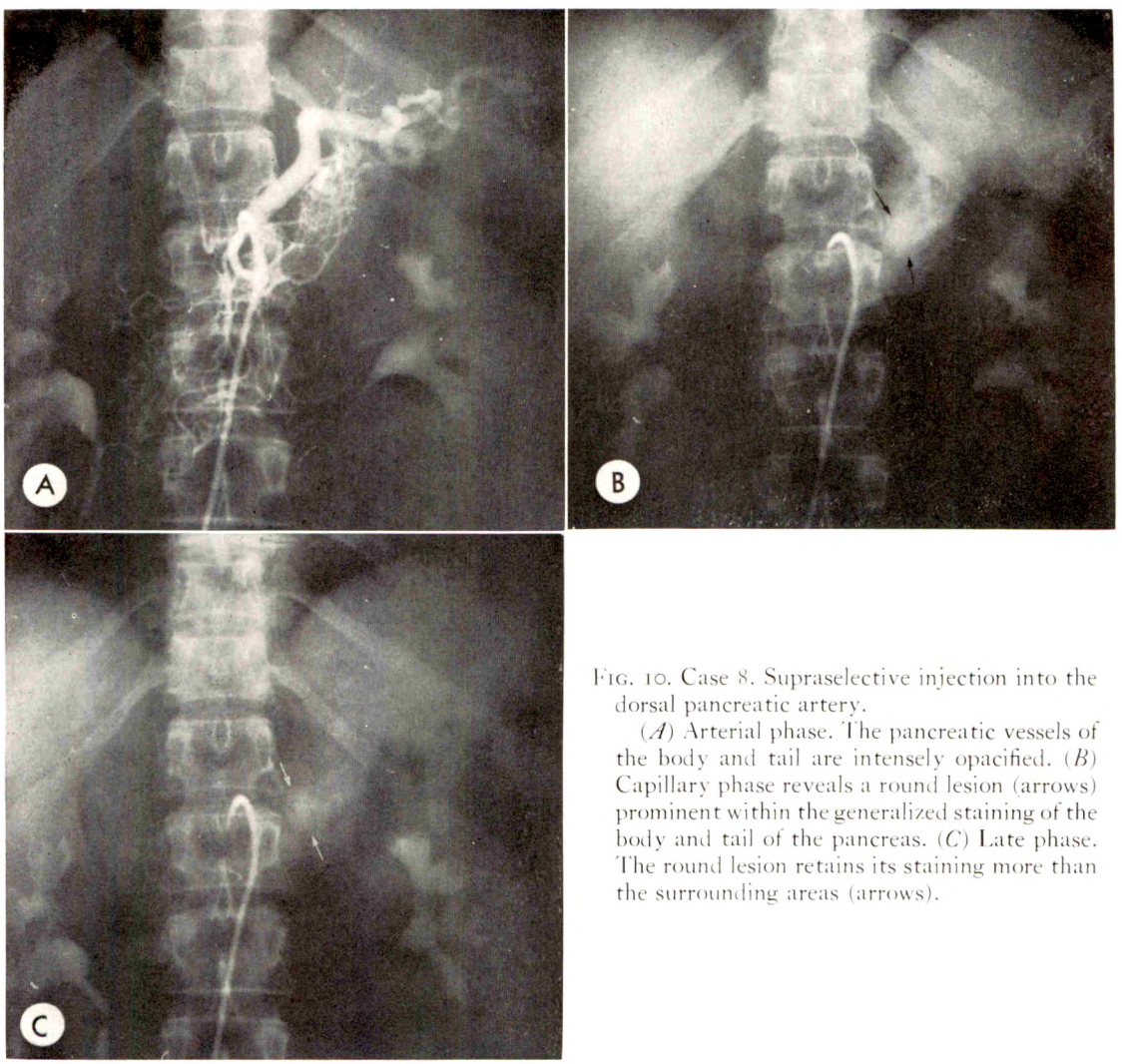


Fig. 10. Case 8. Supraselective injection into the dorsal pancreatic artery.

(*A*) Arterial phase. The pancreatic vessels of the body and tail are intensely opacified. (*B*) Capillary phase reveals a round lesion (arrows) prominent within the generalized staining of the body and tail of the pancreas. (*C*) Late phase. The round lesion retains its staining more than the surrounding areas (arrows).

and the patient's subsequent clinical course support this negative finding.

A fourth patient (Case 8) was studied because of severe hypoglycemia with epileptic attacks. Celiac and mesenteric angiography revealed normal vascularization of the whole pancreas. During the supraselective injection into the dorsal pancreatic artery (Fig. 10*A*) in the capillary phase, a dense round lesion became prominent within the generalized area of staining of the tail of the pancreas (Fig. 10*B*), persisting beyond the capillary phase (Fig. 10*C*). This was suggestive of islet-cell tumor; however, a positive diagnosis was not made, because the lesion was not well defined and was not seen on the selective celiac and mesenteric injections. Further laboratory tests and the subsequent 4 year clinical course also ruled out this diagnosis.

GROUP D

Five patients (Cases 9–13) were studied because of hypoglycemia. All were angiographically negative. In 1 patient (Case 9) accidental administration of diabinese was eventually discovered as the cause of severe hypoglycemic coma. Three patients (Cases 10–12) were further studied after negative angiography and the diagnosis of islet-cell tumor was ruled out on laboratory and clinical grounds. The fifth patient

(Case 13) showed prominent staining of the tail of the pancreas during the capillary phase, and draining veins of the tail of the pancreas were clearly seen in the venous phase (Fig. 11). In spite of the appearance of draining veins, a tumor was not diagnosed angiographically. Because of strong clinical suspicion the patient underwent surgery, but no tumor was found.

DISCUSSION

The material in the present series differs from other reported series in that it includes the total experience with angiography for islet-cell tumor. We believe that the inclusion of negative and false positive results as well as cases angiographically simulating islet-cell tumor gives a more representative idea of the diagnostic value of angiography in this condition.

Hypoglycemia was the indication for angiography in 13 patients (Cases 1–13). Only 4 of these (Cases 1–4) had proven benign beta-cell adenomas. In 9 patients (Cases 5–13) the diagnosis of insulinoma was eventually ruled out. The relatively large number of negative cases studied reflects the fact that in the first 3 years insulin assay was not available to us. It is worth noting that out of 13 cases studied only 1 false positive was encountered (Case 5).

Of 4 proven cases of insulinoma, 3 were positively diagnosed and 1 was suspected on angiography. Eleven out of 13 hypoglycemic patients were accurately assessed by angiography. Other authors reporting larger series have also noted the reliability of angiography in the diagnosis of beta-cell tumors.[1,2,5,7]

Our experience with patients studied for suspected non-beta-cell tumor is smaller. Of 3 patients studied one was angiographically false negative. This was a cystic 4 mm. benign non-beta-cell adenoma in the head of the pancreas. Two patients were false positive.

Four patients in this series were studied without suspicion of endocrine tumor: 2 had large tumors in the upper abdomen and 2 had hepatomegaly. In 3 of them a correct angiographic diagnosis was made.

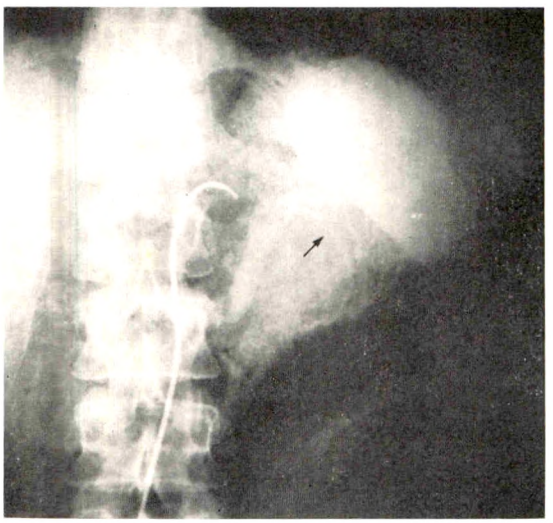


Fig. 1. Case 13. Normal celiac angiogram.
Late capillary and early portovenous phase showing venous drainage from the densely opacified tail of the pancreas (arrow).

The accepted angiographic criteria for diagnosis of benign beta-cell tumors include an early fine reticular network in the arterial phase, developing into a dense round well defined lesion in the capillary phase and persisting into the late portovenous phase. Boijsen and Samuelsson[2] have described draining veins from the beta-cell tumor and displacement of pancreatic vessels in most of their cases. In our experience persistence of the staining into the portovenous phase was seen in all cases but to a varying extent: in 2 patients the staining disappeared at the end of the portovenous phase, while in the third it persisted beyond the end of the portovenous phase. Van de Weyer *et al.*[18] have found some cases of beta-cell tumors without persistence of the staining into the late portovenous phase; however, the persistence of the staining, especially beyond the portovenous phase, favors the diagnosis.

This persistence of the staining into the portovenous phase, however, is not pathognomonic: in 3 false positive cases, 1 of metastatic carcinoma (Fig. 8) and 2 of duodenal ulcers penetrating into the pan-

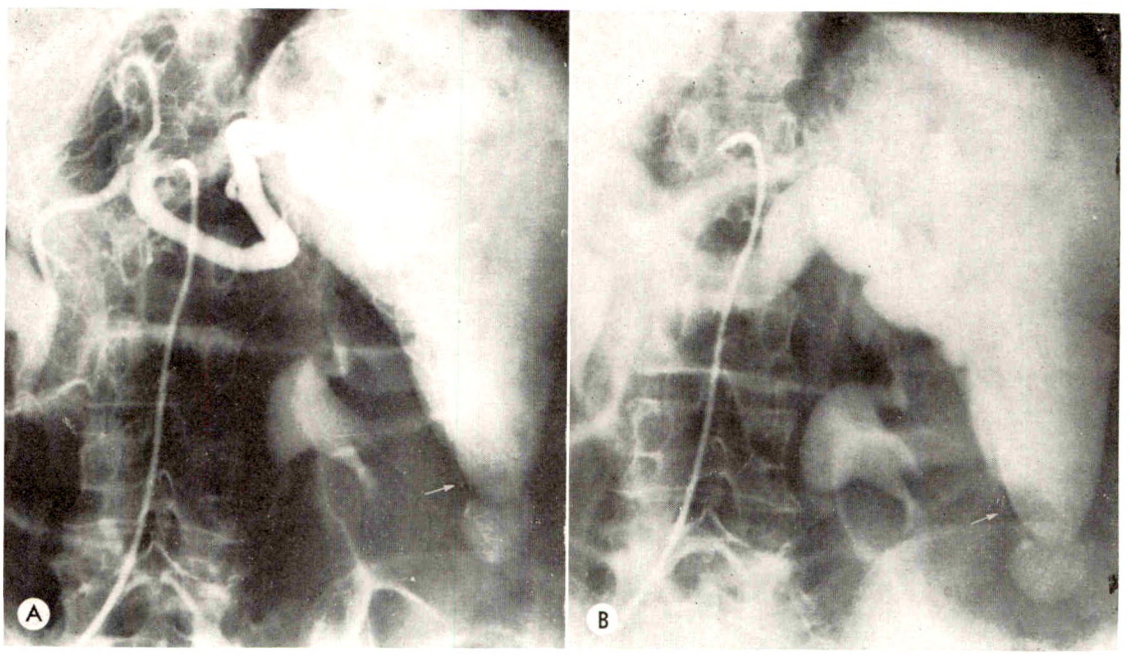


Fig. 12. Celiac angiograms (from a case not included in the present report). Aberrant spleen. (*A*) The arterial supply (arrow) and (*B*) the draining vein (arrow) are visualized.

creas (Fig. 5), the staining persisted into the late portovenous phase.

Draining veins, as described by Boijsen and Samuelsson[2] were not seen in our positive cases. On the other hand 1 of our angiographically negative cases demonstrated large draining veins in the area of the tail of the pancreas (Fig. 11). Draining veins may also occasionally be seen in aberrant spleens (Fig. 12), therefore their diagnostic value is doubtful. Displacement of vessels was also not seen in our benign functional beta-cell tumors.

From the study of this material, several points have emerged regarding differential diagnosis. Other lesions within the pancreas may give rise to angiographic appearances simulating islet-cell tumors.

Two patients with pancreatic hemangioma were seen, 1 multiple (Case 6) and 1 solitary (Case 19). Only 1 case of pancreatic hemangioma has been alluded to in the literature.[6] The arterial and capillary phases were very similar to islet-cell adenomas (Fig. 6; and 7); however, the staining disappeared before the portovenous phase in both cases. In 1 patient (Fig. 7) the angiographic demonstration of hemangiomata in the liver gave the clue to the correct preoperative diagnosis. In the other patient (Case 19) the correct diagnosis was made only at surgery when multiple hemangiomata were seen in the liver. The angiographic association of similar vascular lesions in the pancreas and the liver is therefore consistent with either metastatic islet-cell tumor or hemangiomata in both organs.

One unusual case of pancreatic metastasis from adenocarcinoma of the stomach was seen (Case 20). The angiographic appearance was extremely suggestive of islet-cell tumor, except that the borders of the lesion seemed more clearly demarcated in the capillary phase (Fig. 8). The presence of a similar lesion in the liver, in this case with a known primary, suggested the diagnosis of metastatic tumor (Fig. 8*B*). Supraselective pancreatic injection has been advised as a means of improving visualization of small pancreatic tumors.[14] However, as pointed out by Boijsen and

Samuelsson and demonstrated in our case (Fig. 10), the injection of a relatively large amount of opaque material into a pancreatic artery, may give rise to the accumulation of opaque medium in a limited area of the pancreas, causing misinterpretation.

Differential diagnostic errors may also arise from extrapancreatic lesions or structures, which overlie the area of the pancreas. Duodenal ulcers penetrating into the head of the pancreas may be mistaken for islet-cell adenomas; however, the presence of circumferential feeding arteries seems to be the clue to the correct diagnosis. Recently, we have seen such circumferential arteries in other cases of penetrating duodenal ulcer. It appears that both the round staining and the circumferential vessels are secondary to a severe inflammatory reaction. This appearance should be distinguished from the reticular pattern of islet-cell tumor.

The incidental injection of a relatively large amount of contrast material into the phrenic artery during celiac angiography may densely opacify the left adrenal gland. If this shadow is superimposed on the area of the pancreas, it may simulate an islet-cell tumor as was in our case (Fig. 9).

A small aberrant accessory spleen may simulate an islet-cell tumor in the tail of the pancreas. In our case the staining in the capillary phase was more clearly demarcated and did not persist into the portovenous phase. In other cases the staining may persist for a longer time[2] and hilar vessels (artery and vein) of the aberrant spleen may be clearly seen (Fig. 12).

Due to the tortuous course of the splenic vein one of its segments seen on end persisting into the late venous phase may give rise to a small dense round shadow superimposed on the tail of the pancreas (Fig. 9).

Two large nonfunctioning islet-cell tumors are included in this series (Cases 17 and 18). The first was an unusually large benign nonfunctioning alpha-islet-cell tumor. To the best of our knowledge this is the second case of alpha-cell tumor and the first nonfunctioning benign type visualized by angiography.[9] The rich network of large pathologic vessels with early venous drainage and a nonhomogeneous capillary phase (Fig. 3) is similar to the angiographic appearance of malignant islet-cell tumors as described by others.[2,6,10,11,13,16,17] These tumors are difficult to differentiate from pancreatic cystadenomas or cystadenocarcinomas which are richly vascularized with nonhomogeneous staining in the capillary phase.[4,15,16,17] The only point in the present case that could have helped in the differential diagnosis was the presence of calcifications usually absent in cystadenoma.

In the second patient with a large nonfunctioning malignant islet-cell tumor, early venous filling was even more prominent and the capillary phase staining was more homogeneous. It appears that the rich network of large pathologic vessels, the early venous filling and the more or less homogeneous capillary staining are not necessarily features of malignancy, but are related to the type and the size of the tumor. Therefore, in very large tumors a firm angiographic diagnosis of malignancy is only possible in metastatic lesions are demonstrated.

SUMMARY

This report deals with 20 patients studied angiographically with the clinical suspicion of islet-cell tumor whether or not such a tumor was eventually confirmed. Cases were also included in which angiography was performed for other reasons and arteriographic features simulating islet-cell tumor were noted.

The angiographic features of functioning and nonfunctioning benign and malignant islet-cell tumors are described. Points of differential diagnosis of pancreatic and extrapancreatic lesions and structures are discussed.

The inclusion of surgically verified cases, examples of false positive and false negative diagnosis as well as lesions simulating islet-cell tumors and other negative cases gives a more reliable picture of the values of

angiography in the diagnosis of islet-cell tumor.

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THE MANY FACES OF ISLET CELL TUMORS*

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FUNCTIONAL islet cell tumors have the capability of producing numerous hormones both indigenous to the normal pancreas, such as gastrin, secretin, insulin, and glucagon; and ectopic, specifically melanocyte stimulating hormone (MSH), adrenocorticotropic hormone (ACTH), and serotonin.[7,8,11,13,16,18,20,21,24] Each of these hormones is associated with a distinct clinical syndrome. While some syndromes such as the Zollinger-Ellison syndrome or the one associated with hypoglycemia are relatively common and well known, others are rare and therefore unfamiliar to many.

During the past 3 years we have had an opportunity to examine several patients with the syndromes related to overproduction of the indigenous hormones of the pancreas. These syndromes are the Watery Diarrhea Hypokalemia Achlorhydria (WDHA), Zollinger-Ellison (Z.E.), and those associated with the overproduction of insulin and glucagon. It is the purpose of this paper to emphasize the clinical manifestations of these syndromes.

REPORT OF CASES

CASE 1. A 44 year old rancher presented with a history of intermittent diarrhea of 1 year's duration and a 62 pound weight loss. Hospital admission was prompted by delirium and irrational behavior which began 1 month earlier. On admission, the patient was found to be markedly hypokalemic. Barium studies and other routine roentgenographic examinations done because of the severe diarrhea were negative. Gastric analysis showed hypochlorhydria. The patient's diarrhea was continuous and profuse, unrelieved by medication. Despite vigorous attempts serum potassium levels could not be adequately maintained. Because of the patient's rapidly deteriorating condition, upper gastrointestinal and barium enema examinations were repeated but again no abnormality was demonstrated. Abdominal angiography done to rule out vascular anomalies or neoplasm revealed a vascular tumor in the region of the tail of the pancreas (Fig. 1*A*). There was a prolonged blush and large draining veins could be seen (Fig. 1*B*). Multiple hypervascular nodules were demonstrated in the liver (Fig. 2). These findings were confirmed at the time of surgery. The histologic diagnosis of the tumor was malignant nonbeta cell pancreatic islet cell tumor with liver metastases. Following surgery, diarrhea and severe potassium loss continued and the patient died 2 weeks thereafter.

Comment. The Watery Diarrhea Hypokalemia Achlorhydria syndrome, initially labeled "pancreatic cholera,"[19] is caused by a nonbeta islet cell tumor. The symptoms consist of profuse watery diarrhea, hypokalemia, and gastric *hypo*secretion. The etiologic agent is a secretin-like substance.[24,30] The type of tumor cell within the islet has not yet been classified; in some tumors an increased number of alpha cells has been noted.[1,13] Kraft *et al.*,[13] in their series of 20 patients, reported that stool volumes averaged 5.8 liters and potassium loss assessed in 14 patients averaged 350 mEq. per day (average normal loss is 13 mEq. per day).

Since most of the symptoms are referable to the gastrointestinal tract in general, and to diarrhea in particular, barium studies are invariably performed, but are nearly always normal. Most of the tumors are too small to be detected by the barium examination. Therapy is usually delayed because the symptoms are rarely associated with the correct diagnosis, and only knowledge of the syndrome may alert the physician that the etiology is an islet cell tumor. In contrast to the Zollinger-Ellison syndrome which is also caused by an islet cell tumor and may have diarrhea as a prominent symptom, the WDHA patients with few exceptions are achlorhydric.[4] Gastric

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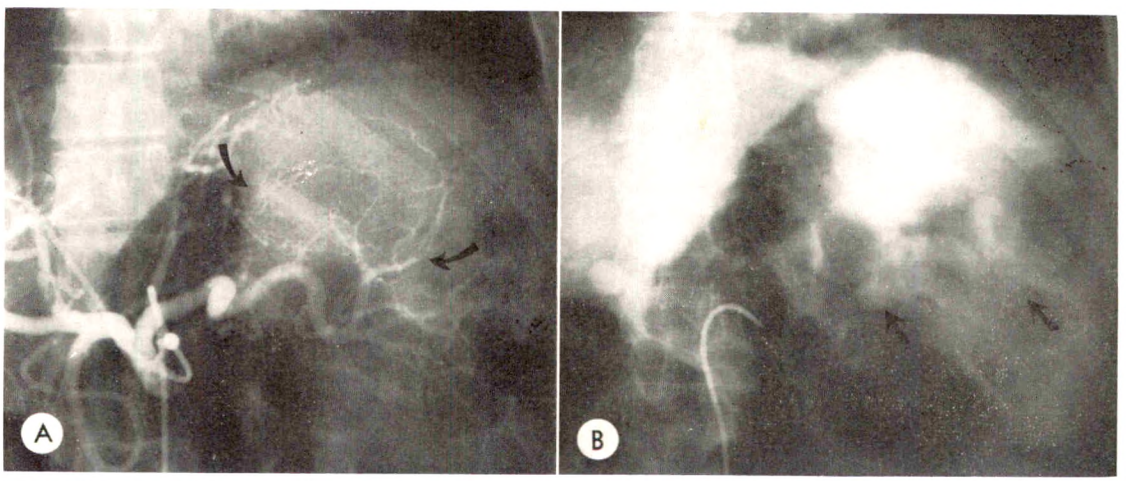


FIG. 1. Case I. (*A*) Arterial phase of angiography. Note the displacement of vessels in an area adjacent to the gastric fundus (arrows). There are also a few tumor vessels present. The main blood supply is from the splenic artery. (*B*) Late venous phase. The densely opacified tumor is well seen. Note the huge veins (arrows) draining the tumor.

and duodenal ulcers are not seen. In many patients the serum calcium is elevated, a finding which is directly related to the severity of the diarrhea. Patients have undergone neck exploration because of the hypercalcemia, but in none was there evidence of parathyroid adenoma. With the removal of the pancreatic tumor, the serum calcium levels return to normal. Glucose metabolism may also be altered, perhaps due to the potassium deficiency. Approximately one-half of the tumors reported have been malignant. Most of these have had metastases at the time of surgery. The tumors have been located principally in the body and tail of the pancreas and have ranged in size from 1 to 7 cm. The treatment is surgical and the prognosis depends upon early diagnosis. When no metastases are present, the lesions are potentially curable. However, the average length of survival of patients with metastases has been only 1 year.

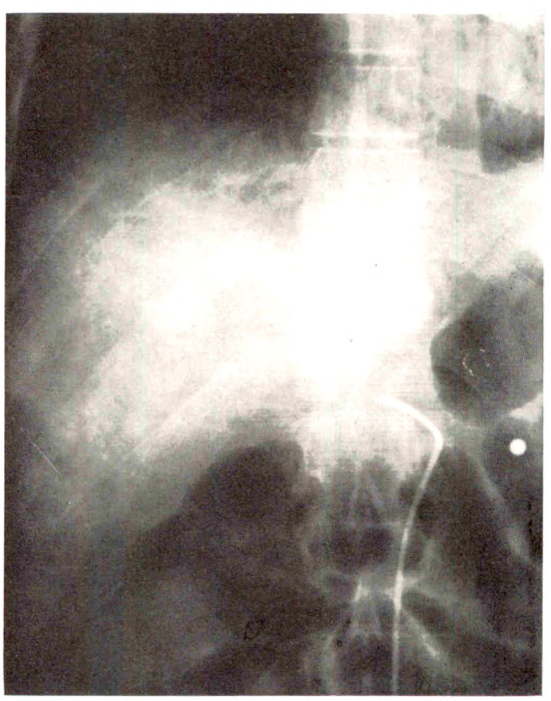


FIG. 2. Case I. Capillary phase during selective hepatic artery injection. Note the mottled appearance of the liver due to the multiple metastases. The somewhat distended gallbladder is well seen (arrow).

CASE II. This 43 year old male had several hospital admissions because of severe upper abdominal pain, intermittent diarrhea, and history of ulcer disease. Because his pain became progressively worse he was readmitted. During the past 6 months the patient lost 25 pounds of weight. Gastric secretion studies showed basic acid secretion values of 42 mEq./hr. which, following stimulation with betazole hydrochloride, rose to 65 mEq./hr. Serum gastrin levels were elevated to 650 to 900 μg. per milliliter. Roentgenograms showed

gastric hypersecretion, probable ulcer in the second part of the duodenum (Fig. 3), and a small bowel pattern suggestive of a malabsorption syndrome (Fig. 4). Because of a question of sprue, the patient was treated with a gluten free diet for several weeks with no significant clinical improvement. The patient underwent an abdominal exploration at which time a large nodule in the junction betwen the head and the body of the pancreas was found. The nodule was excised, and the diagnosis of nonbeta cell tumor of the pancreas was made. A subtotal gastric resection was performed. The patient has done well following surgery.

Comment. The Zollinger-Ellison syndrome results from a nonbeta cell tumor of the pancreas. The tumor cell type within the islet is not definitely known. The delta cell has been implicated by some authors.[15] A gastrin-like material has been isolated from the tumor and is considered the etiologic agent.

Gastric hypersecretion ulcerations, hyperacidity, and enteritis are the hallmark of this syndrome. However, according to Ellison and Wilson,[8] in approximately 5 per cent of the cases, achlorhydria or hypochlorhydria have been found. This may be related to the severe diarrhea which was present in 40 per cent of patients. Diarrhea was the sole presenting symptom in 7 per cent of the cases. Approximately 60 per cent of the tumors were malignant, and the majority had metastases at the time of surgery. The presence of generalized microadenomatoses, multiple tumors, and a high incidence of malignancy with liver metastases, serves to limit the importance of tumor resection. Zollinger and Ellison advocate as the treatment of choice total gastrectomy and, where feasible, tumor resection. In many instances death has been from multiple operations and other complications of the ulcer diatheses, frequently related to the delayed diagnosis. Ellison and Wilson[8] state that approximately 80 per cent of the patients have been symptomatic for 1 year or longer prior to establishment of the correct diagnosis. In 25 per cent of patients with the Z.E. syndrome other endocrine adenomas, particularly parathyroid and pituitary, occur simultaneously.

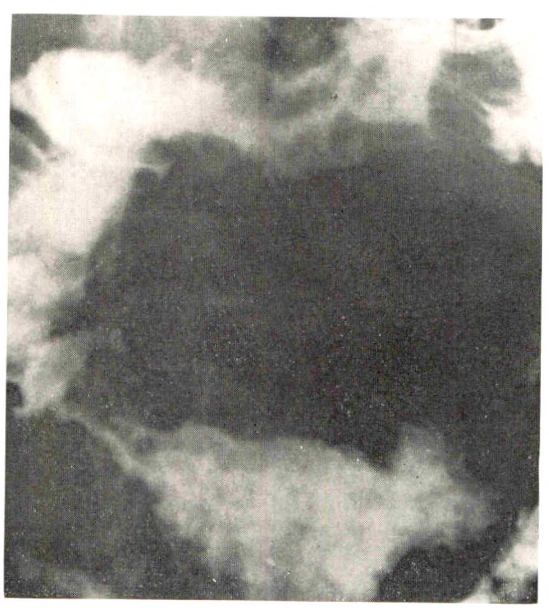


Fig. 3. Case II. Spot roentgenogram taken during upper gastrointestinal examination showing an ulcer (arrow) in the second part of the duodenum. The duodenum distal to the ulcer is distended and filled with secretions.

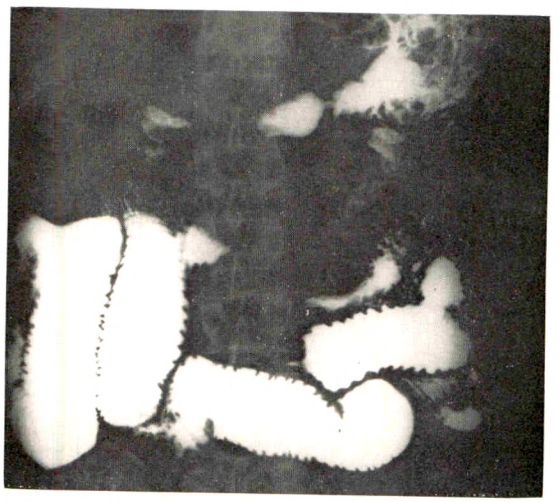


Fig. 4. Case II. Roentgenogram made during small bowel series shows dilatation and segmentation of the barium in the jejunum and proximal ileum. There was significant irritability and hypermotility of the distended loops.

Case III. A 58 year old man was admitted to the University of Utah Medical Center because

of sudden personality changes characterized by aggressive and disoriented behavior. Significant findings were 24 hour fasting blood surgars below 40 mg. per cent. Angiography demonstrated a well circumscribed area of vascular blush with neovascularity and enlarged draining veins (Fig. 5, *A* and *B*). At surgery islet cell adenoma was found. The patient's symptoms disappeared following surgery.

Comment. Probably the best known and most common of the islet cell tumors is the beta cell type, the so-called insulinoma. "Whipple's triad," first enunciated in 1938, remains the basis for the diagnosis in most cases: (1) fasting blood sugar below 40 mg. per cent; (2) symptoms which can be provoked by fasting; and (3) relief of symptoms and hypoglycemia by glucose.[18]

In nearly all patients the symptoms can be provoked by 24 to 48 hours prolonged fasting. The symptoms mimic other more common clinical entities, such as Central Nervous System organic lesions and other behavioral disorders. Personality changes, particularly aggressive and antisocial behavior, seizures, and positive neurologic deficits are common presenting symptoms. The pathophysiology is the inappropriate secretion of insulin or an insulin-like substance; and according to Levin,[15] the most common causes for spontaneous hypoglycemic episodes after the age of 4 are functional disorders or beta cell islet tumors of the pancreas. Ninety per cent of the tumors are benign. In 10 per cent multiple tumors are present. The distribution of the tumor is equal throughout the pancreas. A small number (2 per cent) of beta cell islet tumors arise ectopically, most often in the duodenal wall. An association of these neoplasms with adenomas elsewhere, particularly in the parathyroid and pituitary, is common—a relationship which appears higher in patients with diffuse adenomatous islet involvement, multiple tumors, and the occurrence of the disease in the first and second decades of life.

CASE IV. A 42 year old woman who presented with mild diabetes, an enlarged liver, and bullous eczematoid dermatitis of the extremities. Liver scan with I^{131} rose bengal demonstrated 2 "cold" lesions in the right lobe of the liver. Abdominal angiography showed a tumor blush in the tail of the pancreas and splaying of intrahepatic vessels in areas corresponding to the 2 lesions seen on the scan (Fig. 6, *A* and *B*). Exploratory laparotomy and biopsy of the pancreatic and liver lesions showed malignant islet cell adenoma—alpha cell type. A local resection of the pancreatic lesion was undertaken. Despite surgery, there had been slow but definite progression in the severity of the diabetes.

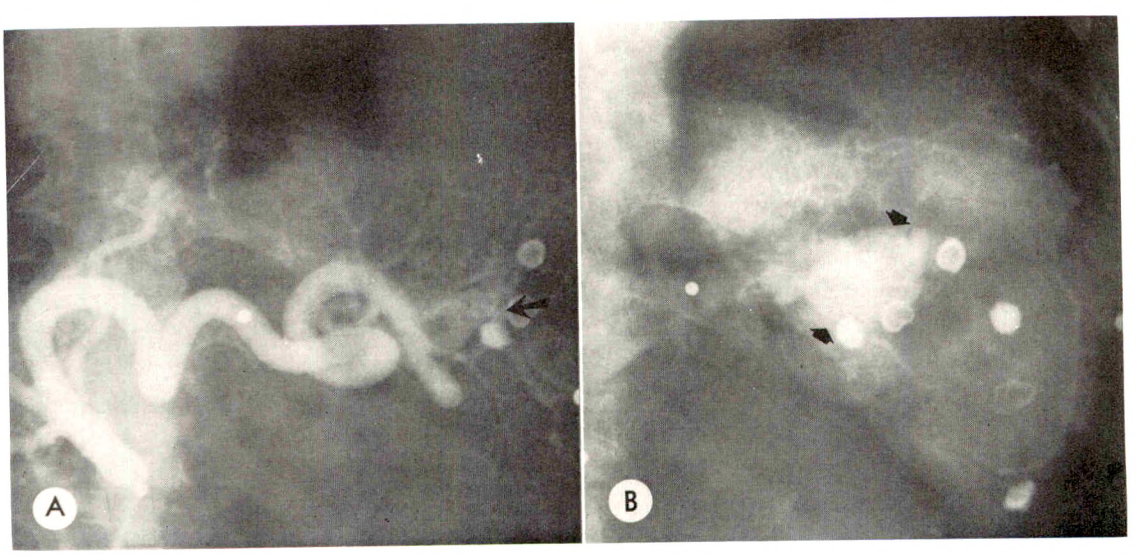


FIG. 5. Case III. (*A*) Arterial phase of splenic artery injection shows early tumor vessels near the hilum of the spleen (arrows). (*B*) Note the dense blush of the tumor on the venous phase (arrows).

Comment. The alpha cell type tumor is the least common of the islet cell tumors. The etyiologic agent is glucagon, which when hypersecreted produces a state of hyperglycemia. The first case was reported in 1966.[21] Since then a few isolated case reports have appeared in the literature.[15] Chemical and ultrastructure studies indicate the alpha cell nature of these tumors. Abnormal carbohydrate metabolism and chronic skin lesions, both nonspecific signs, have been common denominators in the few reported cases. Lack of response to intravenous glucagon which normally results in a rapid rise in serum insulin and glucose can be used as a screening test.

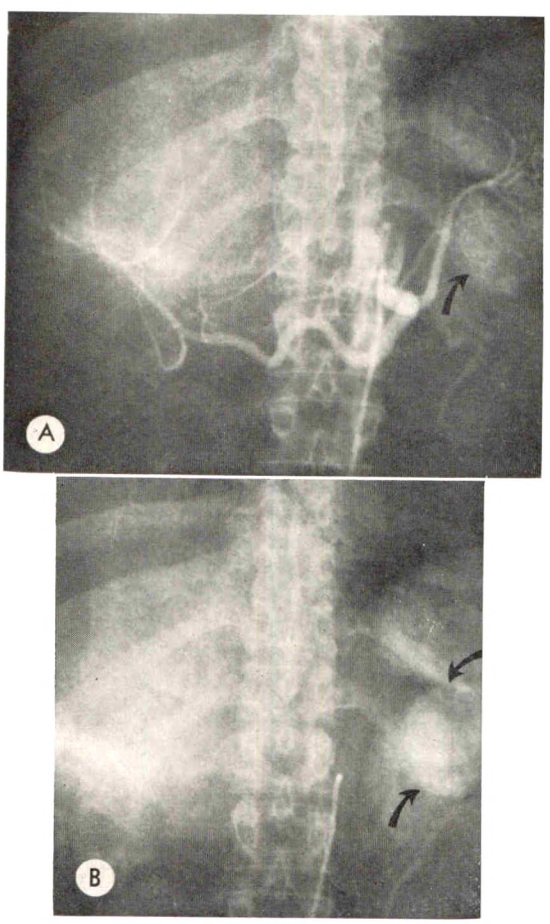


Fig. 6. Case IV. Celiac arteriograms. (*A*) Arterial phase. Note the tumor in the region of the splenic hilum (arrow). The marked displacement and splaying of vessels caused by large hepatic metastases is also clearly noted. (*B*) Venous phase. The primary tumor is distinctly seen around the hilum of the spleen (arrows). Avascular metastases occupy most of the upper two-thirds of the liver. Smaller avascular lesions are present in the lower part of the right lobe of the liver.

DISCUSSION

The 4 cases which have been presented are but a few examples of the manifold clinical syndromes associated with islet cell tumors. These tumors may be functional or nonfunctional. The functional variety has the potential to produce any one or more of several hormones, each most likely related to a specific cell type within the islet. Alpha and beta cells have been definitely linked to glucagon[21] and insulin[15,18] production, respectively. The cell type of the other hormones are not yet identified with certainty. The hormones produced may be indigenous to the pancreas or ectopic. The cases presented in this paper include the syndromes related to the indigenous hormones (secretin, gastrin, insulin, and glucagon). At the light microscope level, regardless of hormone production, the tumors themselves closely resemble each other.[6,15] They also are often histologically indistinguishable from carcinoid of the gastrointestinal tract or bronchi, which are serotonin producers.[7,11] The hormone production potential is shared with several tumors outside the pancreas: oat cell carcinoma; medullary thyroid carcinoma; thymoma; neuroblastoma; and ovarian teratoma—all tumors that show a close morphologic similarity to the islet cell variety. Several authors have postulated that this entire spectrum of tumors arises from a common cell with multiple hormone producing potential. Weichert *et al.*[27] feel that this totipotential cell may be neurectodermal in origin, perhaps the argentaffin cell or a precursor.

The radiologist plays a pivotal role in the evaluation of the 2 primary gastrointestinal syndromes, Z.E. and WDHA. The WDHA patients present with diarrhea, often continuous, and the Z.E. patients with gastric hypersecretion and multiple ulcers. While the diagnosis of the Z.E. syndrome can be made on upper gastrointestinal examination if atypical ulcers, gas-

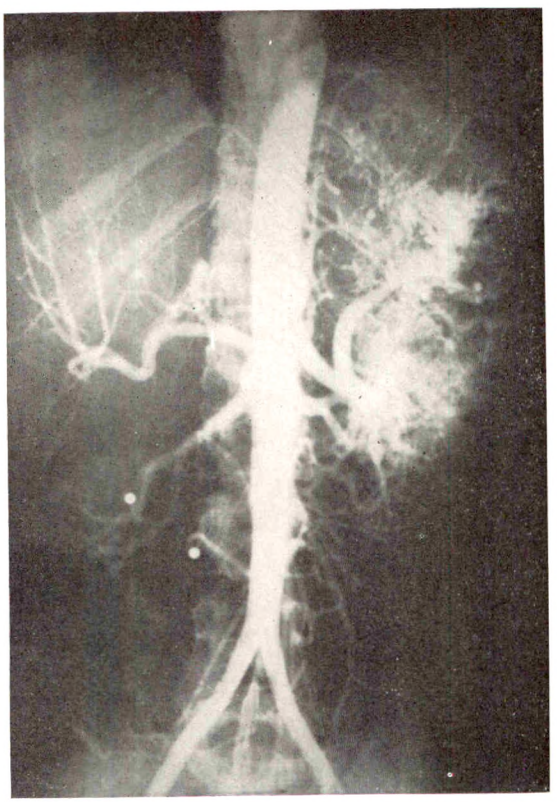


FIG. 7. Angiogram of a highly vascular large nonsecreting islet cell tumor.

tric hypersecretion, and a pattern of enteritis in the proximal small bowel are present,[5] the diagnosis of WDHA syndrome is usually established only after angiography is done.

The glucose related tumors, insulinomas and glucagonomas, are usually diagnosed before the patients are seen in the X-ray Department. Here the role of the radiologist is generally to confirm the clinical impression and possibly to localize the lesion. Localization is important since many of the tumors are small and cannot be palpated during surgery. In addition, angiography can serve to document the presence or absence of metastases. The angiographic findings of these tumors have been well described.[1,4,6,12,23] Vascular lesions have been demonstrated in a high percentage of cases. Insulinomas, the most reported of these tumors, have been identified up to 75 per cent of the time preoperatively.[9,17,20] Angiographically as well as histologically, the different tumors cannot be distinguished one from another.[17] When demonstrated, all appear as well circumscribed vascular tumors with a late capillary blush.

The clinical symptoms of the nonfunctioning islet cell tumors usually relate to the size of the tumor and to the presence of metastasis. The symptoms are nonspecific: pain; weight loss; occasionally gastrointestinal bleeding; etc. Large tumors can be diagnosed on barium studies, small ones only by angiography. The tumors, especially the large ones, are very vascular and easily recognized on the angiogram (Fig. 7).

When tumors producing aberrant hormones are present, the clinical symptoms will relate to the produced hormones. Thus, corticotropin secreting islet cell tumors will produce a Cushing's like syndrome. A strikingly high number of the patients with these aberrant tumors show hyperpigmentation of the skin and an increase in plasma level of melanocyte stimulating hormone (MSH). However, like other ectopic sites of corticotropin producing tumors, the clinical features may be quite subtle, and only a high degree of suspicion will result in a correct diagnosis.[15]

Carcinoid syndrome, clinically characterized by flushing, diarrhea, and elevated blood levels of serotonin and 5-hydroxyindolacetic acid in the urine, may also be produced by islet cell tumors.

Despite the great differences in the clinical manifestations of these tumors, angiography remains the method of choice for roentgenographic demonstration of the lesions. It is hoped that this review will serve to better the understanding of the "many faces" of the islet cell tumors.

SUMMARY

The clinical symptoms of the variety of syndromes associated with islet cell tumors are described. Islet cell tumors have the potential to excrete a variety of endogenous and exogenous hormones and consequently a variety of clinical syndromes result.

Case reports of patients with Zollinger-Ellison, WDHA, insulinoma, and glucagon secreting tumors are presented. The Z.E. and WDHA syndromes are primarily gastrointestinal syndromes and the radiologist plays a pivotal role in establishing the diagnosis of these patients. In the glucose relating tumors, the insulinomas and glucagonomas, the main role of the radiologist is in confirming clinical impression and localizing the lesion.

Early diagnosis is important because all these tumors are potentially malignant and cure can only be achieved if they are detected prior to the dissemination of metastases.

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VOL. 119, NO. 1

SELECTIVE ARTERIOGRAPHY IN THE DIAGNOSIS OF CHRONIC INFLAMMATORY PANCREATIC DISEASE*

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DESPITE its frequency, chronic pancreatitis has always been a most difficult disease to diagnose correctly. The symptoms produced may be nonspecific, the laboratory findings are generally inconclusive and the clinical signs are vague.

We have attempted to diagnose chronic pancreatitis by angiographic methods. We believe angiography to be the best means available because of its accuracy.

MATERIAL

Selective superior mesenteric and celiac arteriography was performed in 84 patients suspected of having pancreatic disease. Three of these patients had clinical and laboratory findings that were compatible with the initial onset of acute pancreatitis. Because there were no definite diagnostic arteriographic findings, these cases were eliminated from our study. Of the remaining 81 patients, pancreatic carcinoma was arteriographically indicated and surgically confirmed in another 3 cases. These were also eliminated from this series.

The final 78 cases were studied because:

a. chronic pancreatitis was clinically suggested,
b. conventional roentgenographic studies indicated the presence of pseudocyst or pancreatic calcification,
c. clinical and laboratory findings ruled out other diseases and chronic pancreatitis was thus considered by process of elimination, and
d. chronic pancreatitis was a differential diagnosis.

Of the 78 cases studied, 44 underwent surgery on the basis of the angiographic evidence observed (Table 1). Of these 44 patients, our positive results were confirmed both surgically and pathologically, either by subtotal pancreatectomy or by pancreatic biopsy in 56 per cent of them. In 9 more cases (20 per cent) our positive findings were supported by surgical inspection only. Surgery revealed only 1 false-positive (2 per cent) and 1 false-negative (2 per cent) cases. Angiographically equivocal findings were obtained in a total of 7 cases (15 per cent).

Thirty-five of our patients were not subjected to surgery, although chronic pancreatitis was angiographically indicated in 33 of them. Of these, 24 cases (68 per cent) showed clinical, laboratory and roentgenographic evidence of chronic pancreatitis either before angiography was performed or on re-evaluation of the clinical data after angiography gave positive indication of chronic pancreatitis. In this same group, on the basis of clinical evidence only, angiography was falsely positive in 9 cases (22 per cent), falsely negative in one (2 per cent) and equivocal in one (2 per cent).

To summarize, angiographic findings positive for chronic pancreatitis were confirmed by surgical inspection, pathologic examination or clinical, laboratory and conventional roentgenologic studies in a total of 59 out of 78 cases or 75 per cent. Angiography gave false-positive results in 10 cases (13 per cent), false-negative re-

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TABLE I

SUMMARY OF ANGIOGRAPHIC FINDINGS

Diagnostic Methods	Angiographic Findings		
	Positive	Equivocal	Negative
Tissue diagnosis of chronic pancreatitis, pseudocyst or abscess	26	0	0
Surgical inspection suggestive of chronic pancreatitis; no tissue taken	9	3	1
Surgical inspection not suggestive of chronic pancreatitis	1	4	0
Clinical, laboratory and roentgenographic findings suggestive of chronic pancreatitis before angiography	12	1	1
Re-evaluation of clinical findings after positive angiogram compatible with chronic pancreatitis	12	0	0
Re-evaluation of clinical findings after positive angiogram could not confirm chronic pancreatitis	8	0	0

Table summary: 45 per cent of cases angiographically positive confirmed surgically and/or pathologically; 30 per cent positive angiographically supported by clinical evidence; 25 per cent false positive, negative and equivocal.

sults in 2 cases (2 per cent), and equivocal results in 8 instances (10 per cent).

The presenting complaint in all patients subjected to arteriography consisted of either progressive, excruciating abdominal pain or several recurrent episodes of acute abdominal distress. Other, more common manifestations noted in these patients were marked weight loss, anorexia, nausea, intermittent ethanol intake. Examination in most cases revealed the presence of varying degrees of upper abdominal tenderness. Occasionally, a palpable mass with poorly defined margins was noted. The rest of the physical examination was essentially noncontributary. Laboratory studies in some acutely ill patients indicated elevated amylase and lipase levels in both blood and urine and increased serum bilirubin levels in others. The majority of the patients, however, suffered vague abdominal pains and weight loss without elevation of the pancreatic enzyme titer. Other laboratory studies, including liver function tests, were mostly within normal limits, except for a small number of cases who had associated cirrhosis.

Conventional roentgenologic examinations consisting of plain roentgenography and barium contrast studies of both upper and lower gastrointestinal tracts were interpreted as normal in the majority of cases. Associated benign gastrointestinal disease, chiefly peptic ulceration, was present in less than 10 per cent of the patients. Varying degrees of pancreatic calcification were found in 5 per cent of the patients studied. Widening of the duodenal loop or extrinsic pressure on the stomach were encountered in 15 per cent of cases.

At laparotomy, gross abnormalities in the abdominal viscera were noted. Four patients had ascites with or without a collection of high amylase peritoneal effusion, especially on the left side.

Correlation of pancreatic angiographic findings with surgical evidence was attempted. Resection of the distal 90 per cent of the pancreas *en bloc* with the spleen was carried out in 15 cases. Pancreatic biopsy was performed in an additional 11 cases. In those patients not subjected to pancreatic resection, inspection and palpation of the pancreas also helped to assess the angiographic diagnosis. More commonly, plastic changes consisting of nodularity, fibrosis, cyst formation and calcification were present. Cystic changes in the pancreas were generally found within the substance of the gland and were not more than 3–5 cm. in diameter. In some instances, large collections of peripancreatic exudate were encountered. Occasionally, collections of free peritoneal fluid with high amylase titers and intraabdominal fat necrosis were present.

TECHNIQUE

A fine degree of technical skill is required for successful angiographic examination of the pancreas because the blood supply to this organ is relatively small. Success further depends upon optimal opacification of the pancreatic arteries. For this reason, routine celiac and superior mesenteric arteriographies must be performed to allow for the normal variations which occur between these major arteries in supplying the pancreas.

We use a moderately stiff, No. 7 French BD Green catheter with a single curve, occasionally a J-shape, and modified to conform to the individual's arterial pathway and size. The catheter is introduced by the Seldinger technique of percutaneous femoral puncture.

To obtain good arteriograms of the pancreas, we find it essential to adjust our contrast medium injections according to the rate of blood flow in the celiac and/or superior mesenteric arteries. This is achieved by administering a test injection estimated to the approximate rate of arterial blood flow under fluoroscopic control and by videotape record. In most cases this is about 12 cc./sec.; therefore, 6 cc. of contrast medium is injected in ½ sec. by automatic injector. If, under these conditions, contrast material regurgitates into the aorta, the arterial flow rate has been exceeded and the rate of contrast medium injection is correspondingly reduced. If, on the other hand, the contrast material appears to be diluted (by visual estimation) or is rapidly moved, the arterial flow is in excess of the injection rate and the latter is increased. Further test injections are made, as necessary, with estimated alterations in the contrast medium injection rate used. We prefer to use vascoray as contrast medium (iothalamate, meglumine 52 per cent, sodium 26 per cent). A 3.0 to 3.5 second injection of vascoray is given with total volume adjusted to the arterial flow rate. This ordinarily represents 40–50 cc. of contrast medium.

We employ a film program that adequately covers the entire splenohepatoportal system and includes 2 films per second for 3 seconds, 1 film per second for 5 seconds, and 1 film every 3 seconds for 18 seconds. This program thus provides a total of 17 films in 26 seconds.

In order to obtain the best possible visualizations, we have found it necessary to refine our filming technique. We use a short exposure time, 0.035 sec., to prevent blurring of the arteries by motion. This requires a generator capable of delivering an 800 ma. current. A small, 1 mm. focal spot and low voltage (75–85 kv.) are required. For best results, we use a Vacuum Cassette CP 420 changer with Kodak RPR film. This helps eliminate scattered radiation and provides finer resolution. The stereo method is routinely used for a three-dimensional concept which is of great assistance in the identification of individual vessels and in the often difficult determination of vessel position. This is followed by the use of the magnification technique, focused on the pancreas, which is very helpful in detecting minute vascular changes such as rat-tailing and small artery beading.

RESULTS

ANGIOGRAPHIC FINDINGS

(Table II)

1. *Hypervascularity.* The pancreatic blood supply is quite small and inconspicuous. However, by employing a fine degree of technical care including magnification, the arterial network is recognizable in nearly all cases. The arterial supply for the head of the pancreas and its uncinate process arises from the gastroduodenal artery branch of the celiac which forms the superior pancreatic arcade (Fig. 1, *A* and *B*). This anastomoses with the inferior pancreatic arcade of the superior mesenteric artery. The pancreatic head, therefore, has a bidirectional flow from the celiac and superior mesenteric arteries. In the majority of cases, the pancreatic arcades can be fully visualized by celiac injection. Occasionally, however, the superior

TABLE II

FREQUENCY OF FINDINGS

(Based on 76 cases)

Angiographic Feature	No. of Cases
1. Hypervascularity	45
2. Hypovascularity	15
3. Enlargement (partial or generalized, without large detectable cyst)	22
4. Pseudocyst and abscess	8
5. Vascular irregularity	74
6. Nonfunctioning splenic vein	11
7. Calcification	5
8. Pancreatic blush	70

mesenteric injection dominates bidirectional flow and the pancreatic arcade is more readily visible *via* this route.

Those arteries that supply the body and tail of the pancreas show a great degree of normal variability and originate from the splenic artery. Sometimes, as a normal variation, the superior mesenteric artery supplies all or part of the tail and/or body of the pancreas (Fig. 2*A*). Superselective injection of the splenic or gastroduodenal arteries or the inferior pancreatic arcade favors improved visualization of this arterial supply and its anastomoses.

In the active stages of chronic pancreatitis, partial or total hypervascularity of the organ is observed (Fig. 1, *A* and *B*; 2*A*; and 3*B*). This is most often associated with irregularity and tortuosity of small arterial twigs. We have observed pancreatic hypervascularity in some normal individuals who had no clinical suggestion or laboratory indication of disease. In these instances, however, the other arteriographic signs of chronic pancreatitis were absent.

In severe fibrosis of the pancreas and in cases of calcification, we have observed hypovascularity instead of hypervascularity. Other changes, such as arterial beading and irregularity must then be relied upon to indicate the true nature of the disease.

2. *Arterial irregularities.* This involves small arterial branches which are beaded and tortuous. Unlike carcinoma of the pancreas, the large-caliber arteries do not show any encasement or segmental narrowing as some authors have reported[7,9] (Fig. 6, *B* and *C*). This helps to differentiate the 2 entities. Also, the diffuse or multifocal distribution of arterial beading is characteristic of an inflammatory process.

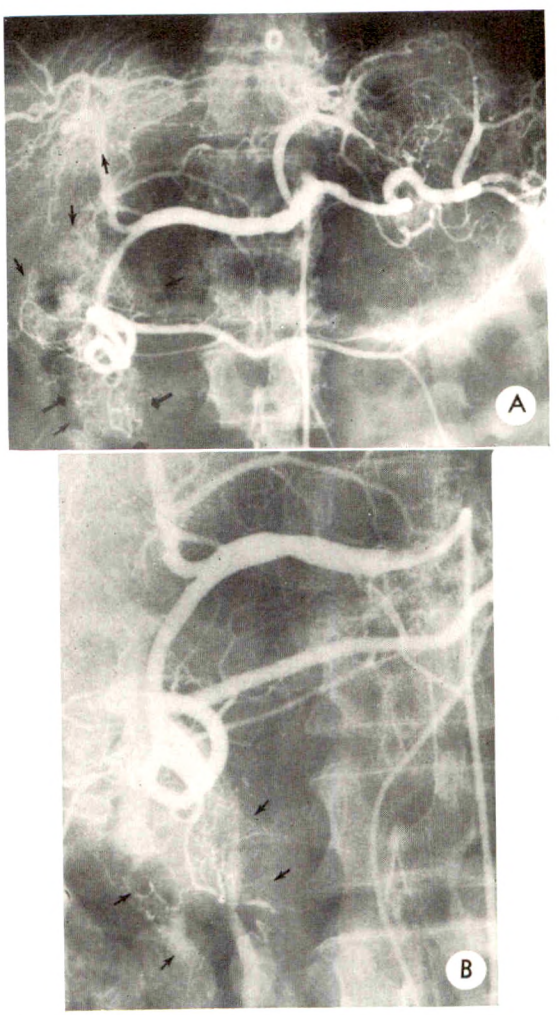


FIG. 1. Hypervascularity, blush and enlargement localized to the head of the pancreas. (*A*) Late arterial phase of celiac injection shows increased vascularity of the head. The blush has already appeared and outlines the enlarged organ (arrows). The lower arrows point to the uncinate process. The rest of the pancreas does not show hypervascularity or blush. (*B*) Superselective injection of the hepatic artery and use of the magnification technique have improved the demonstration of small vascular irregularities and beading of the uncinate process.

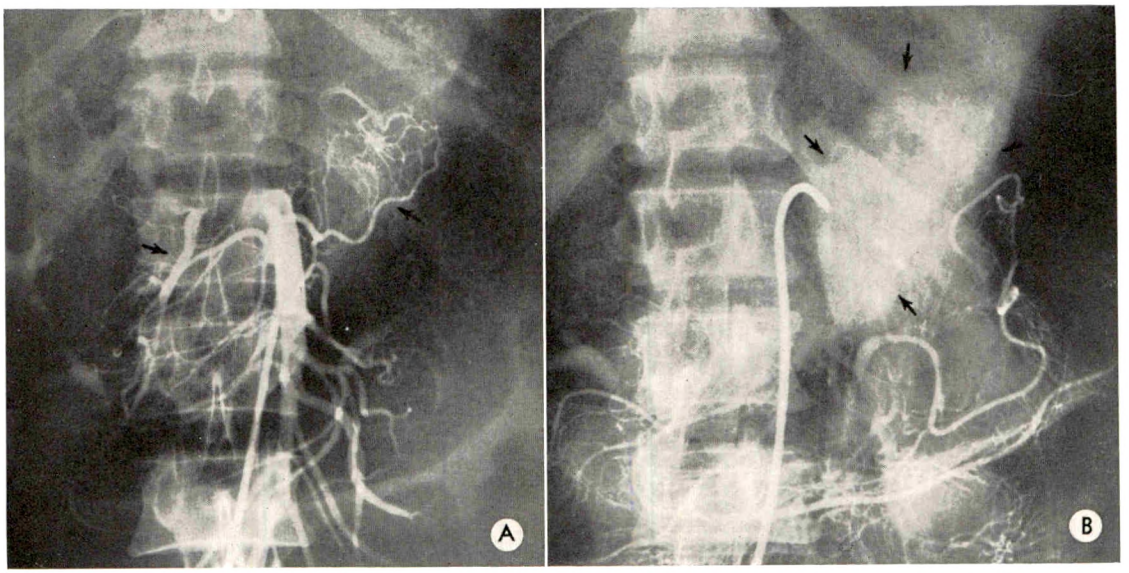


Fig. 2. Tail and body supplied by superior mesenteric artery. (*A*) The entire body and tail of the pancreas are supplied by a jejunal branch of the superior mesenteric artery (small arrow). Vascular irregularity of arterial twigs and hypervascularity of the tail and body are noted. Retrograde filling of the gastroduodenal artery (large arrow) *via* inferior pancreatic arcade is seen. (*B*) Capillary phase. Diffuse, dense blush outlines the enlarged tail and body which show an irregular border (arrows).

Beading of arterial twigs is best recognized by the magnification technique (Fig. 1*B*; and 2*A*).

The tortuous and beaded arterial twigs resemble arteriosclerotic disease; however, practically all of our patients were below the age for atherosclerosis and there were no other associated arterial irregularities in other visceral branches to cause any difficulty in differentiation of these 2 patterns. In addition, the presence of associated pancreatic blush, as will be described, has not been demonstrated in studies of the celiac axis in elderly patients. This further rules out the possibility of arteriosclerotic disease.

3. *Pancreatic blush.* A 3–4 second injection of contrast medium into the celiac axis does not produce any staining of the pancreas in the capillary phase. Occasionally, there is a faint "blush" when there is a normal hypervascularity of the pancreas, but this is not associated with the above vascular changes and in these normal cases the blush is homogeneous. Superselective and excessive injection of the pancreatic arteries also produces a blush which again is not associated with vascular irregularity and beading. The blush associated with chronic pancreatitis is diffuse and involves large areas of the pancreas (Fig. 1*A*; and 2*B*). This blushing is nonhomogeneous and sometimes outlines the entire organ (Fig. 3*C*). The blush is best visualized from the end arterial phase, during the capillary phase and into the early venous phase, or within 9 seconds of injection. After 9 seconds, the blush rapidly disappears.

Blushing is the single most common diagnostic finding. Four of our patients exhibited pancreatic calcification in plain roentgenograms. Identification of the blush in these calcified areas was difficult but could be readily seen in the other regions.

4. *Pancreatic enlargement and vessel displacement.* All or part of the inflamed pancreas may be enlarged. This is best recognized in those phases where pancreatic blushing is seen (Fig. 1*A*; and 2*B*). In most cases, enlargement of the blushed organ is due to multiple small cystic changes which

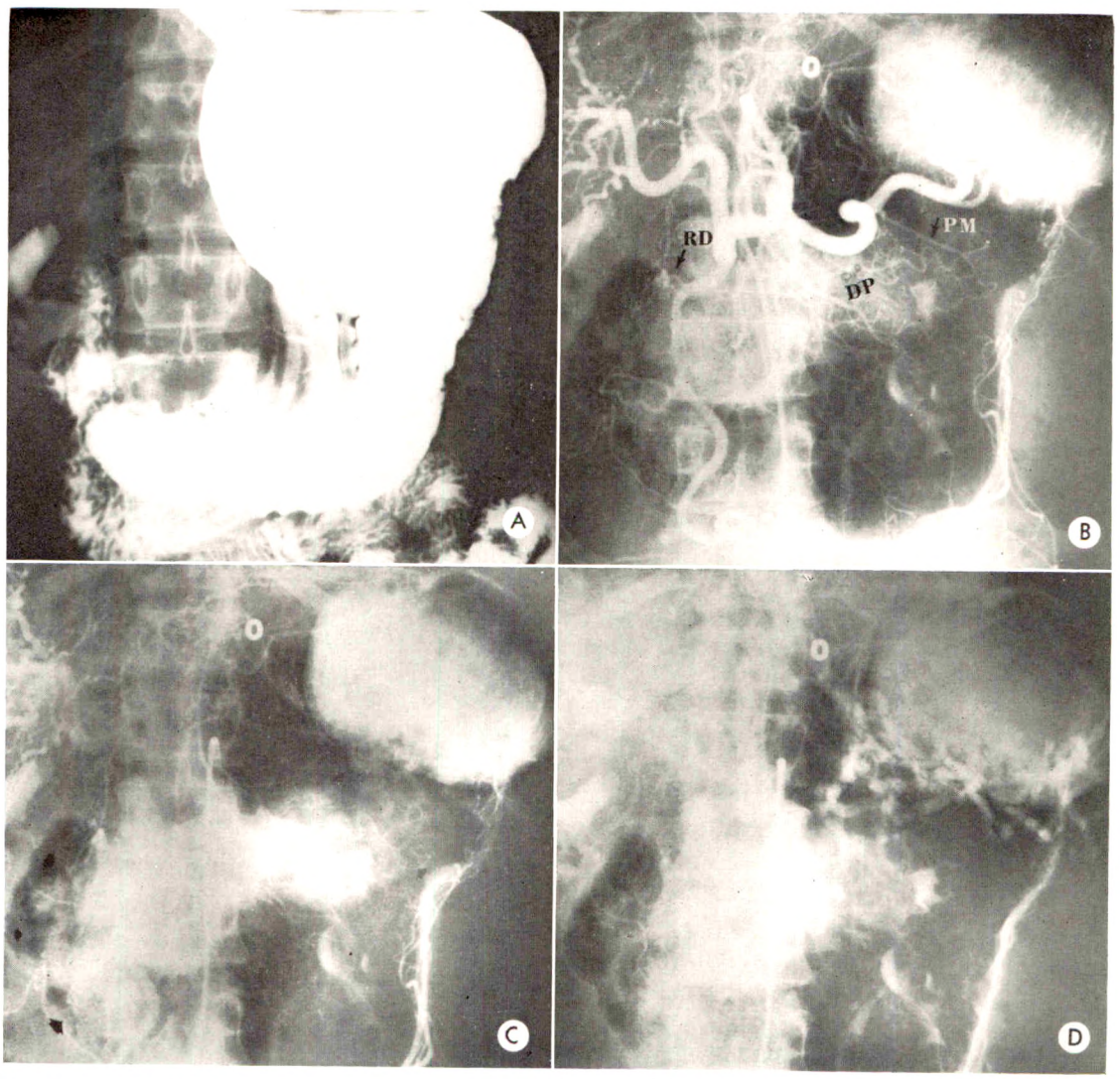


FIG. 3. Acute recurrent stage of chronic pancreatitis. All signs of chronic pancreatitis are present. (*A*) Extrinsic pressure noted on the lesser curvature of the gastric antrum by the inflamed organ. (*B*) Late arterial phase of celiac arteriography. Hypervascularity, vascular irregularity of small arterial twigs and blushing are observed. (*C*) A dense blush of the entire organ is seen. Note the irregular border of the enlarged, inflamed organ (small arrowheads). The gas-filled, second portion of the duodenum outlines the contours of the head of the pancreas (large arrowheads). (*D*) Venous phase. Nonfunctional splenic vein; the spleen drains *via* several collateral veins. This type of varicosity is difficult to visualize by barium study, as is this case, and would not cause bleeding.

give the blush its irregular pattern (Fig. 4*B*). Pancreatic enlargement may also be caused by swelling of the organ in the acute recurrent stage of chronic pancreatitis (Fig. 3, *B* and *C*). Displacement of the pancreatic vessels may also be present in the former, but it is not a consistent finding (Fig. 4*A*). Marked displacement of the vessels indicates the presence of a large pseudocyst and the stereo technique is of great assistance in the identification of displacement in the anteroposterior direction which is otherwise easily missed (Fig. 5*A*).

5. *Nonfunctioning splenic vein.* In 14 per cent of our cases, the splenic vein was not visualized or, despite faint visualization,

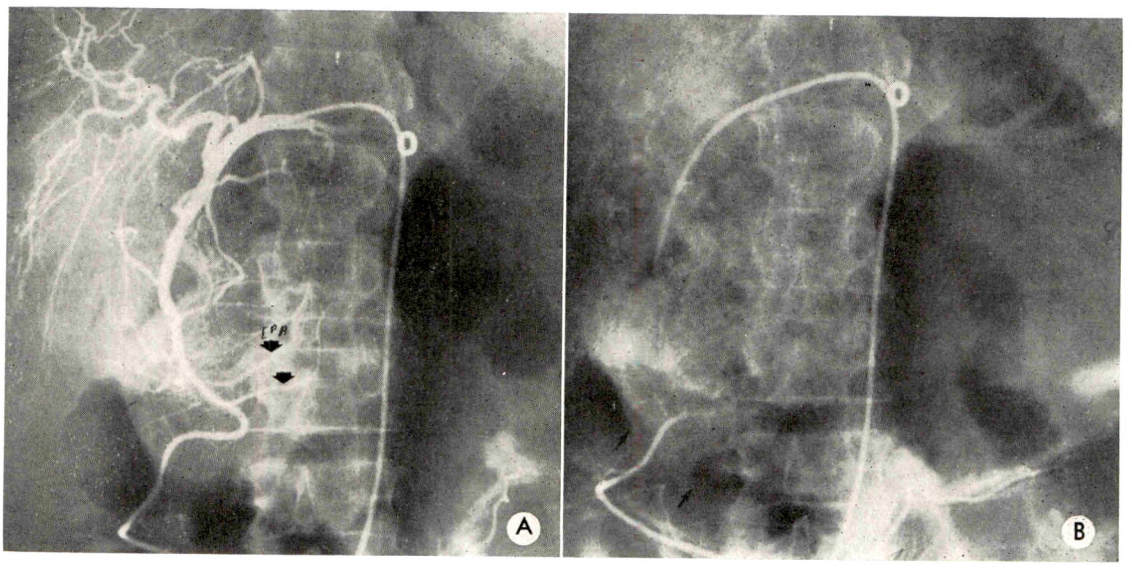


Fig. 4. Dense blush, multiple small cystic changes and large pseudocyst. (*A*) Superselective gastroduodenal injection. There is good visualization of the entire pancreatic head arterial supply which is displaced by multiple small cystic changes. (*B*) Capillary phase. There is a dense, irregular blush of the head. Multiple small radiolucencies correspond to the cystic changes which cause enlargement of this portion of the pancreas. The faint smooth border of a large pseudocyst (arrows) impinges upon the gas-filled second portion of the duodenum. Subselective injection always exaggerates the blush, but this degree of blushing cannot be entirely attributed to artifact and indicates inflammation.

was of small caliber and irregular and drainage of the spleen occurred *via* collaterals, namely the gastroepiploic, marginal omental, short gastric and retroperitoneal collateral veins (Fig. 3*D*; and 5*B*). In none of these cases, however, was there thrombosis or occlusion of the splenic vein at surgery. In all cases there were varying degrees of splenic enlargement due to stasis. We believe that the nonfunctional splenic vein could be the result of either compression or reaction to the inflammatory process of the adjacent pancreatic body and tail.

DISCUSSION

While the angiographic findings of carcinoma of the pancreas have been elaborated by several authors,[3,4,8,10] little has been written concerning the angiographic manifestations of chronic pancreatitis. Diversity of opinion exists as to the importance of some of the angiographic features of chronic pancreatitis that have been described. Rösch and Bret[11] have observed hypervascularity and pancreatic enlargement in 80 per cent of their 42 cases. However, Hepp *et al.*[6] have found marked hypovascularity without any evidence of hypervascularization in all of their 48 cases. In addition, Hepp *et al.*[6] and Moskowitz *et al.*[9] have noted narrowing of the larger arteries surrounding the pancreas. Nebesar and Pollard[10] have found the diagnosis of chronic pancreatitis difficult with diagnostic accuracy occurring in only 3 out of 10 cases.

Such inconsistent findings have led Boijsen[2] to believe that angiographic diagnosis of chronic pancreatitis has yet to achieve notable accuracy. Our findings have indicated that an array of manifestations, which varies with the stage of the disease, must form the basis for diagnosis, not just 1 or 2 signs. Also, as Boijsen[2] has implied, technical improvement is the most important consideration in reducing the number of false-positive and especially false-negative diagnoses of chronic pancreatitis. Fredens *et al.*[5] have suggested

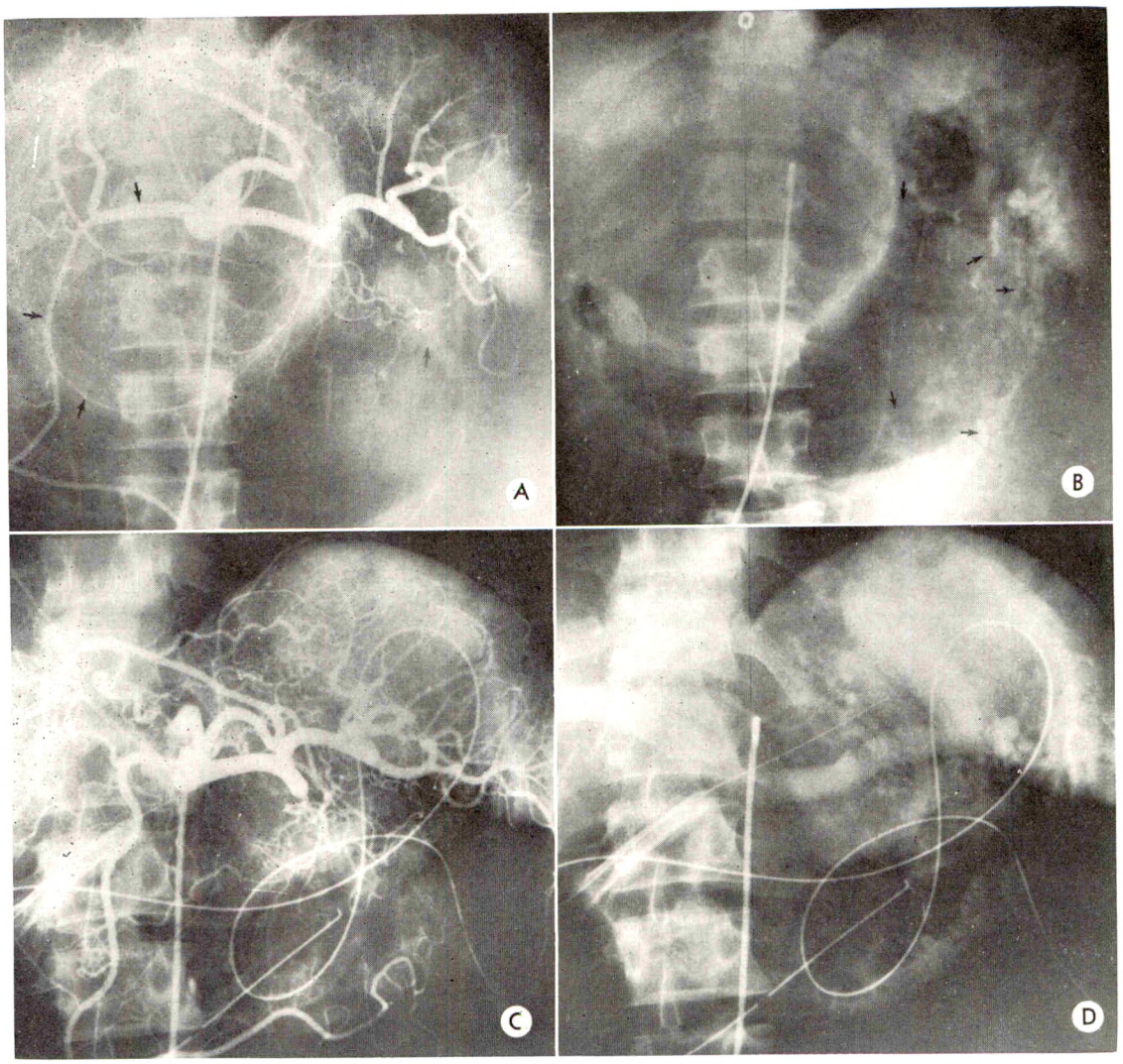


FIG. 5. Pseudocyst and nonfunctioning splenic vein. (*A*) Mid-arterial phase of celiac injection demonstrates marked displacement of hepatic, gastroduodenal and left gastric arteries draping around the large pseudocyst of the head of the pancreas (small arrow). Note that the blush is not noticeable in the inflamed pancreatic tissue immediately adjacent to the pseudocyst. In the tail, away from the cyst, the faint blush and mild hypervascularity are readily detectable (large arrows). (*B*) The splenic vein (upper arrow) is occluded by pressure of pseudocyst. The spleen drains *via* multiple collateral veins. There is moderate splenic enlargement that is usually associated with such findings. The thick-walled, infected pseudocyst is relatively radiolucent. (*C*) Arterial phase following surgery and drainage of the pseudocyst shows return of arterial branches to normal positions. The hypervascularity and vascular irregularity and blush of the inflamed tail is now more apparent. These changes are not seen in the head region which was virtually destroyed by the inflammatory process. (*D*) After removal of extrinsic pressure the splenic vein is readily draining into the portal vein and the collaterals are no longer visible. The splenic size has returned to normal.

that technical excellence is important in the improvement of diagnostic accuracy in chronic pancreatitis. It is for this reason that we emphasize the previously described technique, including the use of stereoscopic views, for identification of the pancreatic blood supply, followed by magnification for visualization of small vascular changes.

We encountered 3 cases of acute pancreatitis in the first episode. These patients do not exhibit any consistent diagnostic

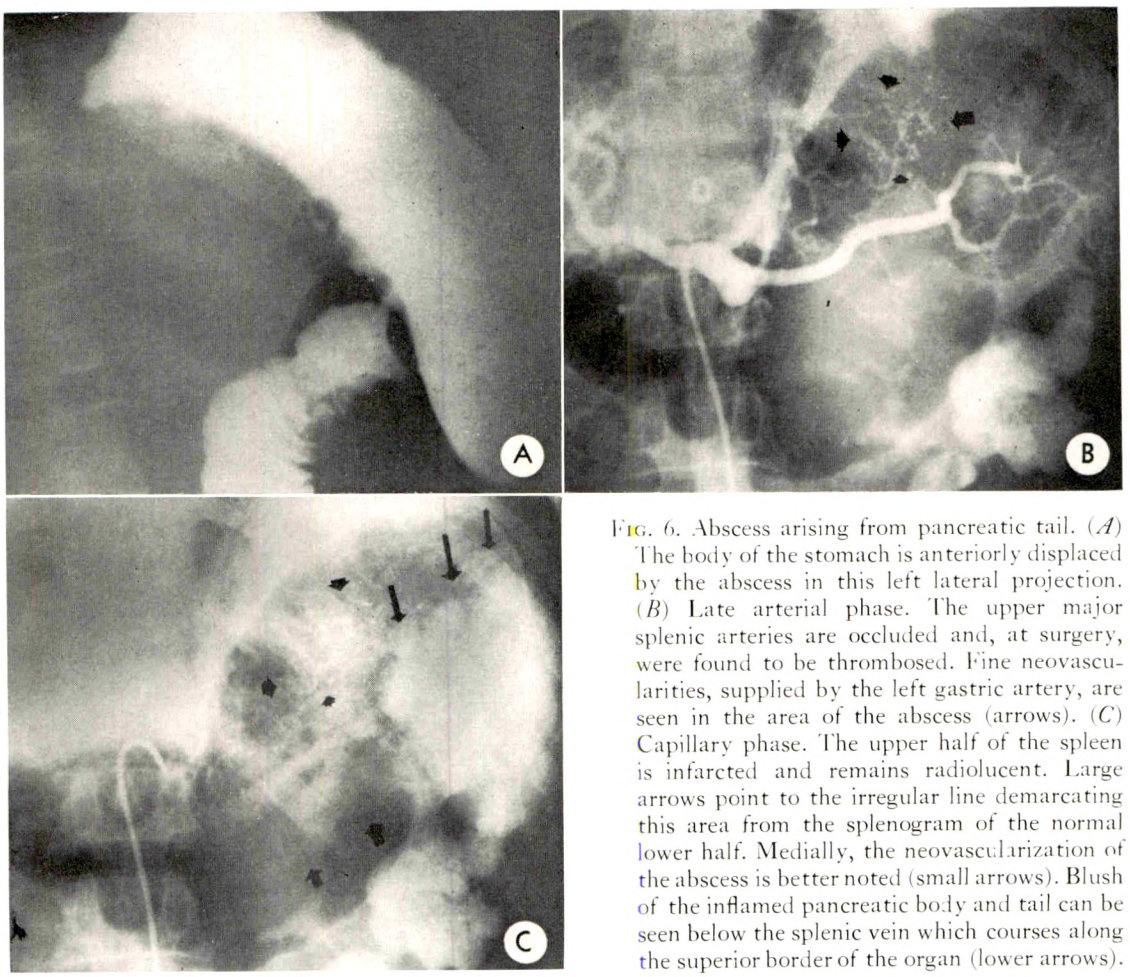


Fig. 6. Abscess arising from pancreatic tail. (*A*) The body of the stomach is anteriorly displaced by the abscess in this left lateral projection. (*B*) Late arterial phase. The upper major splenic arteries are occluded and, at surgery, were found to be thrombosed. Fine neovascularities, supplied by the left gastric artery, are seen in the area of the abscess (arrows). (*C*) Capillary phase. The upper half of the spleen is infarcted and remains radiolucent. Large arrows point to the irregular line demarcating this area from the splenogram of the normal lower half. Medially, the neovascularization of the abscess is better noted (small arrows). Blush of the inflamed pancreatic body and tail can be seen below the splenic vein which courses along the superior border of the organ (lower arrows).

angiographic features. Displacement and enlargement of the major branches of the celiac axis, which have been observed by Aakhus *et al.*,[1] are relative findings and require comparative study after subsidence of inflammation and cannot, therefore, provide an adequate basis for diagnosis of the disease.

A diagnosis of acute recurrent pancreatitis should be founded upon recognition of hypervascularity, vascular irregularity, enlargement of the inflamed organ and a dense blush which is best seen in the capillary phase. Displacement of pancreatic arteries is only seen when there is associated cystic formation.

Correlation of blush intensity to the severity of disease is, at best, difficult. It is, however, directly proportional to the degree of hypervascularity present, which is an indication of the stage of the disease.

Identification of blush over the vertebrae is difficult even using the subtraction technique. A good anatomic landmark is the splenic vein which courses along the superior border of the pancreas (Fig. 5*D*; and 6*C*). The blush of the head is not as readily seen as that of the body and tail but the presence of gas in the second portion of the duodenum provides a helpful landmark for recognition of this blush. If a large pseudocyst is present, recognition of blush in the inflamed pancreatic tissue adjacent to the pseudocyst is most difficult (Fig. 5*A*).

Regions of sclerotic or calcified pancreatic tissue exhibit a reduced amount of blushing and a paucity of the vasculature. As the chronic disease progresses, there is an increased degree of fibrosis, the organ no

longer appears hypervascular, the vascular irregularity becomes more pronounced, the blush becomes spotty and the organ enlargement subsides.

Finally, although infrequently found, we believe that the nonfunctioning splenic vein in the absence of adjacent arterial encasement is the most specific sign of chronic pancreatitis in all stages of this disease.[3]

SUMMARY

Use of an improved technique, including stereoscopic views and magnification, has made selective arteriography the most revealing test in diagnosing chronic pancreatitis. Our findings were surgically and pathologically confirmed in 45 per cent of 78 cases. In another 30 per cent, confirmation was based on clinical and laboratory findings.

Focal or generalized pancreatic blush, small arterial beading and irregularity are the most common findings. Hypervascularity and enlargement of the organ in acute recurrent or active stages and hypovascularity in advanced fibrosis were observed. Nonfunctioning splenic vein, seen in 14 per cent of cases, and in absence of adjacent arterial encasement, is the most reliable sign of pancreatitis of tail and body.

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VOL. 119, NO. 1

EFFECTS OF PANCREATITIS ON THE SMALL BOWEL AND COLON: SPREAD ALONG MESENTERIC PLANES*

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THE purpose of this report is to document that localized changes on characteristic portions of the small intestine and colon are produced by the extravasated enzymes of pancreatitis which follow definite anatomic planes. The mesenteric pathways most commonly involved which direct the spread of the pancreatic enzymes to remote sites in the alimentary tract are the transverse mesocolon and the small bowel mesentery.

ANATOMIC CONSIDERATIONS

Virtually all of the pancreas is an extraperitoneal organ. However, the reflections of the posterior parietal peritoneum to form the 2 leaves of the transverse mesocolon result in a nonperitonealized bare area. This extends across the lower border of the organ anteriorly (Fig. 1). On the right, the transverse mesocolon begins at the point where the anterior hepatic flexure crosses immediately ventral to the second portion of the duodenum. The bare area thus extends as a broad strip across the infraampullary portion of the descending duodenum. The ascending colon and the proximal, more posterior hepatic flexure are completely extraperitoneal.

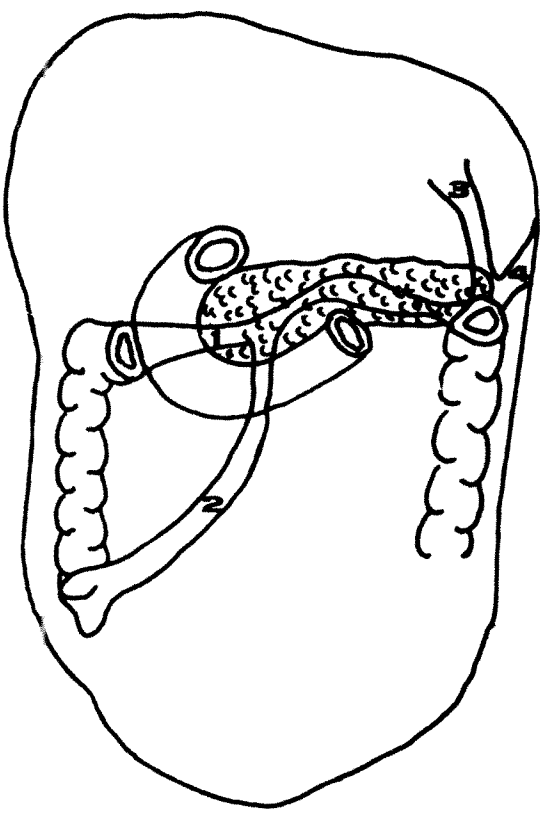


FIG. 1. Relationships of the pancreas to the transverse mesocolon (1) and small bowel mesentery (2). The lienorenal (3) and phrenicocolic (4) ligaments are shown.

The tail of the pancreas, after extending across the left kidney, is actually intraperitoneal in that it is incorporated or ensheathed within the leaves of the lienorenal ligament. Anatomic continuity is established by the peritoneal reflections constituting the supporting ligaments of the left upper quadrant. Figures 2 and 3 show the relationships of the tail of the pancreas within the lienorenal ligament to the transverse mesocolon, phrenicocolic ligament, and the peritoneal reflections of the descending colon. The phrenicocolic ligament[8] is attached to the anatomic splenic flexure of the colon and represents the most lateral continuity of the transverse mesocolon and lienorenal ligament. It thereby also marks the site of transition from the intraperitoneal transverse colon to the extraperitoneal descending colon.

Of further anatomic significance is the

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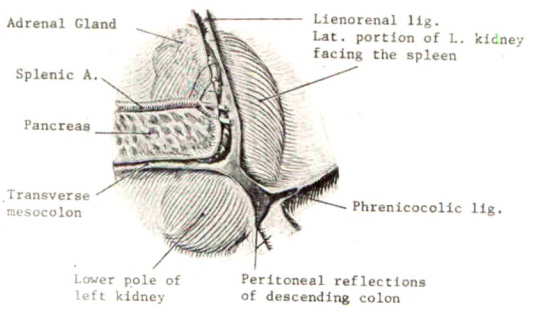


FIG. 2. Posterior peritoneal reflections of the left upper quadrant, demonstrating continuity of the transverse mesocolon and lienorenal ligament with the phrenicocolic ligament and the peritoneal reflections of the descending colon. The tail of the pancreas resides within the lienorenal ligament. (After Corning.[4])

fact that the root of the small bowel mesentery at its origin near the inferior portion of the pancreas is continuous with that of the transverse mesocolon (Fig. 1). It is in relationship at this point to the mid-portion of the transverse duodenum. It then extends obliquely toward the right lower quadrant to insert, most commonly, at the cecocolic junction.[21] Anatomic continuity is thus established along the root of the mesentery from the pancreas to jejunal loops, ileal loops, and the cecum.

In this way, mesenteric planes are provided for the direct spread of extravasated pancreatic enzymes (Fig. 4): (1) along the mesocolon to the transverse colon. Extension along its planes to its lateral limits proceeds (a) on the right, to the hepatic flexure or, perhaps *via* the duodenum, distal ascending colon and (b) on the left, to the anatomic splenic flexure just below the tip of the spleen; and (2) along the small bowel mesentery to jejunal loops and the ileocecal region.

1. EFFECTS OF PANCREATITIS ON COLON: SPREAD ALONG TRANSVERSE MESOCOLON

The segment of large intestine involved by pancreatitis reflects the pathophysiology of the disease and the anatomic relation-

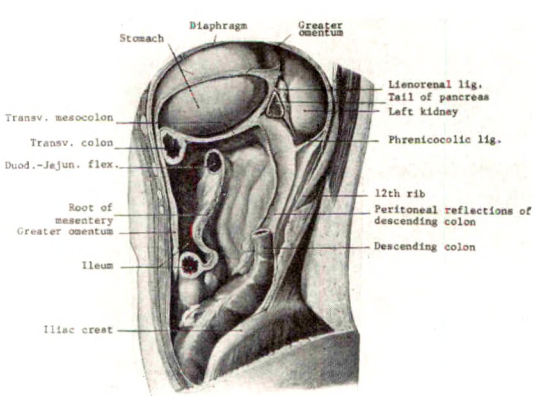


FIG. 3. Drawing of left parasagittal section, emphasizing peritoneal reflections. Note the tail of the pancreas within the lienorenal ligament and its continuity with the transverse mesocolon and phrenicocolic ligament. (After Corning.[4])

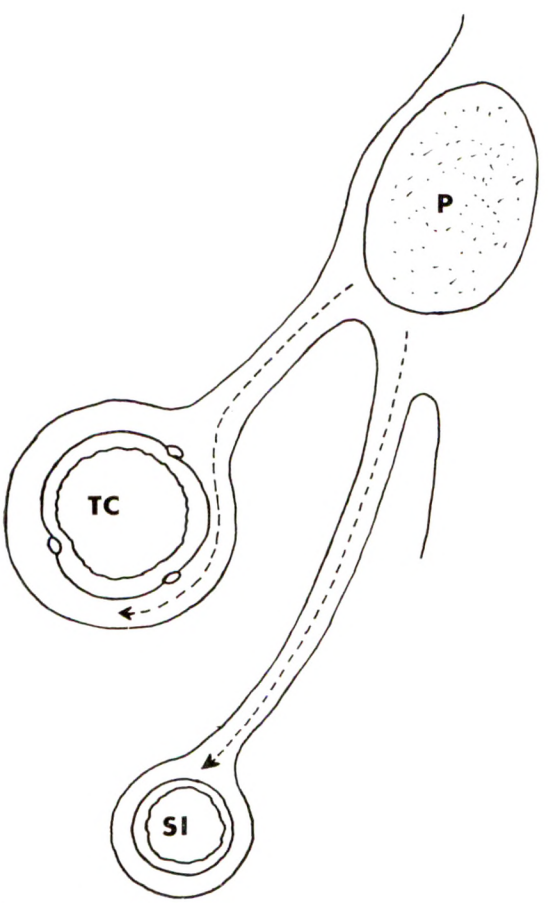


FIG. 4. Sagittal diagram illustrates pathway of exudate from pancreas (P) within the mesocolon to the transverse colon (TC), characteristically toward its lower border, and *via* the small bowel mesentery to intestinal loops (SI.)

ships of the pancreas to the fascial planes of the transverse mesocolon. Inflammation localized to the head of the organ, as most typified by acute suppurative pancreatitis, may extend to the hepatic flexure or, perhaps *via* a duodenal fistula, to the distal ascending colon. Diffuse pancreatitis may spread to involve the entire transverse colon, with a definite tendency to exert its major effects upon the splenic flexure.

REPORT OF A CASE

Stricture of the distal ascending colon secondary to suppurative pancreatitis. A 76 year old white female was admitted with acute severe epigastric pain which was constant and nonradiating. This was accompanied by nausea and vomiting. The pain gradually spread to involve the entire abdomen as well as both flanks. There was no history of dark urine, light stools, hematemesis or melena.

Eight years prior to admission, the patient first noted the onset of intermittent attacks of midepigastric pain usually initiated by fatty food intake and associated with mild nausea and vomiting. The pain was usually relieved by vomiting. She was told at that time that she had gallbladder disease and was placed on a low fat diet. Five years prior to admission, following an episode of unrelenting abdominal pain, an exploratory laparotomy disclosed a large pancreatic abscess involving the body of the pancreas. *A. aerogenes* was cultured. The abscess was drained and she did well postoperatively.

Pertinent physical and laboratory findings included generalized abdominal guarding with some rebound tenderness and referred pain to the epigastrium, leukocytosis and an initial serum amylase of 2,800 units (normal 40–140). On conservative therapy, the abdominal distress improved and the serum amylase quickly returned to normal.

Two oral cholecystographies showed nonvisualization of the gallbladder. Intravenous cholangiography (Fig. 5*A*) revealed a common bile duct dilated to 1.6 cm. with incomplete obstruction of its distal segment. There was no evidence of calculi but the gallbladder failed to opacify. Coincident renal excretion strongly suggested incomplete obstruction of the right ureteropelvic junction at the same level as that involving the common bile duct. This was confirmed on a subsequent excretory urogram. A barium enema study demonstrated a circumferential constricting lesion of the distal ascending colon with the appearance of an annular carcinoma (Fig. 5, *B* and *C*).

The patient was considered to have recurrent acute pancreatitis secondary to biliary tract disease, and a carcinoma of the colon. Exploratory laparotomy disclosed a large peripancreatic abscess with retroperitoneal extension down to and involving the hepatic flexure and distal ascending colon, where an extramural inflammatory stricture was encountered. Areas of focal fat necrosis were present in the transverse mesocolon, pericolonic tissue and greater omentum. No neoplasm was present. The gallbladder was chronically inflamed, but contained no calculi. A right hemicolectomy with an ileotransverse colostomy, cholecystectomy with a negative common duct exploration, and drainage of the peripancreatic abscess were performed. *E. coli* and the *Klebsiella enterobacter* were cultured.

Her postoperative course was complicated by large quantities of drainage from the T-tube in amounts of 1,500–4,000 cc./day. This was demonstrated to contain up to 30,000 units amylase, verifying gross drainage of pancreatic juice into the duodenum. A T-tube cholangiogram revealed a sinus tract connection from the midportion of the third duodenum to the extraperitoneal space (Fig. 5*D*). An upper gastrointestinal series then demonstrated gross extravasation from the duodenal loop extraperitoneally toward the right (Fig. 5, *E* and *F*). The huge sinus drained along the superior contour of the proximal transverse colon and anastomosing ileum with an extension downward in the flank to the original site of stricture in the ascending colon. The fistula gradually closed clinically and a follow-up T-tube cholangiogram was negative, without evidence of extravasation from the duodenum. Five and a half weeks after surgery, the patient was discharged with the T-tube clamped but in place.

Comment. The mimicry of an annular carcinoma by the inflammatory process extending from the pancreas is striking. The lesion presents as a relatively short area of narrowing with apparent shelf-like

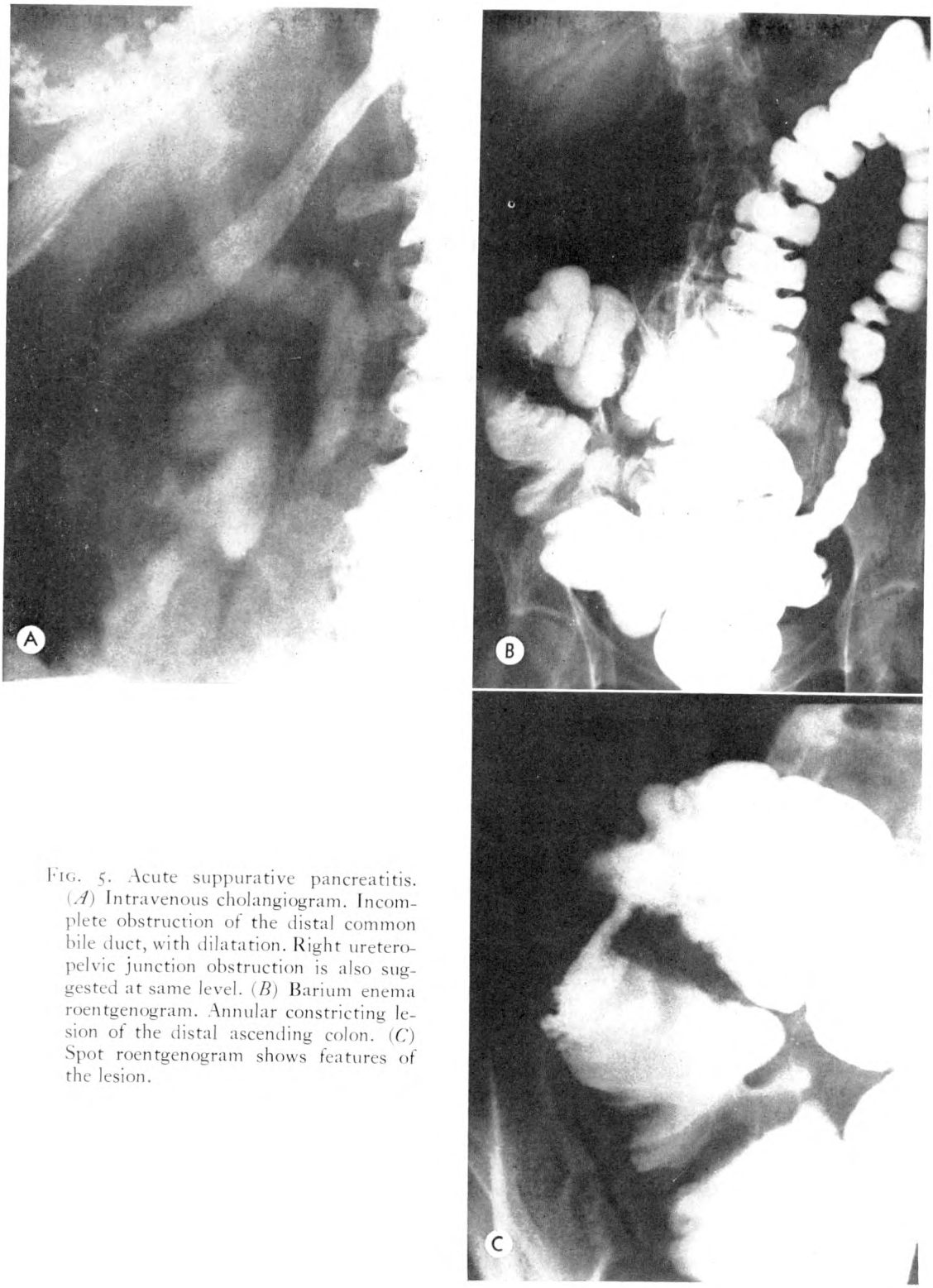


FIG. 5. Acute suppurative pancreatitis. (*A*) Intravenous cholangiogram. Incomplete obstruction of the distal common bile duct, with dilatation. Right ureteropelvic junction obstruction is also suggested at same level. (*B*) Barium enema roentgenogram. Annular constricting lesion of the distal ascending colon. (*C*) Spot roentgenogram shows features of the lesion.

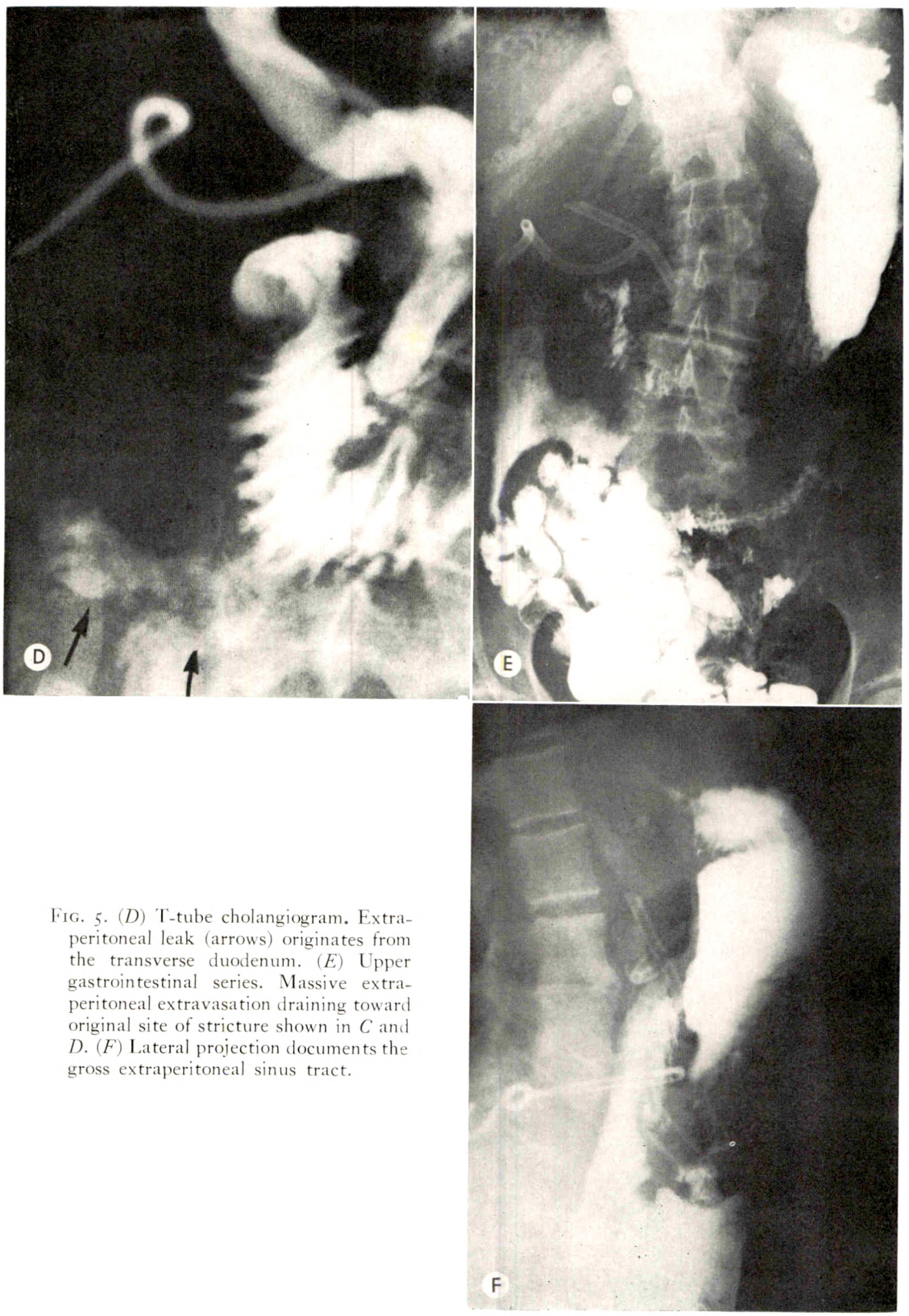


Fig. 5. (*D*) T-tube cholangiogram. Extraperitoneal leak (arrows) originates from the transverse duodenum. (*E*) Upper gastrointestinal series. Massive extraperitoneal extravasation draining toward original site of stricture shown in *C* and *D*. (*F*) Lateral projection documents the gross extraperitoneal sinus tract.

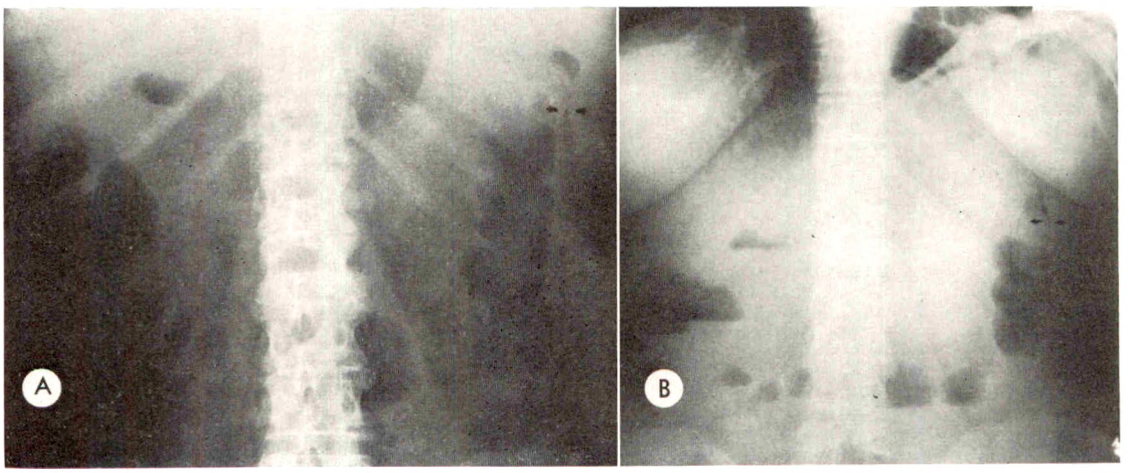


FIG. 6. Acute pancreatitis. (*A*) Supine and (*B*) erect roentgenograms. Dilatation of the transverse colon extends to a narrowed segment at the splenic flexure (arrows).

overhanging margins. The distal ascending colon is an unusual site to be involved by pancreatitis. It occurred *via* a fistula from the pancreas to the duodenum and then *via* gross extraperitoneal extravasation from the duodenum. Areas of fat necrosis were conspicuous along the pathway clearly documented by the contrast studies. Obstruction of the common bile duct and right ureteropelvic junction in the same plane, in association with the colonic changes, indicates tumefactive effusion of the anterior pararenal space.[10]

On plain film roentgenograms, the most common abnormality in the large intestine seen in cases of acute pancreatitis is gaseous distention of the transverse colon. This often represents a paralytic ileus secondary to the extravasated enzymes within the transverse mesocolon. It has recently been shown that the natural extension of the inflammatory process into the phrenicocolic ligament may result in severe spasm or an incomplete mechanical obstruction at the level of the anatomic splenic flexure immediately inferior to the tip of the spleen.[8] The obstruction need not be permanent and may resolve as the pancreatitis subsides. Figure 6, *A* and *B*, illustrates dilatation of the transverse colon up to a strikingly narrowed segment of the splenic flexure during an acute episode of pancreatitis. Barium enema study 1 week after conservative therapy showed no abnormalities, indicating that this was spasm due to the irritating effects of the enzymes within the phrenicocolic ligament. However, if the inflammation is more severe and particularly if accompanied by fat necrosis, narrowing of the splenic flexure with irregular nodular or serrated margins and distorted mucosal folds may be found secondary to the extramural inflammatory infiltrate. A granulomatous and fibrotic reaction may then result in a stricture of the splenic flexure.

REPORT OF A CASE

Stricture of the splenic flexure secondary to traumatic pancreatitis. This 47 year old Negro male was first admitted because of injuries sustained while drinking on the previous day. In addition to face and head lacerations, there were fractures of the left 5th–9th anterior ribs, an acutely tender abdomen and moderate distention. There was mild left upper quadrant and left lower quadrant tenderness. Laboratory findings included a leukocytosis, a hematocrit of 42 per cent which remained stable, a serum amylase of 275–340 units, and negative stool guaiac tests. A chest roentgenogram showed atelectasis and questionable pneumonia at the left base and an abdominal roentgenogram

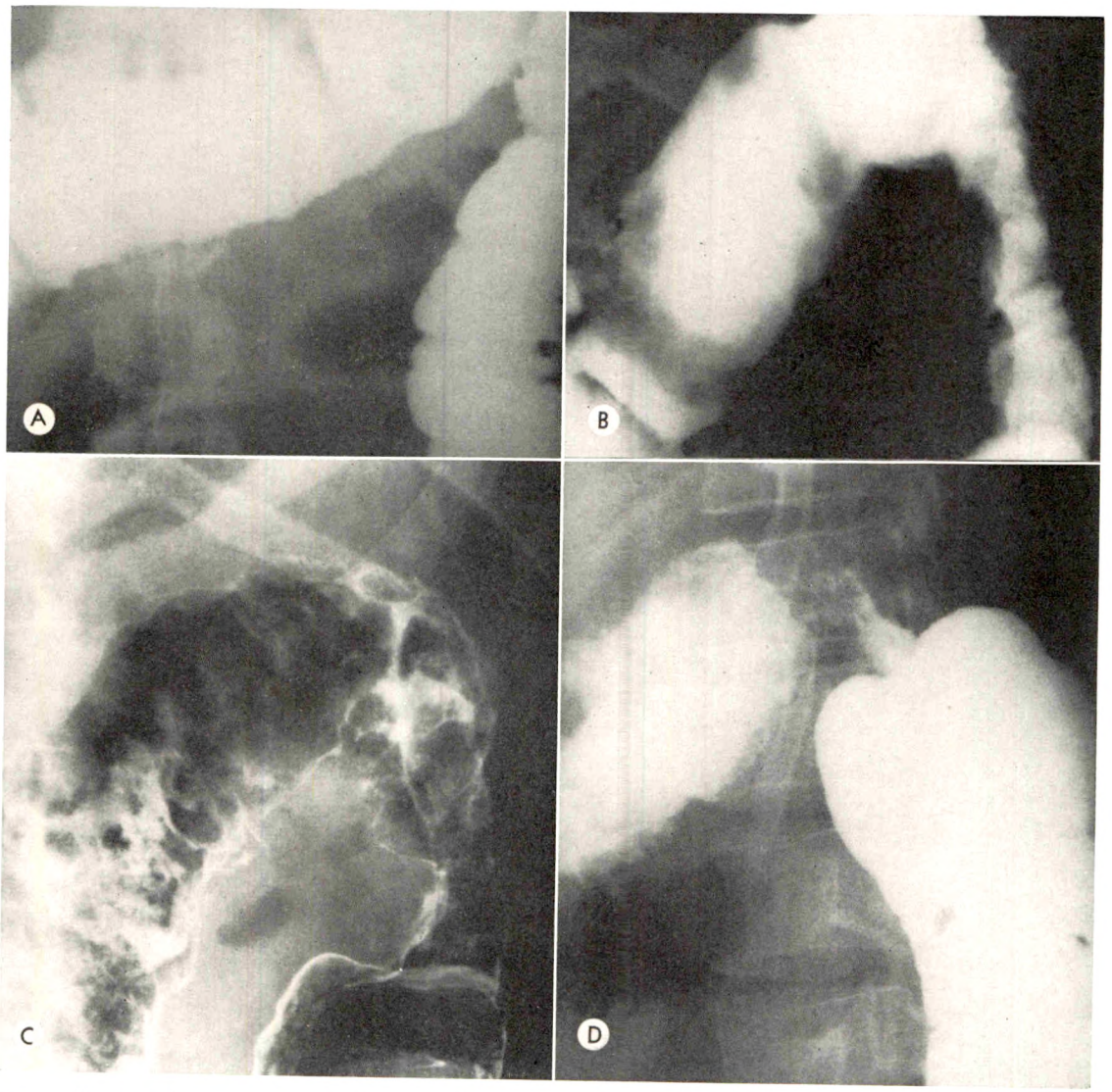


Fig. 7. Traumatic pancreatitis. (*A*) Flattening of the inferior haustral contour of the transverse colon. Pseudosacculations result on the uninvolved superior border. The process ends at the level of the phrenicocolic ligament. (*B* and *C*) Scalloped narrowing of the splenic flexure on filled and air contrast studies. The intramural lesions end precisely at the level of the phrenicocolic ligament. (*D*) Three months later there is a marked stenosis of the splenic flexure.

revealed gaseous distention of the transverse colon. Barium enema examination disclosed irregular flattening of the lower border of the mildly distended transverse colon (Fig. 7*A*). Narrowing with irregular nodular defects of the splenic flexure were confirmed on fluoroscopy with air contrast studies (Fig. 7, *B* and *C*). The process ended precisely at the level of the phrenicocolic ligament. An upper gastrointestinal and small bowel series was negative.

Following 1 month of hospitalization and conservative therapy, he was discharged asymptomatic. Two months later, he began to have daily episodes of lower abdominal cramping pain and was readmitted following a barium enema examination demonstrating a severe stricture of the splenic flexure (Fig. 7*D*). Complete blood cell count and serum amylase determination were within normal limits. Stool guaiac test was 2–3+ on 2 occasions.

Surgical resection of the splenic flexure revealed on pathologic study a chronic inflam-

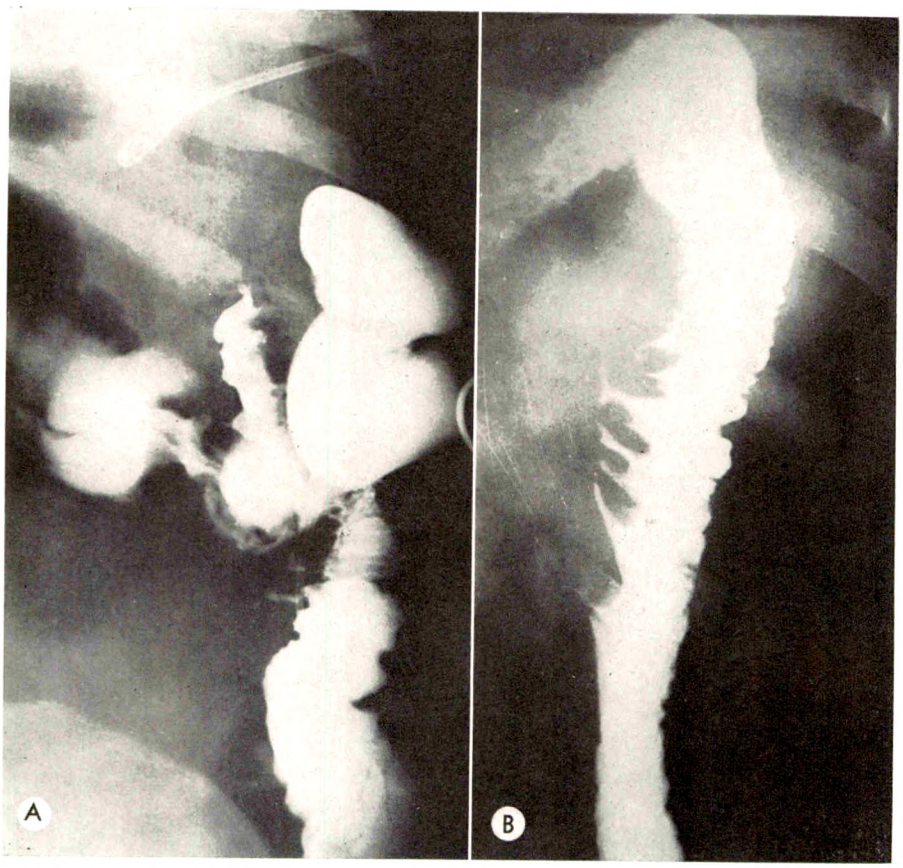


FIG. 8. Gas producing abscess of pancreas. (*A*) Multiple gaseous lucencies in tail of pancreas at site of draining catheter. Barium enema roentgenogram demonstrates extension *via* phrenicocolic ligament to anatomic splenic flexure with multiple sinus tracts of varying size. (*B*) Four months later, multiple residual sinus tracts are present. (Courtesy of Jeanne Baer, M.D., St. Luke's Hospital, New York, New York.)

matory process with marked stenosis of the colon to a circumference of 1.5 cm., secondary to traumatic pancreatitis. Histologic sections through the area of constriction showed a great deal of inflammatory exudate in the mucosa. Focal ulcerations of the mucosa were also present. The exudate was comprised mostly of plasma cells and lymphocytes and extended from the mucosa through the muscularis. Granulation tissue and fibrosis were evident here. There was no evidence of any remnant of a diverticulum and no evidence of diverticulosis proximal or distal to the stenotic zone.

Comment. Asymmetric involvement of the lower contour of the transverse colon (Fig. 7*A*) is characteristic of extension of disease from the pancreas along the transverse mesocolon.[12] The lesions originally demonstrated in the splenic flexure (Fig. 7, *B* and *C*) are obviously organic and not functional in nature and thus indicate direct passage of considerable pancreatic enzymes into the phrenicocolic ligament. A fracture of the left 11th posterior rib may result in hemorrhage into this ligament[8] but none was present in this particular site. From the severe changes in the splenic flexure, the possible future development of a fibrotic stricture might have been anticipated and serial evaluations by barium enema studies obtained. Noteworthy is the fact that the process narrowed the lumen to a diameter of less than 3 mm. in only 3 months.

If the inflammatory process erodes through the wall of the colon at this site,

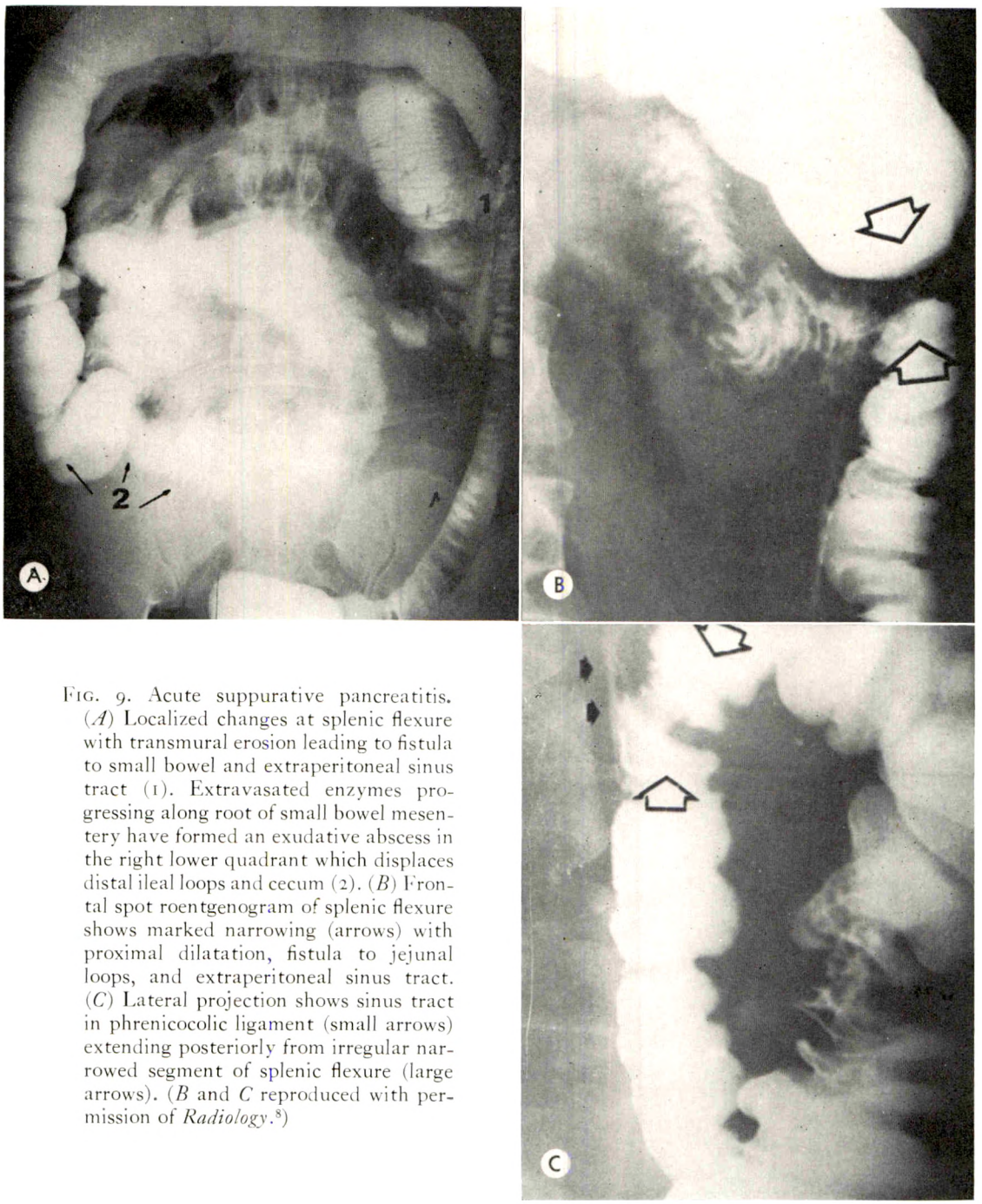


Fig. 9. Acute suppurative pancreatitis. (*A*) Localized changes at splenic flexure with transmural erosion leading to fistula to small bowel and extraperitoneal sinus tract (1). Extravasated enzymes progressing along root of small bowel mesentery have formed an exudative abscess in the right lower quadrant which displaces distal ileal loops and cecum (2). (*B*) Frontal spot roentgenogram of splenic flexure shows marked narrowing (arrows) with proximal dilatation, fistula to jejunal loops, and extraperitoneal sinus tract. (*C*) Lateral projection shows sinus tract in phrenicocolic ligament (small arrows) extending posteriorly from irregular narrowed segment of splenic flexure (large arrows). (*B* and *C* reproduced with permission of *Radiology*.[8])

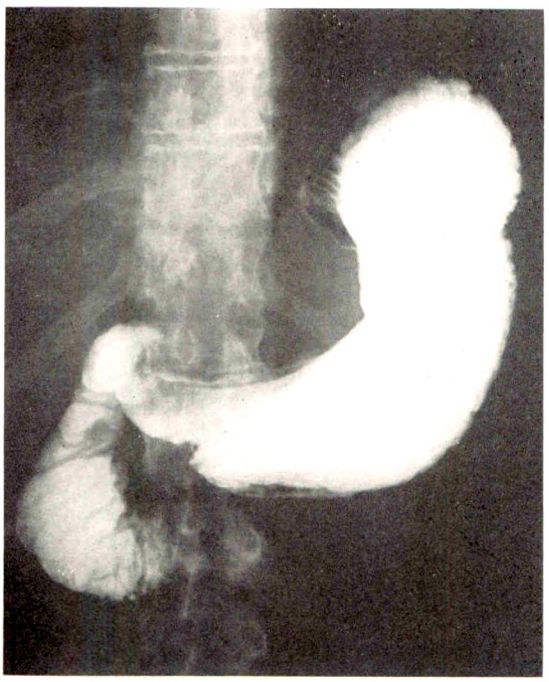


FIG. 10. Obstruction of the midportion of the transverse duodenum by pancreatitis extending down the mesenteric root.

extraperitoneal sinus tracts or fistulas to adherent small bowel loops may be demonstrated (Fig. 8, *A* and *B*; and 9, *A–C*).

2. EFFECTS OF PANCREATITIS ON DUODENUM, SMALL BOWEL AND CECUM: SPREAD ALONG SMALL BOWEL MESENTERY

The inflammatory process associated with extravasated pancreatic enzymes may disseminate along the root of the small bowel mesentery, either for a portion or its entire length.

Compression of the third portion of the duodenum may result from an indurated mesenteric root. This typically occurs in its midportion which may appear sharply cut off (Fig. 10), with dilatation and stasis proximally. Such a process undoubtedly accounts for many instances of the duodenal "ileus" seen in acute pancreatitis.

REPORT OF A CASE

Malabsorption and jejunal atrophy secondary to chronic pancreatitis. This 58 year old white male suffered from a malabsorption syndrome of unknown etiology for a period of 8 years, ultimately resulting in his death.

Figure 11 shows severe changes involving the duodenum and multiple jejunal loops.

Postmortem examination revealed the pancreas to be extensively replaced by dense fibrocollagenous tissue as well as adipose tissue with large areas of fat necrosis. The jejunum showed atrophic mucosa with occasional areas of fine scarring up to 1 cm. in diameter. The muscular layers were thin. Interestingly, 7 years ante mortem a surgical pathology report was said to reveal chronic ulcers and atrophy of the jejunal mucosa. The postmortem pathology impression was chronic pancreatitis.

Acute pancreatitis may clinically mimic an acute appendicitis, with peritoneal signs localized predominantly in the right lower quadrant. This is a reflection of the process spreading along the length of the

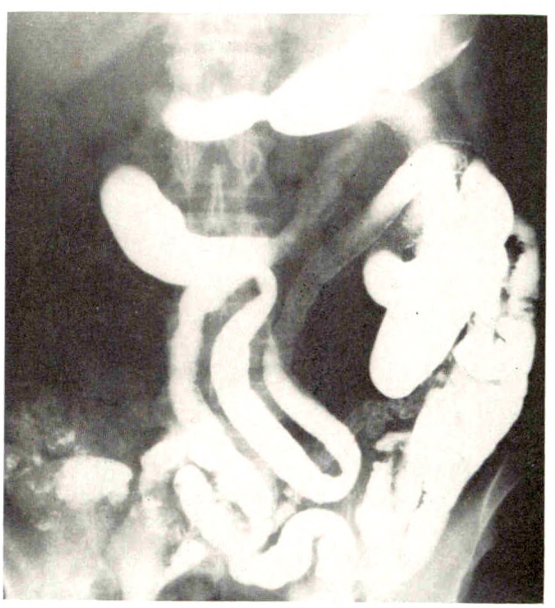


FIG. 11. Chronic pancreatitis. The most pronounced changes in this case involve the duodenum and proximal jejunum which are strikingly abnormal in appearance. The duodenal loop has a dilated moulage-like appearance. The duodenum and loops of jejunum have a tubular "toothpaste"-like aspect due to the absence of a mucosal relief pattern. Despite the cast-like appearance, the involved loops are flaccid, distensible and lack evidence of fixation, rigidity or thickening.

root of the small bowel mesentery to result in inflammatory changes of distal ileal loops or the cecum. Plain film roentgenographic findings may then include localized ileus of small bowel loops in the right lower quadrant (Fig. 12). This may be a transient, irritative phenomenon. At times, it may portend the development of an exudative abscess which can be demonstrated by its displacement of or mass effects upon ileal loops and the cecum (Fig. 9*A*; 13, *A* and *B*; and 14). Since this occurs only with severe pancreatitis, concomitant effects upon other portions of the bowel are usually present (Fig. 9*A*; 13, *A* and *B*; and 14).

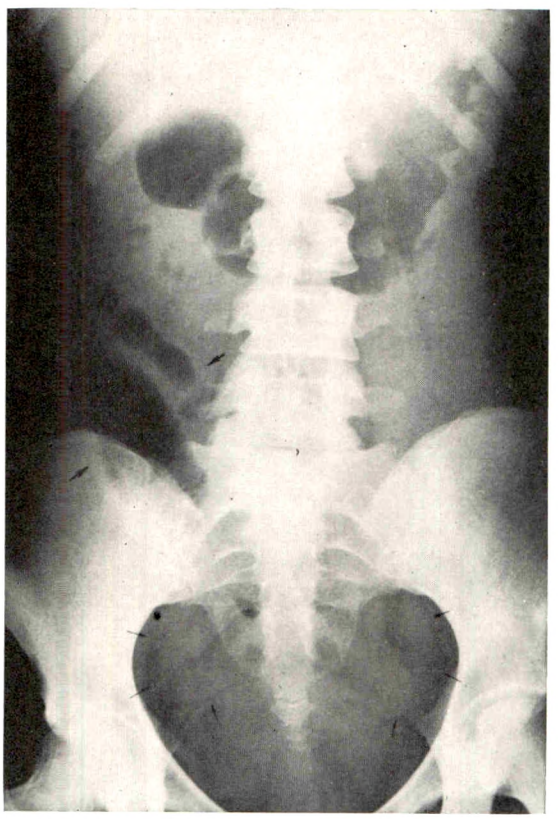


Fig. 12. Acute hemorrhagic pancreatitis. Extravasated enzymes passing down the length of the root of the small bowel mesentery produce localized distention of distal ileal loops (large arrows). There is also free intraperitoneal fluid in the pelvis (small arrows).

DISCUSSION

Acute inflammation of the pancreas may be classified into 4 groups: (1) acute edematous pancreatitis; (2) acute hemorrhagic pancreatitis; (3) acute gangrenous pancreatitis; and (4) acute suppurative pancreatitis. Each may be associated with areas of fat necrosis. Waring and Griffiths[22] have observed at operation the extravasated pancreatic enzymes and inflammatory process infiltrating between the layers of the transverse mesocolon and into the root of the small bowel mesentery. Fat necrosis is most common in these 2 sites and in the omentum.[22]

Baylin and Weeks[2] reported a case of fatal acute hemorrhagic pancreatic necrosis and noted the persistent spasm with alteration of the mucosal pattern in the mid-transverse colon and splenic flexure on barium enema examination. In 1956, Price[14] coined the term "colon cut-off" sign in acute pancreatitis for isolated gaseous distention of the ascending colon and hepatic flexure with sharp delimitation of the gas shadow just to the left of the flexure. In 2 patients, he noted a cellulitis-like process spreading from the edematous head of the pancreas between the 2 layers of the transverse mesocolon to the mesocolic border of the colon just to the left of the hepatic flexure. Aronson and Davis[1] documented a "cut-off" of gas in the proximal transverse colon on plain film roentgenograms and a persistent irregular annular constriction just distal to the hepatic flexure during an acute episode of chronic relapsing pancreatitis. The appearance closely resembled an annular carcinoma on barium enema examination. However, air studies the next day and a repeat barium enema examination 3 days later demonstrated that the lesion was no longer present and it was therefore attributed to localized spasm from irritation by the acute pancreatitis. Since Price's report, however, the term has lost its precise definition and has been applied to multiple sites of spasm and narrowing of the colon secondary to pan-

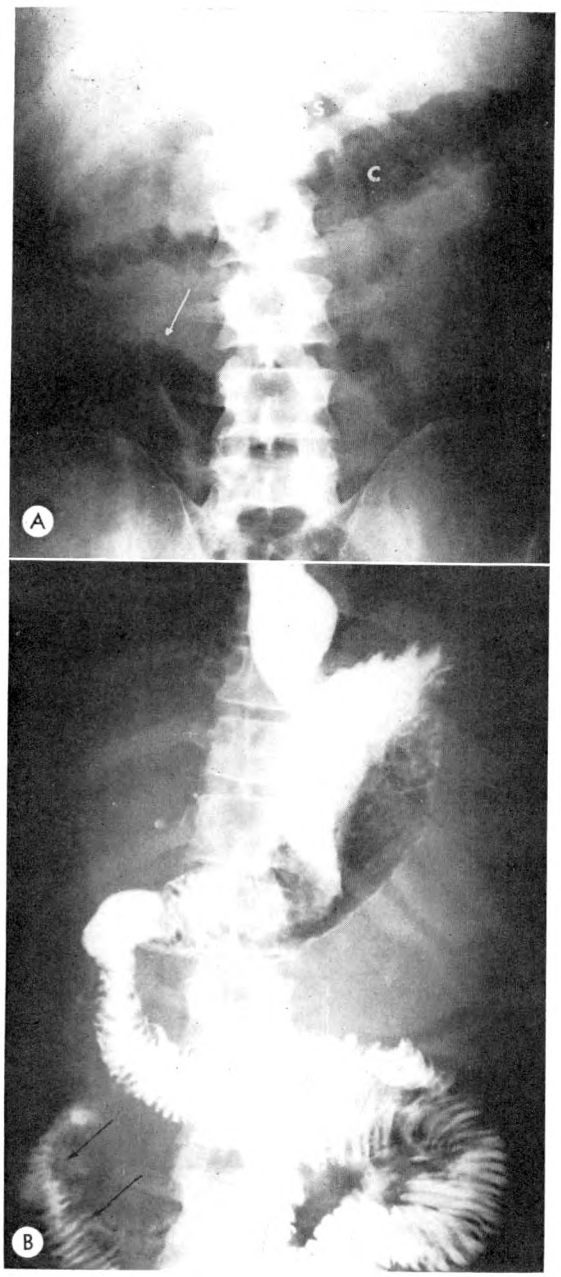


FIG. 13. Acute suppurative pancreatitis. (*A*) Admission roentgenogram shows small bowel ileus predominantly in the right lower quadrant (arrow). Note the normal relationship of the stomach (S) and transverse colon (C). (*B*) Barium study 8 days later documents the presence of an exudative abscess displacing distal ileal loops (arrows) at the end of the small bowel mesentery and the development of a large pseudocyst in the tail of the pancreas. (Reproduced with permission of *Radiology*.[9])

creatitis. Stuart[20] employed it to designate collapse with absence of gas in the midportion of the transverse colon which appeared "cut-off" from the gas containing hepatic and splenic flexures. This was a constant finding in both supine and erect plain roentgenograms of the abdomen in 6 patients with acute pancreatitis. Brascho and co-authors[3] found the gaseous dilatation to involve the ascending and entire transverse colon, with the "cut-off" point at the splenic flexure. On barium enema study, narrowing of the splenic flexure with irregular serrated margins and dis-



FIG. 14. Acute pancreatitis extending the length of the small bowel mesentery has resulted in a right lower quadrant exudative abscess displacing the terminal ileum and cecum (arrows). Changes are also seen in the midtransverse colon near the confluence of the roots of the transverse mesocolon and small bowel mesentery. (Reproduced with permission of *Radiology*.[9])

torted mucosal folds may be found secondary to the extramural process. Occasionally, a significant degree of retrograde obstruction may be met. Schwartz and Nadelhaft[18] have cited changes in the splenic flexure secondary to pancreatitis which may simulate carcinoma. Sahebjami and co-authors[17] reported a patient who presented with massive rectal bleeding as the major clinical manifestation of acute pancreatitis. A persistently narrowed segment of colon at the splenic flexure was proved at autopsy to be secondary to a large hematoma in the hilum of the spleen which had dissected along the transverse colon and perforated the serosal surface to communicate with the lumen at the splenic flexure.

Forlini[5] reported the first case of stenosis of the colon secondary to chronic pancreatitis. The splenic flexure of the colon was found to be constricted by an inflammatory mass, and was adherent to the tail of the pancreas. Gross and microscopic examination of the pancreas showed acute and chronic pancreatitis, with fat necrosis. Remington *et al.*[15] reported 2 similar cases with roentgenologic findings. In the first patient, having a history of surgically verified chronic pancreatitis with definite fat necrosis, chronic constipation developed 10 years later with attacks of left upper quadrant pain initiated by bowel movements. Barium enema examination revealed a constricting lesion of the splenic flexure, interpreted as a carcinoma. At operation, the lesion was found to be adherent to the splenic end of the pancreas. Pathologic examination of the resected specimen showed an acute and subacute granulomatous reaction located extrinsically, but producing secondary thickening of the bowel wall with marked narrowing of the lumen. Both the intact mucosa and the outer coats of the bowel were found to contain many inflammatory cells. The second patient presented with obstipation and cramping abdominal pains 5 years after an episode of surgically verified acute hemorrhagic pancreatitis. Barium enema study revealed a constricting lesion at the splenic flexure. At operation, the splenic flexure of the colon was found to be adherent to the tail of the pancreas, and the body and tail of the pancreas were definitely thickened. The base of the constricted segment contained areas of fat necrosis. Histologic sections of the colonic lesion exhibited a chronic inflammatory reaction and fibrous tissue.

Previous observations in the literature regarding the effects of pancreatitis on the small intestine are rare and also largely anecdotal. Compression of the third duodenum by an indurated mesenteric root in acute pancreatitis was noted by Simon and Lerner.[19] It has long been recognized that "sentinel loops" from localized paralytic ileus of the jejunum may accompany pancreatitis.[7] Glenn and Baylin[6] noted jejunal changes characterized by marked persistent spasm of some segments and apparent dilatation of others, with coarsening of the mucosal folds. Functional changes of spasm in the ileocecal region were observed in 1 reported case.

Some of the migratory aspects of pancreatitis have been illustrated by Poppel.[13] In many instances, the inflammatory collections presented at a distance as the initial phase of the disease. Pancreatography was found useful to demonstrate the fistulous communications between the pancreas and these inflammatory collections

Of interest are the vascular lesions described by Rich and Duff.[16] In their series of cases of hemorrhagic pancreatitis they found a peculiar type of vascular necrosis involving the adventitia, media, and internal elastic lamina. The first alterations in the media were always found in the outer layer, the muscle fibers of which appeared necrotic, while those near the intima remained intact. The damage in most cases however, proceded rapidly to involve the entire thickness of the vessel wall with final destruction of the intima.

It was these vascular lesions, which they were able to reproduce experimentally by use of trypsin, that they believed explained the hemorrhage seen in hemorrhagic pancreatitis.

It appears reasonable to assume that these same pancreatic enzymes, liberated out of the pancreatic bed and disseminated along the mesenteric planes described, have similar effects upon the vasculature of the bowel. Microaneurysms, stenoses and occlusions have been described in mesenteric vessels following acute pancreatitis. The fibrotic strictures, either in the small bowel or in the colon, may therefore be on an ischemic basis. Many of the pathologic features in the case of severe jejunal changes included in this report substantiate this possibility, although unfortunately the operative specimen included no mesentery for evaluation. In ischemic colitis, the commonest site involved is the splenic flexure and some cases progress to stricture formation. A similar ischemic mechanism may underlie the fibrotic stenoses of this same segment in pancreatitis. Furthermore, it appears that some of the features of pancreatic malabsorption may be on a vascular basis. Localized anatomic changes of mesenteric vessels from the digestive effects of pancreatic enzymes may result in vascular shunts and ischemic malabsorption.

SUMMARY

1. Pancreatitis extends along the mesenteric planes of the transverse mesocolon to the large intestine and *via* the small bowel mesentery to the jejunum, ileum and cecum.

2. In the large intestine, the transverse colon and particularly the splenic flexure are most commonly involved. The distal ascending colon may be involved by way of a duodenal fistula.

3. In the small bowel, the inflammatory process may selectively involve jejunal loops or may progress down the length of the mesentery to localize in the ileocecal area. Malabsorption may result from the effects of pancreatitis on the small bowel.

4. The precise anatomic pathways and roentgenologic findings are described and illustrated. The lesions produced range from transient spasm to the development of obstructive strictures and remote exudative abscesses.

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ANGIOGRAPHIC EVALUATION OF CYSTIC LESIONS OF THE SPLEEN*

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CYSTIC lesions of the spleen are uncommon. Because they are often difficult to diagnose clinically and because routine radiologic examination cannot always reveal findings which are diagnostic, the benefit of selective abdominal angiography should be better recognized.

Previous reports have reviewed the manifestations of splenic tumors on routine roentgenograms and have emphasized the value of preoperative selective angiography in their diagnosis.[2,13] Preoperative diagnosis of spleen cysts by selective angiography, however, has not been sufficiently promoted in the radiologic literature. Moreover, no description of the angiographic appearance of spleen lymphangioma has been reported.

The purpose of this report is to both demonstrate the distinctive angiographic appearance of spleen cysts and spleen lymphangioma and correlate the latter with findings from pathological specimens and isotope studies.

Our study is of 4 patients with masses of the left upper quadrant in whom selective abdominal angiography was used to confirm the presence of cystic lesion(s) within the spleen. The angiographic appearance in these cases allowed for accurate preoperative diagnosis and was invaluable in guiding subsequent surgical management.

REPORT OF CASES

CASE I. A 22 year old woman was admitted to the hospital with a 4 month history of pain in the left upper quadrant and left shoulder. She had fallen 8 months earlier, landing on top of a chair with her left side. Physical examination revealed a mass in the left upper quadrant of the abdomen; barium enema examination, a study of the upper gastrointestinal tract (Fig. 1), and an excretory urogram showed a mass displacing the stomach and splenic flexure anteriorly, and the left kidney inferiorly. A Tc^{99m} sulfur colloid scintiphotogram revealed a large, sharply outlined, space-occupying lesion in the upper part of the spleen.

Celiac angiography showed depressed splenic and left renal arteries, with intrasplenic arterial branches stretched smoothly about a huge avascular space. Exploratory laparotomy and splenectomy revealed a spleen of 1,166 gm. containing a cyst 12 cm. in diameter. The cyst contained 980 cc. of brownish-green fluid; histologic examination disclosed a "fibrous" false splenic cyst which contained cholesterol deposits and blood. Trauma was considered the most likely etiology.

CASE II. A 22 year old woman developed pain along the left costal margin 8 months after an uneventful influenza-like episode. At the time of the influenza, a chest roentgenogram

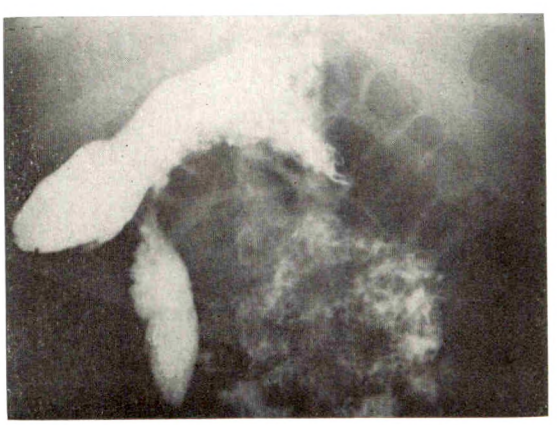


FIG. 1. Case I. Roentgenogram from examination of upper gastrointestinal tract shows a left upper quadrant mass displacing the stomach to the right and the splenic flexure anteriorly and to the right.

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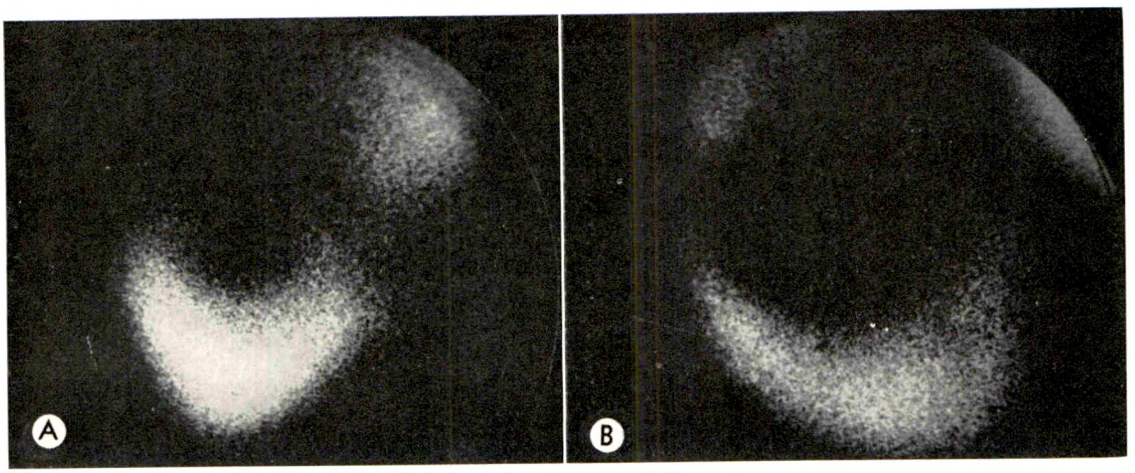


Fig. 2. Case II. (*A*) Left lateral and (*B*) posterior Tc^{99m} sulfur colloid scintiphotograms show a large, smooth unlabeled area within the spleen.

showed normal findings; 3 months later, however, a repeat chest roentgenogram suggested splenomegaly. The patient subsequently experienced pain in the left upper quadrant for the week preceding her menses, at which time she was admitted to the hospital. Physical examination revealed a tender mass in the left upper quadrant of the abdomen. Spleen scintiphotograms showed a smooth, unlabeled area about 10 cm. in diameter, "most likely a spleen cyst" (Fig. 2, *A* and *B*).

Celiac angiography showed a mass of the left upper quadrant displacing the stomach to the right (Fig. 3, *A* and *B*), and splenic arterial branches smoothly draped about a 20×20 cm. avascular mass. Exploratory laparotomy and splenectomy disclosed a 2,950 gm. "cystic spleen" measuring 28×19×12 cm. and con-

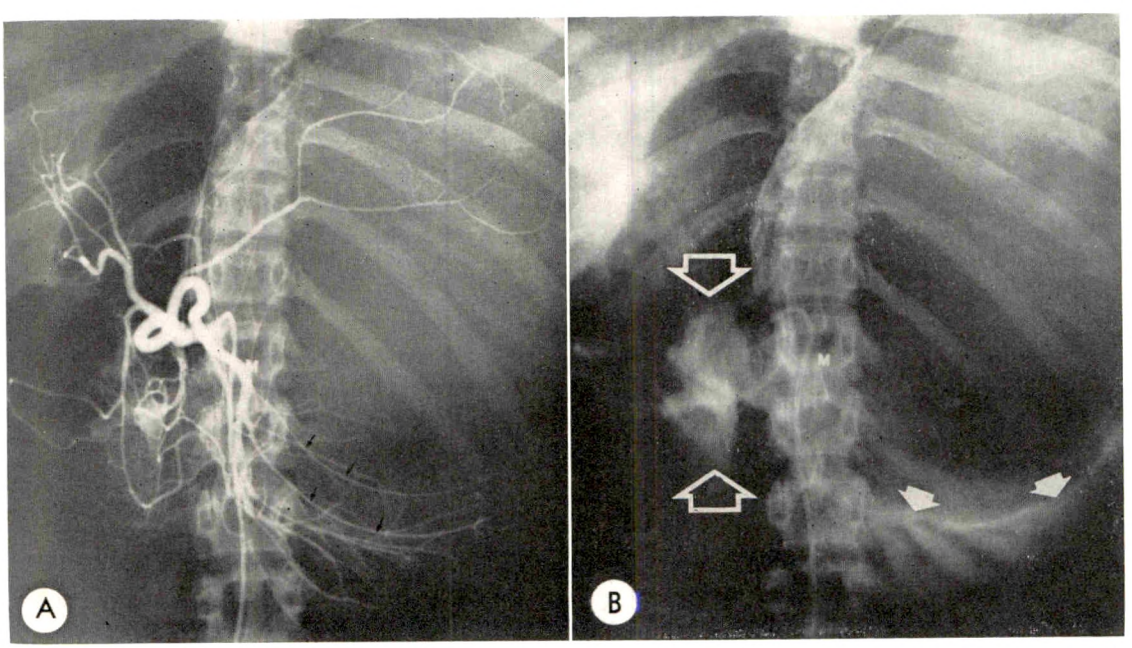


Fig. 3. Case II. (*A*) Arterial phase of selective celiac angiography showing splenic arterial branches (small black arrows) draped smoothly about a huge avascular mass. (*B*) Parenchymogram shows avascular mass with rim of normal spleen tissue inferiorly (solid arrows). Note vertical blush to right of spine (open arrows) believed to represent compressed pancreas (confirmed at surgery).

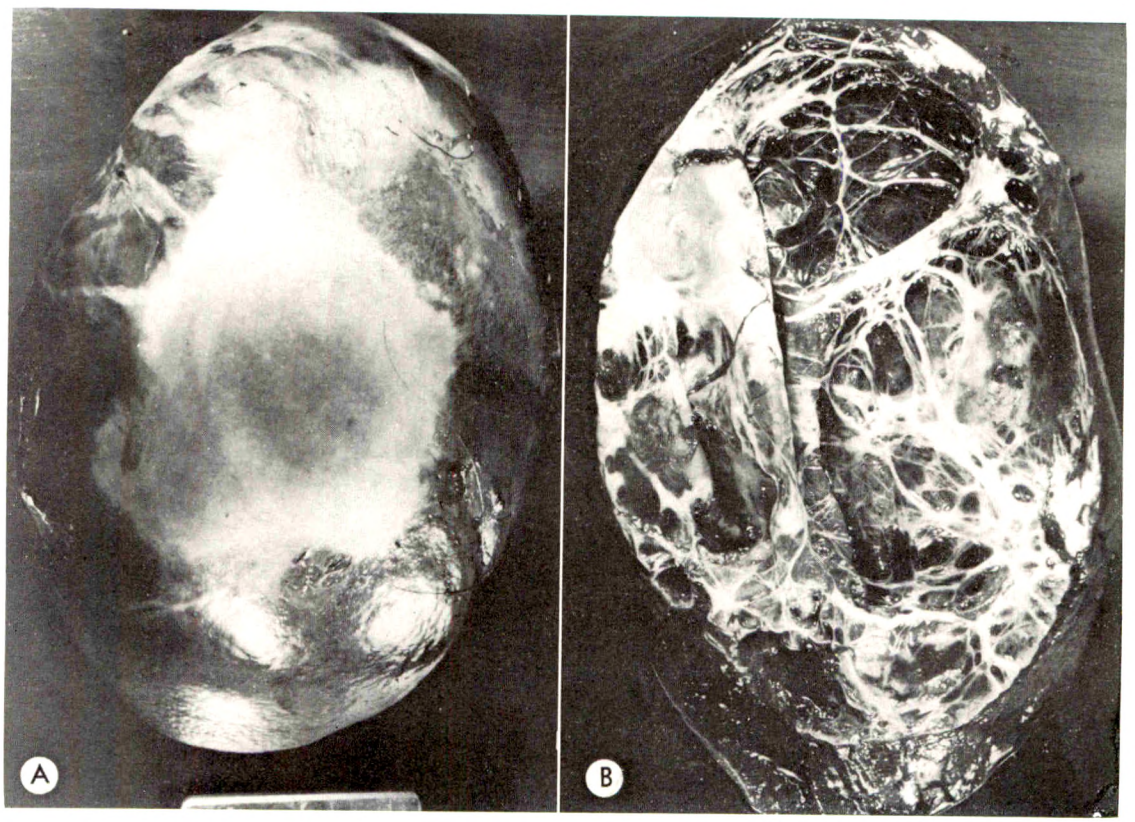


FIG. 4. Case II. (*A*) Gross surgical specimen of "cystic spleen" with rim of parenchyma situated inferiorly. (*B*) Cut specimen demonstrating trabeculated inner contour.

taining 2,300 cc. of brown fluid (Fig. 4, *A* and *B*). The majority of the cystic wall was without epithelial lining and was comprised of dense fibrous tissue with blood and calcium. Trauma was believed to be the etiology of this false cyst.

CASE III. A 21 year old man was admitted to the hospital for treatment of atopic dermatitis. His routine chest roentgenogram suggested a mass in the left upper quadrant of the abdomen (Fig. 5). The patient had sustained trauma to the left side of the abdomen by a baseball 4 years before. An excretory urogram confirmed a mass in the left upper quadrant which was depressing the left kidney (Fig. 6).

Selective angiography revealed splenic arterial branches stretched about 2 avascular masses—one, 12 cm. in diameter, comprising the midportion of the spleen and the other, 4 cm., located superiorly (Fig. 7, *A* and *B*). Exploratory laparotomy and splenectomy disclosed a 1,350 gm. spleen measuring 19 cm. in diameter and containing 1,500 cc. of dark brown fluid. Two adjacent cysts with diameters of 12

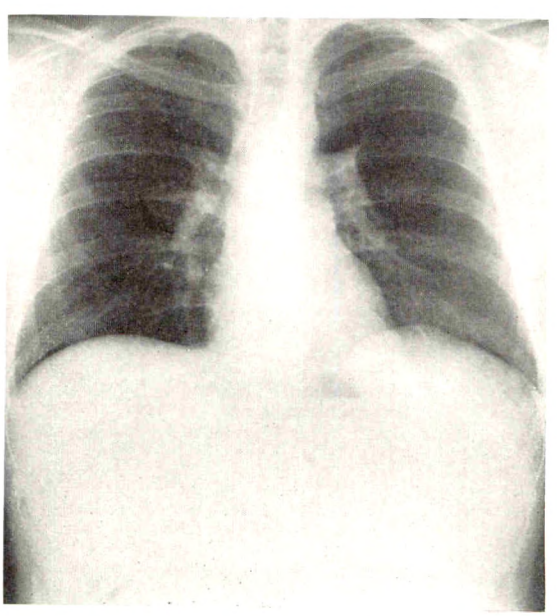


FIG. 5. Case III. Routine chest roentgenogram shows air-filled gastric fundus displaced to the right by (suggestion of) a mass of the left upper quadrant.

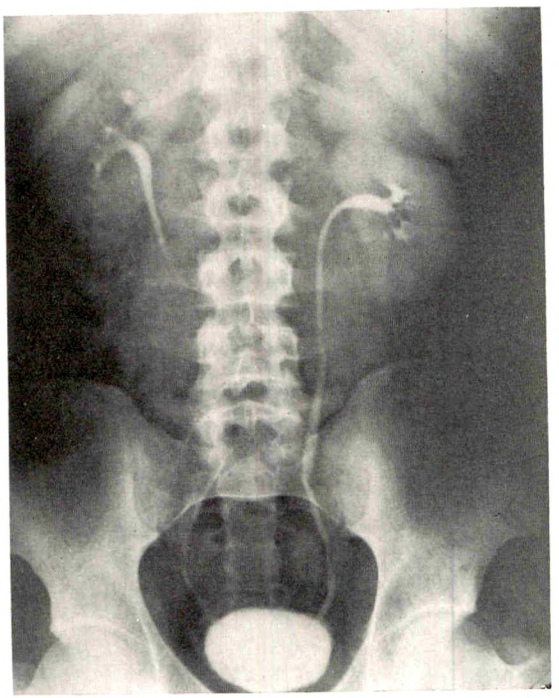


Fig. 6. Case III. Excretory urogram shows left kidney depressed by a mass in the left upper quadrant—probably the spleen.

cm. and 4 cm. were seen (Fig. 8, *A* and *B*). Histologic examination revealed a fibrous wall without a true epithelial lining. Previous trauma was believed to be the cause of this false cyst.

Case IV. A 21 year old man was admitted to the hospital for splenectomy because of prolonged splenomegaly of unknown etiology. Five years before the patient was seen in a clinic because of painless neck swelling of 3 weeks' duration. A chest roentgenogram showed calcifications in the neck mass (Fig. 9) and there was splenomegaly. Hematologic study showed thalassemia minor. An esophagogram, bone survey, and excretory urogram yielded normal findings. A biopsy specimen of the neck lymph nodes showed cystic spaces filled with blood and focal calcification; otherwise the lymph nodes were normal.

Four years later, the neck mass had grown to 3 cm. × 4 cm. and the spleen had quadrupled in size. The neck mass was removed and showed endothelial-lined, blood-containing, vascular spaces; the diagnosis was cavernous hemangioma. Prophylactic splenectomy was advised, and Tc^{99m} sulfur colloid scintiphotograms of the liver and spleen (Fig. 10, *A* and *B*) showed normal liver, but an enlarged spleen with multiple areas of decreased labeling, a "swiss-cheese" appearance. Because Tc^{99m} pertechnetate scintiphotograms of the spleen showed decreased perfusion, a hemangioma was excluded and multiple, benign or infected cysts were suggested as the diagnosis.

Celiac angiography demonstrated splaying of the splenic arterial branches within the parenchyma (Fig. 11, *A–D*). No pathologic

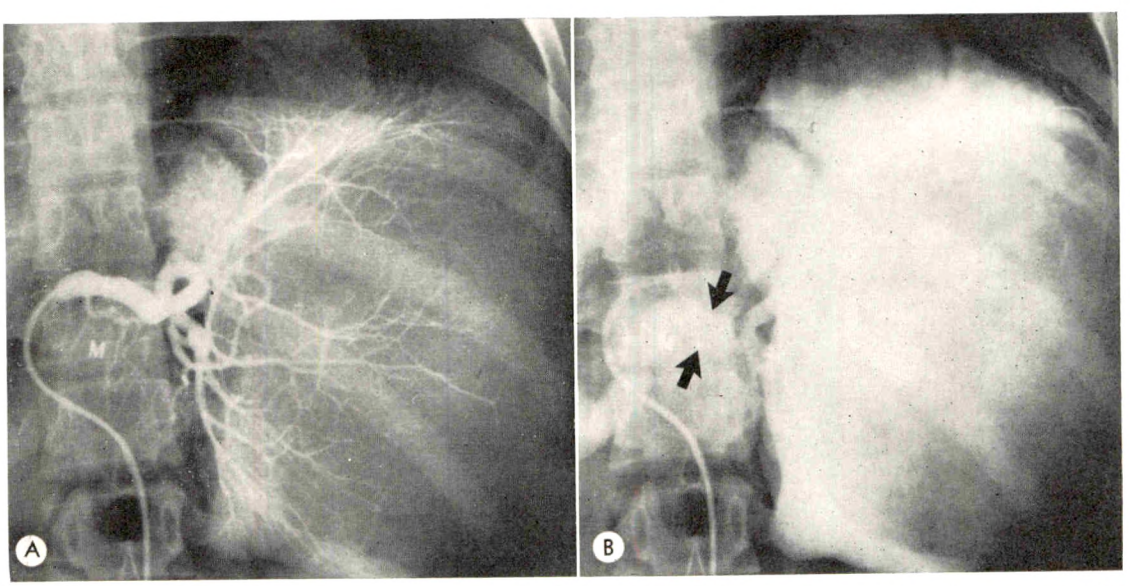


Fig. 7. Case III. (*A*) Selective splenic angiogram shows arterial branches stretched about a large mass in the midportion of the spleen and a second, smaller mass at the superior pole of the spleen. (*B*) Parenchymogram shows both avascular masses with adjacent splenic parenchyma. Note patent splenic vein (arrows).

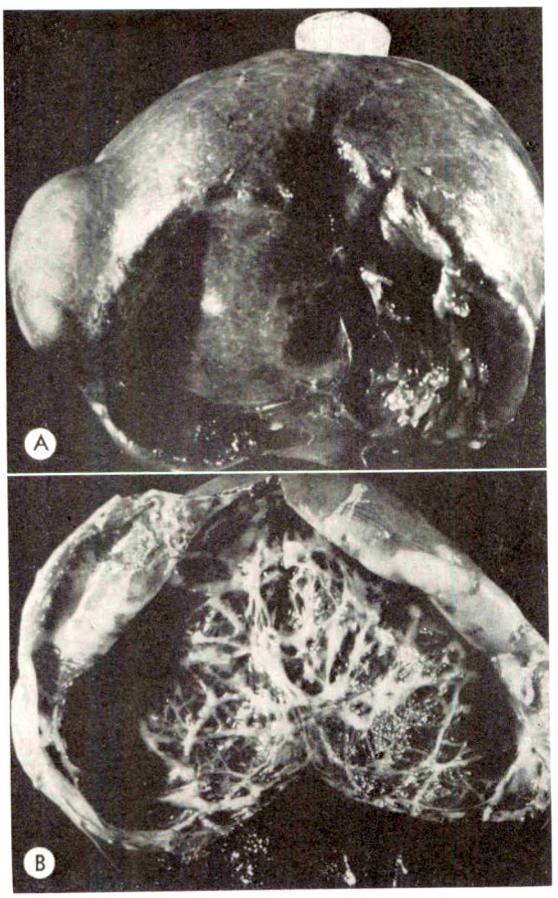


FIG. 8. Case III. (*A*) Gross and (*B*) cut specimens show large cyst with trabeculated inner contour and smaller cyst superiorly.

vessels were seen, but multiple, variable, small lucencies were noted in the parenchymogram. Splenectomy revealed a 25×20×7 cm., 1,600 gram spleen which had cystic areas on a red-purple surface (Fig. 12, *A* and *B*). Histologic examination disclosed small areas of splenic tissue amid dilated spaces containing eosinophilic material and sparce red cells—a lymphangioma (Fig. 12*C*).

DISCUSSION

Of the 4 patients in this study, 3 had masses of the left upper quadrant which were of unknown etiology; 2 of these 3 patients were women whose initial complaint was pain, and the other patient was a man whose routine chest roentgenogram suggested splenomegaly. The fourth patient had clinical, massive splenomegaly believed to be out of proportion to his hematologic disease.

Routine radiologic examinations were nondiagnostic for all 4 patients; isotope studies done on 3 of the 4 patients showed abnormal findings; selective abdominal angiography in each case suggested the presence of cystic lesion(s) within the spleen, and proved to be the best examination for diagnostic purposes.

The distinctive angiographic features in the first 3 cases of spleen cyst are demonstrated in both the arterial phase and in the parenchymogram. In the early arterial phase, intrasplenic arterial branches are smoothly compressed and stretched about the periphery of a relatively ovoid, avascular area within the enlarged spleen. In the parenchymogram, the avascular area is clearly seen, sharply outlined by the surrounding, smooth, splenic parenchyma. Splenic arterial caliber was normal and no pathologic or irregular vessels were seen; the splenic vein was normal in each case.

The angiographic appearance of the fourth case was especially unusual in that the arterial phase showed smaller arterial branches stretched and splayed haphazardly throughout the parenchyma; no pathologic vessels were seen, however. The parenchymogram showed multiple lucencies, ranging from millimeters to several centimeters in diameter and resembling

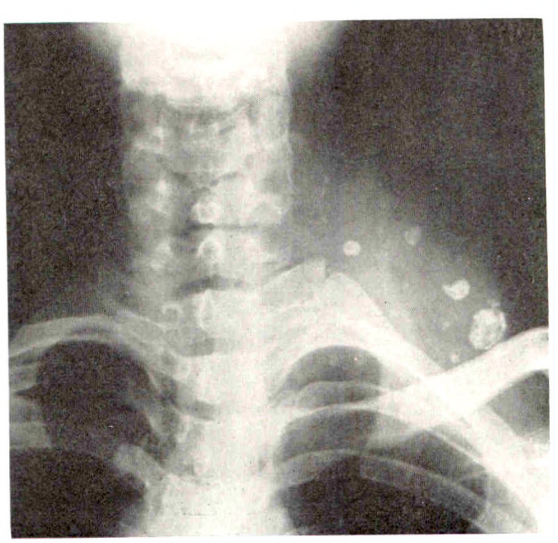


FIG. 9. Case IV. Close-up view (from chest roentgenogram) of calcifications in left neck mass.

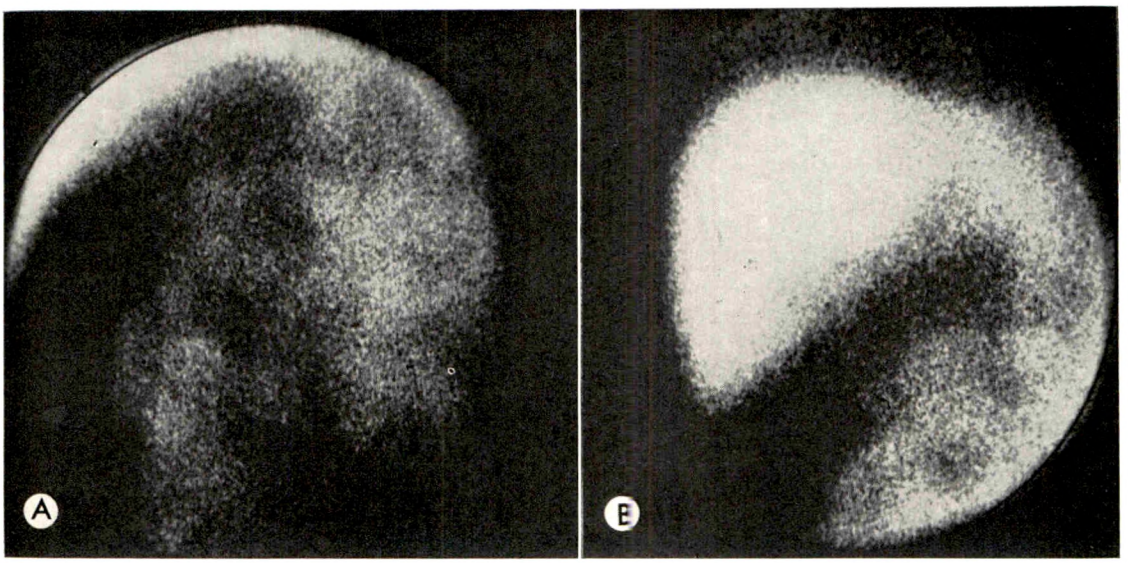


Fig. 10. Case IV. (*A*) Left anterior and (*B*) midline anterior Tc^{99m} sulfur colloid scintiphotograms show normal liver but enlarged spleen with multiple areas of decreased labeling (producing a swiss-cheese appearance).

the swiss-cheese appearance which had been depicted on the isotope study. This appearance strongly suggested the presence of multiple cysts within the spleen. At splenectomy this spleen was, in fact, found to be almost entirely replaced by a lymphangioma comprised of multiple fluid-filled cysts.

In evaluating the presence of cystic lesion(s) seen angiographically within the spleen, several pathologic processes must be considered in the differential diagnosis. Cysts of the spleen have been classified by Fowler[5] as parasitic and nonparasitic in origin. *Echinococcus* is the only parasite that produces splenic cysts; in addition, it is the most common world cause of splenic cysts.[8] Rizk and his co-workers[12] recently described a rim of increased density seen around the cyst wall during the capillary phase of celiac angiography, and they state that it is suggestive of *Echinococcus* disease. Nonparasitic cysts of the spleen are either true cysts (which contain an epithelial lining) or false cysts (which have no cellular lining). True cysts are believed to be congenital in origin; false cysts, on the other hand, are acquired (frequently secondary to trauma or infection) and may calcify. They account for approximately 80 per cent of nonparasitic splenic cysts. Bron and Hoffman[2] state that no difference between true and false splenic cysts is apparent angiographically.

Neoplastic diseases may also angiographically resemble cystic lesion(s) within the spleen. Rare, benign fibromas, osteomas, and chondromas may displace arterial branches and appear to be relatively avascular in the parenchymogram.[8] The more common benign hamartomas and hemangiomas, however, are usually richly vascular with large, plexoid vessels resembling the neovascularity of malignant tumors.[17] Although primary nonlymphomatous malignancies have not commonly been studied angiographically, Edsman[4] describes the angiographic appearance of a splenic endothelioma in which there was distortion and occlusion of smaller vessels, as well as neovascularity. Kupic *et al.*[8] state that metastatic malignancy usually shows up as small relatively avascular areas without tumor vessels; Gould and associates[6] and Rösch[13] have separately reported amputated vessels and tumor neovascularity as positive evidence of metastatic disease. Infiltrative diseases, such as

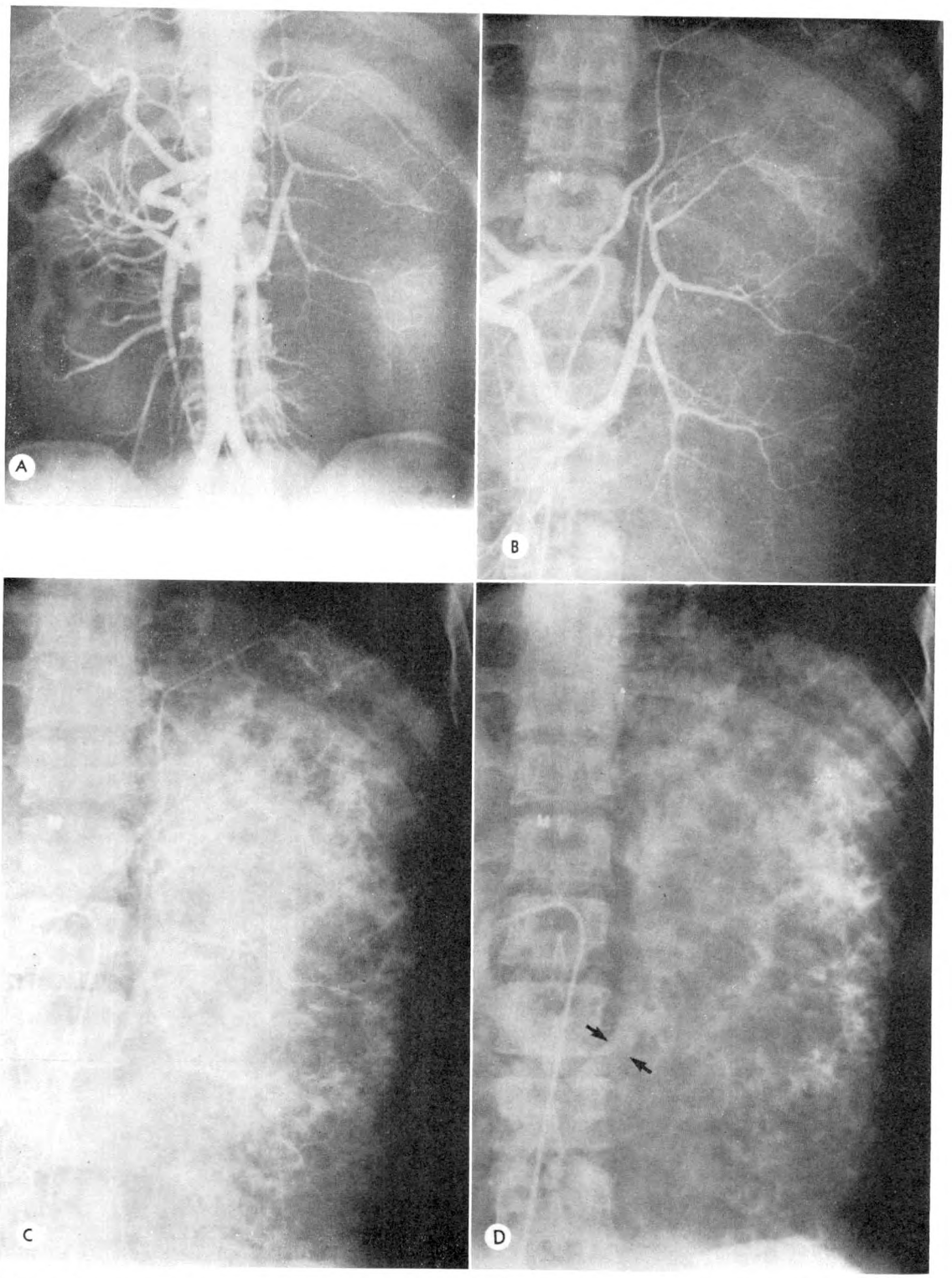


FIG. 11. Case IV. (*A*) Aortogram demonstrates enlarged spleen with straightened and separated intrasplenic arteries. (*B*) Selective splenic angiogram with good delineation of intrasplenic arterial distribution (early arterial phase). (*C*) Splenic angiogram, late arterial phase, of same patient shows multiple parenchymal lucencies superimposed on intrasplenic arteries. (*D*) Parenchymogram demonstrates swiss-cheese appearance. Note patent splenic vein (arrows).



FIG. 12. Case IV. (*A*) Gross surgical specimen demonstrates enlarged spleen with multiple cystic areas on surface. (*B*) Cut specimen shows multiple, variable, small cysts within the parenchyma. (*C*) Microscopic, low-power view of histologic specimen shows multiple, dilated spaces containing eosinophilic material with small areas of spleen tissue (arrows) in the surrounding area.

leukemia, lymphoma, and the reticuloses, may narrow and stretch intrasplenic vessels so as to present a relatively avascular appearance.[8,17] This appearance may also be seen with multiple abscesses or infarcts.[14] Nonetheless, these diseases which present an avascular appearance are rarely a diagnostic problem when consideration is given to history, clinical findings, and roentgenographic evidence of involvement of other systems.

No prior description of the angiographic appearance of a lymphangioma of the spleen has been found in the literature. Of approximately 34 cases reported in the world's literature, the diagnosis was made at autopsy in 11 and after splenectomy in at least 20.[1,3,5,7,9–11,15,16] The tumor has been reported to occur in all age groups, and details available in 15 of the 27 cases reported by Fowler[5] reveal that 11 were women and 4 were men. Lymphangioma of the spleen is a multicystic, benign tumor that contains dilated lymphatic sinuses lined by endothelial cells. Its angiographic appearance is unique and should allow for accurate preoperative diagnosis.

SUMMARY

When a mass of the left upper quadrant is suspected on clinical or routine roentgenographic examination, and a space-occupying lesion of the spleen is seen on isotope study, selective abdominal angiography should be the preoperative diagnostic method of choice.

The stretching and compression of vessels, seen in the arterial phase, and avascular zone(s) seen in the parenchymogram, are characteristic findings for the cystic lesions in the 4 cases presented.

The angiographic appearance of lymphangioma of the spleen is reported here for the first time; it is unique and should allow for accurate preoperative diagnosis.

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Vol. 119, No. 1

SPLENIC ABSCESS AND THE GENE FOR HEMOGLOBIN S*

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SPLENIC abscesses are either primary or secondary. Those secondary to a focus of infection elsewhere in the body carry a high mortality and are rarely diagnosed ante mortem.[30,33] Most of the cases reported in the literature from the temperate regions of the world are of the secondary type. Cases of primary splenic abscess, also called "tropical splenic abscess" were once thought to occur only in Africans,[2] but cases have now been reported throughout the world.[22] However, they do appear to be more common in tropical areas; their etiology is uncertain.

We have recently reviewed 22 cases of splenic abscess seen in Ibadan, Nigeria; 20 were of the primary type and 2 were secondary. Hemoglobin electrophoresis was carried out in 16 of these cases (15 primary abscesses and 1 secondary abscess) and roentgenographic studies were available in all cases.

The purpose of this communication is to report our findings of an association between hemoglobin genotypes and roentgenographic appearances in Nigerian patients with primary splenic abscesses. The etiology of this condition and its relationship to the gene for hemoglobin S (Hb S) are discussed. Clinical features, bacteriology and treatment are also briefly discussed.

MATERIAL AND METHOD

Thirty-eight cases of splenic abscess in Nigerians were coded by the Medical Records Department of the University College Hospital (U.C.H.), Ibadan, in the 9 year period, 1963 to 1971. In 22 of these cases, complete medical records, including the surgical and/or autopsy findings, as well as plain abdominal roentgenograms and an upright chest roentgenogram were available. Special roentgenologic examinations were also done in some cases. These 22 cases are reviewed.

Hemoglobin genotypes were determined by electrophoresis in 16 cases. Family studies were not carried out. It is remotely possible that in an occasional case the hemoglobin genotype will be misinterpreted when it is determined by electrophoresis alone. This can occur if there has been a recent prior blood transfusion, in some patients with genes for hemoglobin S and thalassemia, and when other hemoglobins are present which have an electrophoretic mobility similar to Hb S, such as Hb D.[3] It is unlikely that these factors have introduced any significant errors into the present study.

RESULTS

AGE AND SEX

(Figure 1 and Table 1)

The ages of the patients ranged from 6 months to 70 years; 18 of the 22 cases were in the first 40 years of life. There were 18 males and 4 females.

PRESENTING SYMPTOMS AND CLINICAL FINDINGS

(Table 1)

The duration of symptoms ranged from 3 days to 1 year; it was less than 2 weeks in 12 patients and less than 4 weeks in 18 patients (82 per cent).

The most common presenting symptom was pain located to the left of the midline in the hypochondrium. This pain was sharp or dull in character, often radiating to the left costovertebral region, the left chest or the left shoulder. In 14 cases, the

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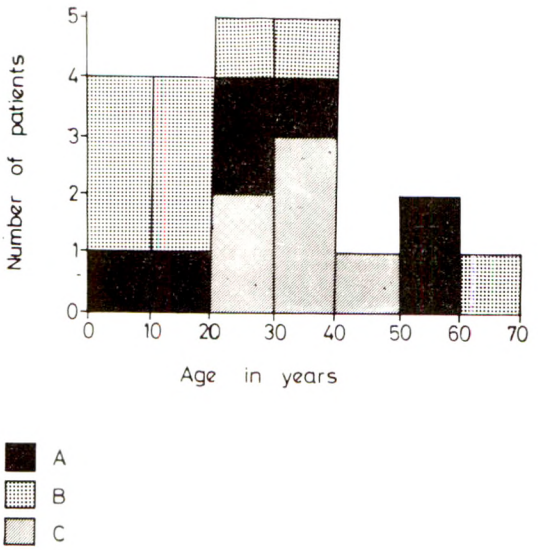


FIG. 1. Age distribution and roentgenologic types of abscesses present in the 22 patients in this study.

pain was aggravated by coughing and was often associated with a high, unremitting fever. Three patients presented with severe chest pain localized to the left side; 1 patient had left shoulder pain; and 3 patients presented with vomiting, diarrhea and fever.

In 16 patients, the spleen was enlarged and tender to palpation. One revealed a succussion splash and fluid thrill (Case 22), and 2 were tympanic to percussion in the left upper abdomen (Cases 2 and 21). Five patients were tender over the abdomen and had other signs of peritonitis. Two had physical signs of a left pleural effusion (Cases 8 and 18); in 1 of these patients, the abscess had ruptured into the pleural cavity (Case 18). Anchovy-sauce-like, brownish-red fluid was obtained from diagnostic splenic aspiration in 5 patients prior to surgery.

HEMATOLOGIC FINDINGS

(Table 1)

Fifteen patients had polymorphonuclear leukocytosis; 6 had normal white blood cell counts; the result of the white blood cell count was not available in 1 case.

Hemoglobin electrophoresis in the 22 patients showed 6 to have hemoglobins S and C (Hb SC), 6 to have hemoglobins A and S (Hb AS), 3 had only hemoglobin A (Hb AA), and 1 was Hb AC. No patient had only hemoglobin S (Hb SS—homozygous sickle cell anemia). Hemoglobin electrophoresis was not carried out in the remaining 6 patients.

The normally observed hemoglobin genotype frequencies in Ibadan vary slightly, depending on the age of the population sampled and other factors. The figures commonly quoted are: Hb AA, 71–79 per cent; Hb AS, 20–27 per cent; Hb SS, 0.1–1.7 per cent; Hb SC, 0.5–0.8 per cent; and Hb S Thalassemia, 0.1 per cent.

BACTERIOLOGIC FINDINGS

(Table 1)

Eleven patients had a sterile culture; in 4 patients, cultures were not done or reports of cultures were not available. Two Hb SC patients grew *Salmonella* species; 3 patients, 2 of whom were Hb SC, grew *Staphylococci pyogenes;* 1 patient (Hb AS) grew *Pseudomonas pyocyanea* and another (Hb AA) grew nonhemolytic *Streptococci.*

ROENTGENOLOGIC FINDINGS

All patients had a chest roentgenogram and at least 1 or 2 plain abdominal roentgenograms (supine and/or erect). Except for 1 case that showed no abnormality (Case 16), the plain film findings could be divided into 3 arbitrary, but distinct groups (Table II). Some roentgenographic findings, which simply indicated the presence of a left hypochondrial mass, were common to all 3 groups. These findings include: elevation of the left hemidiaphragm (Fig. 4–7); displacement of the mediastinum to the right (Fig. 6); displacement of the gaseous stomach shadow to the right (Fig. 3; 4; and 7); and downward displacement of the splenic colon (Fig. 3–5).

Type A: (Mass Type)—7 Cases. This group showed only the roentgenographic findings common to all 3 groups; a soft tissue mass in the left upper quadrant of the abdomen (Fig. 2; and 3). Left lower

TABLE I

SUMMARY OF CASES

Case No.	Age (yr.)	Sex	Length of History	Clinical Features			Hematology			Roentgenologic Group	Findings at Surgery or Autopsy	Bacteriology of Aspirate
				Presenting Symptoms	Temperature	Physical Signs	PCV (per cent)	WBC (cu mm.)	Hb Genotype			
1	25	F	2 months	Lower abdominal pain, vomiting, diarrhea and fever	103° F	Tender left hypochondrium; jaundice; spleen 2 cm. enlarged and tender	23	26,800	AA	C	Massive peritonitis; intrasplenic abscess 5 cm. in diameter; multiple hepatic abscesses with portal vein thrombosis. *Died*	No report
2	5	M	3 weeks	Abdominal swelling	103.4° F	Spleen 12 cm. enlarged, tympanic, soft and tender; left lower lobe crepitation	21	13,000	SC	B	370 ml. of anchovy-sauce intrasplenic pus found	*Staphylococcus pyogenes*
3	32	F	2 weeks	Known case of nephrotic syndrome with fever, vomiting and diarrhea	100.5° F	Puffy face; edema of legs, ascites; enlarged liver 6 cm.	21	13,000	AC	B	Fulminating pyelonephritis; pyemic abscesses in enlarged liver and spleen; amyloidosis of liver, spleen and kidneys. *Died*	No report
4	21	M	9 days	Abdominal pain, anorexia and fever	103° F	Left lower lobe crepitations; spleen 6 cm. enlarged and tender	35	19,200	SC	B	Spleen was 202 gm.; hemorrhage lying free in a thick-walled abscess cavity; duodenal and gastric ulcers	*Salmonella*
5	70	M	2 weeks	Abdominal pain, cough and fever		Spleen 8 cm. enlarged and tender	22	23,000	SC	B	300 ml. of anchovy-sauce fluid replacing the spleen; had osteomyelitis of both tibiae and avascular necrosis of left femoral head	Sterile
6	40	M	3 days	Left shoulder pain, constipation and fever	102° F	Left lower lobe crepitations; tender all over abdomen	31	14,600	AS	C	1.5 liters of hemoperitoneum and pus	Sterile
7	54	M	2 weeks	Swelling of left flank and fever		Spleen enlarged and tender		6,600	AS	A	Intrasplenic abscess adherent to splenic colon	Sterile

PCV = packed cell volume; WBC = white blood cell count; Hb = hemoglobin.

(*Continued on following page*)

TABLE I—(*Continued*)

Case No.	Age (yr.)	Sex	Length of History	Clinical Features			Hematology			Roentgenologic Group	Findings at Surgery or Autopsy	Bacteriology of Aspirate
				Presenting Symptoms	Temperature	Physical Signs	PCV (per cent)	WBC (cu mm.)	Hb Genotype			
8	20	M	4 months	Left hypochondrial swelling and fever	101° F	Spleen 12 cm. enlarged and tender; left lower lobe crepitations and left pleural effusion	28	8,500	SC	B	Intrasplenic abscess with adhesions between spleen, liver, diaphragm, stomach, colon and pancreas	*Staphylococcus pyogenes*
9	15	M	3 days	Abdominal pain and fever	102° F	Spleen 8 cm. enlarged, soft and tender	32	9,350	AS	A	Intrasplenic abscess	Sterile
10	12	F	1 week	Fever, cough and pain in left chest	105° F	Spleen 10 cm. enlarged and tender; left ribs immobilized; left lower lobe crepitations	19	12,100	SC	B	Anchovy-sauce intrasplenic abscess; recurred 3 months after treatment by aspiration	*Salmonella*
11	26	M	3 weeks	Fever and left hypochondrial pain	102.8°F	Spleen 8 cm. enlarged and tender; slight jaundice; crepitations all over left lung	21	15,800	AS	A	Intrasplenic abscess	Sterile
12	33	M	1 week	Left upper abdominal pain radiating to the scrotum	100° F	Tender 8 cm. mass in left hypochondrium	23	7,100	Not done	A (splenic calcification)	Huge intrasplenic abscess	Sterile
13	60	M	2 weeks	Left hypochondrial pain and fever	101° F	Spleen 7 cm. enlarged and tender	29	8,900	AS	A	Dense adhesions between splenic abscess and stomach	*Staphylococcus pyogenes*
14	½	M	1 week	Fever, diarrhea and vomiting	101° F	Tender mass on the left	17	39,000	AS	A	Splenic abscess arising from lower pole and filling the peritoneal cavity, especially the left; cerebral vein thrombosis and lung abscess. *Died*	*Pseudomonas pyocyanea*

TABLE I—(*Continued*)

Case No.	Age (yr.)	Sex	Length of History	Clinical Features			Hematology			Roentgenologic Group	Findings at Surgery or Autopsy	Bacteriology of Aspirate
				Presenting Symptoms	Temperature	Physical Signs	PCV (per cent)	WBC (cu mm.)	Hb Genotype			
15	22	F	1 month	Fever, loss of appetite and left abdominal mass	103° F	Spleen 14 cm. enlarged and tender	16	6,400	AA	A	Intrasplenic abscess; 400 ml. of pus aspirated	Sterile
16	40	M	1 month	Epigastric pain and fever	100° F	Tender all over upper abdomen	25	12,300	Not done	Normal	Splenic abscess, plus dense adhesions between it and contiguous viscera	No report
17	40	M	5 days	Chest pain and cough	104° F	Tender all over abdomen	23	17,100	Not done	C	Subacute bacterial endocarditis involving aortic, mitral and tricuspid valves, splenic abscess. *Died*	No report
18	32	M	(a) 1 year (b) 6 months (c) 2 weeks	(a) Left-sided abdominal pain (b) Diarrhea (c) Cough and leg swelling	104° F	(c) Left pleural effusion	Results missing	Results missing	AA	C	Subdiaphragmatic abscess replacing spleen; small abscess in liver Bilateral pleural effusion; abscess ruptured into pleura and pericardium. *Died*	Nonhemolytic *Streptococcus*
19	26	M	1 month	Left hypochondrial pain and fever	104° F	Tender and rigidity in abdomen	24	28,000	Not done	C	Splenic abscess ruptured into the mediastinum *via* a bronchoperitoneal fistula	Sterile
20	45	M	6 months	Left hypochondrial pain	102° F	Tenderness all over abdomen	29	17,600	Not done	C	Abscess in left hypochondrium replacing spleen, and peritonitis	Sterile
21	8½	M	2 weeks	Pain in left chest and hypochondrium	105° F	Crepitations in left lower lobe; tympanic and tender left hypochondrium	18	19,400	Not done	B	Large abscess cavity replacing the spleen	Sterile
22	12	M	1 week	Fever and left-sided chest pain	103° F	Succussion splash over tender left hypochondrium; left lower lobe crepitations	24	21,700	SC	B	Splenic abscess	Sterile

TABLE II

CLASSIFICATION ACCORDING TO ROENTGENOLOGIC TYPE

Roentgenologic Type	Total No.	Hemoglobin Genotype	Average Duration	Average White Blood Cell Count	Average Temperature	Organisms	No. of Patients Who Died
A	7	5 AS 1 AA 1 Unknown	2 weeks	11,890	101.4° F	1 *Pseudomonas pyocyanea* 1 *Staphylococcus pyogenes*	1
B	8	6 SC 1 AC (secondary abscess) 1 Unknown	3–4 weeks	16,300	103.6° F	2 *Salmonella* 2 *Staphylococcus pyogenes*	1
C	6	2 AA 1 AS 3 Unknown (one is secondary abscess)	15 weeks	20,800	103.1° F	1 *Nonhemolytic Streptococcus*	3
Normal	1	Unknown	1 month	12,300	100° F	Nil	Nil

lobe consolidation and/or a left pleural effusion were commonly present in this group. One case (Case 12; Fig. 3) had a thin hemispherical calcification around the lower pole of the mass (spleen) and multiple calcifications within its central area.

Type B: (Pyogaseous Type)—8 Cases. In addition to the presence of a left upper quadrant mass, this group showed a collection of gas within the mass. In 2 patients (Cases 8 and 21; Fig. 4 and 5), the gas appeared as irregular bubbles of varying sizes, causing a speckled pattern overlying the splenic bed. On erect roentgenograms, an air fluid level was commonly seen in the left upper abdomen (Fig. 6; and 7*B*). Two air-fluid levels were present in some cases, 1 gastric and 1 splenic (Fig. 7).

Type C: (Inflammatory Type)—6 Cases. In these cases, there were dilated loops of jejunum and splenic colon in the left upper quadrant. On erect roentgenograms, the loops showed air fluid levels. More generalized bowel distention, with fluid levels and ascites, was present in some cases. The properitoneal fat line was not seen, and the left flank was bulging in most of these patients. Left lower lobe consolidation and/or a left pleural effusion were commonly present. No definite intrasplenic gas was seen in these patients.

Special Roentgenologic Studies.

Barium Meal: In 4 cases (all Type B), this was done to confirm that the left

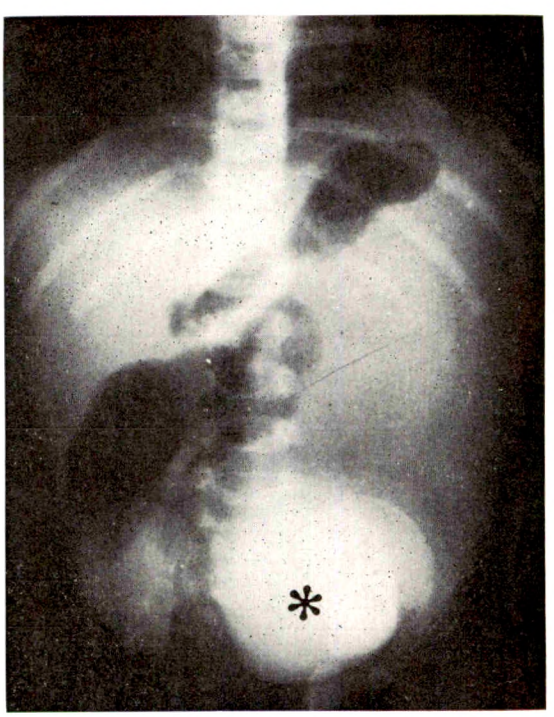


FIG. 2. Case 14. *Type A* splenic abscess in a 6 month old Hb AS patient.

The roentgenographic findings indicate only a left-sided abdominal mass. The dense rounded opacity (*) overlying the pelvis is an umbilical hernia.

upper quadrant air and/or fluid level was in an abscess cavity rather than in the stomach (Fig. 4*B*; and 7*B*). The stomach was characteristically displaced to the right (Fig. 4*B*).

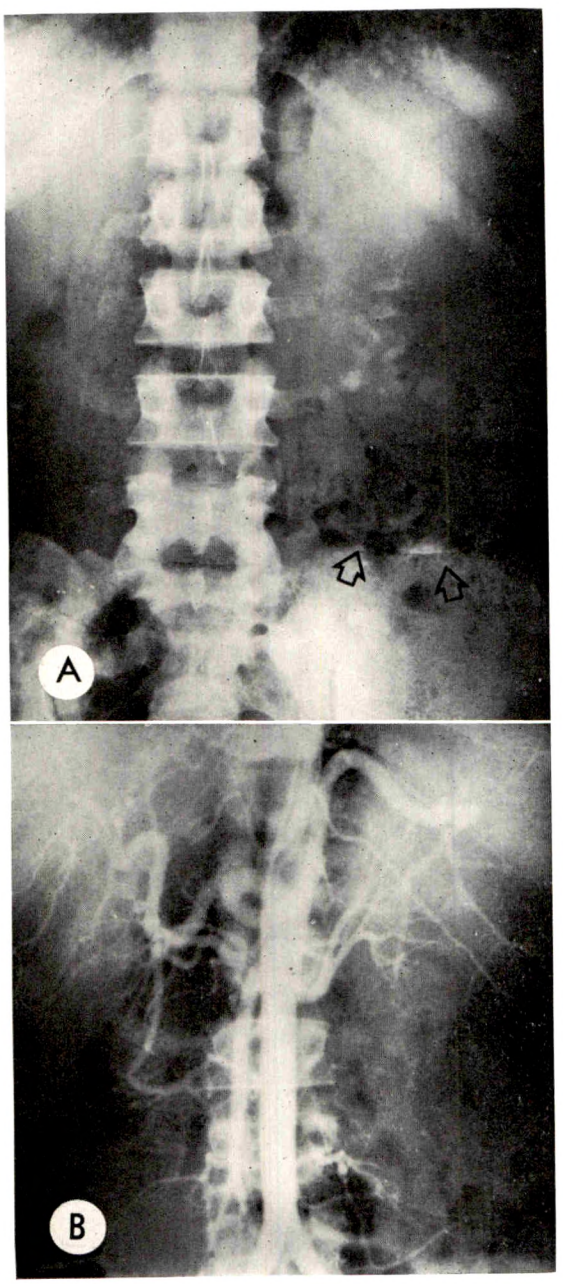


Fig. 3. Case 12. *Type A* splenic abscess in a 33 year old man of unknown hemoglobin genotype.

(*A*) The left upper abdominal mass has calcification within it and marginal calcification at the lower edge (arrows). (*B*) An aortogram demonstrates patency of the splenic artery.

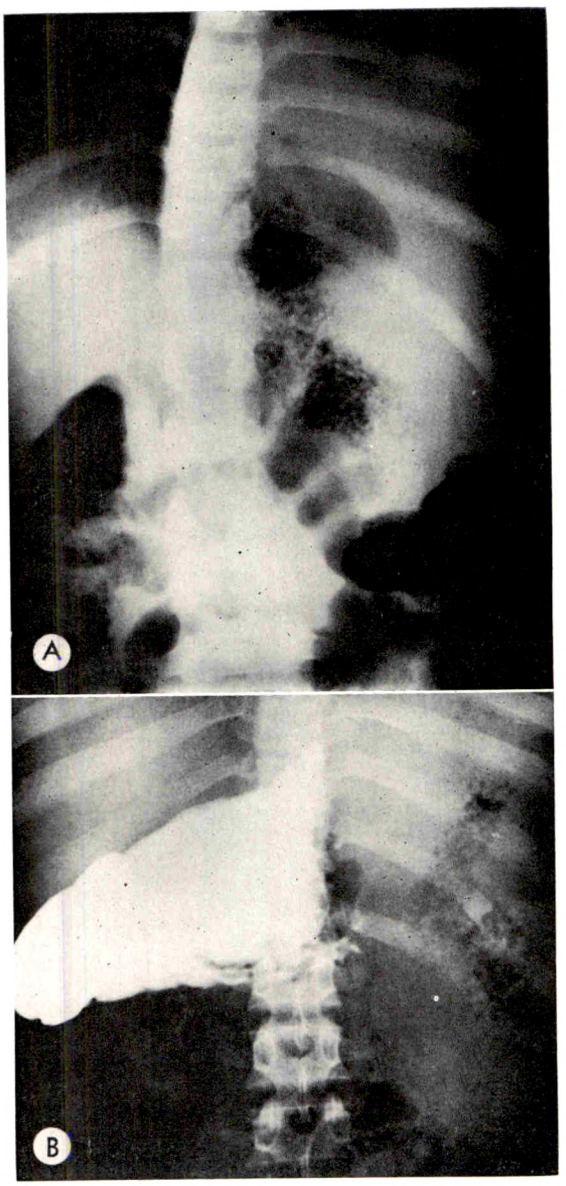


Fig. 4. Case 21. *Type B* splenic abscess in an 8½ year old boy with Hb SC disease.

(*A*) Plain roentgenogram of the abdomen. The left hemidiaphragm is elevated and there is a left upper quadrant mass. The splenic flexure of the colon is depressed. An air-fluid level is present and there are also bubbly gas shadows on the left. (*B*) Barium in the stomach shows that it is displaced to the right and that the bubbly gas shadows are not within it.

Pyogram: In 4 cases, a microsuspen-

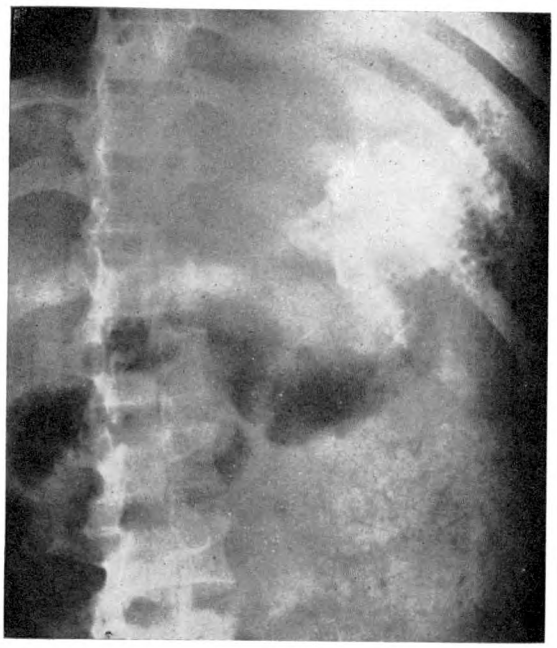


FIG. 5. Case 8. *Type B* splenic abscess in a 20 year old man with Hb SC disease. Sterile contrast material was injected into the abscess at the time of aspiration.

The left hemidiaphragm is elevated and the fecal-filled splenic flexure is depressed by the enlarged spleen. There is a bubbly gas pattern in the splenic region.

sion of sterile barium sulphate (Steripaque*) was injected directly into the abscess cavity to assess its size and location (Fig. 5).

Intravenous Pyelography: This was sometimes necessary to distinguish a splenic mass from a renal mass.

Angiography: In 2 cases (Cases 8 and 12), aortographic studies were done. In both cases, the splenic arteries were patent and appeared of normal caliber (Fig. 3*B*). The abscess cavities were avascular; perisplenic vessels stretched around the spleen demonstrated its increased size. The absence of a persistent splenogram indicated patency of the splenic veins.

DISCUSSION

Secondary splenic abscesses are usually from 1 of 3 sources: metastatic (75 per cent); post-traumatic (15 per cent); or contiguous infection, often from gastric or colonic lesions (10 per cent).[30] The embolic or metastatic infections which led to secondary splenic abscesses are usually carried to the spleen *via* the portal system from intra-abdominal suppurative diseases. The infection may also be conveyed to the spleen through the arterial system from distant infective foci. Only 2 cases in this report are of the secondary or metastatic type: 1 patient had subacute bacterial endocarditis (Case 17) and the other had fulminating pyelonephritis as the primary focus of infection (Case 3). The splenic abscess in one other patient who also had multiple hepatic abscesses and portal vein thrombosis (Case 1) may have been a secondary abscess.

Almost all known pathogenic bacterial organisms, with the exception of *Gonococcus*, have been found in splenic abscesses.[14] They may also be secondary to amebiasis, but this is rare relative to the common occurrence of liver abscess in that disease.[13]

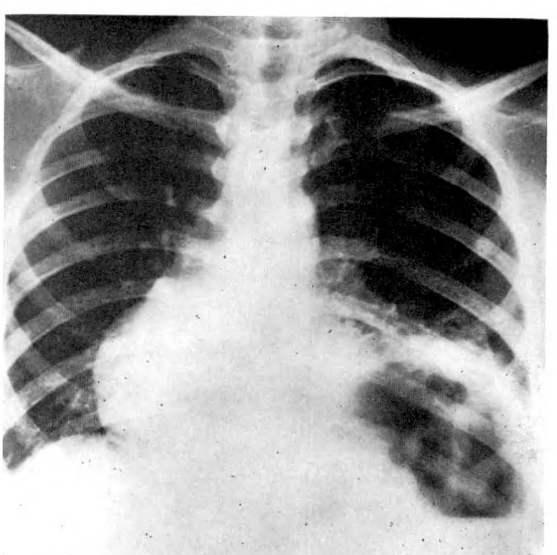


FIG. 6. Case 22. *Type B* splenic abscess in a 12 year old boy with Hb SC disease.

The left hemidiaphragm is elevated and the mediastinum is displaced to the right by the large gas-containing abscess cavity with an air-fluid level. The fluid level is further to the left than one normally sees the gastric air bubble.

* Steripaque. Darmancy and Co. Ltd., Ware, Herts, England.

The etiology of the primary type of splenic abscess is uncertain. Twenty of the 22 cases in this series are the primary type. The first case reported of primary splenic abscess[40] and other early cases were all from East Africa.[2,15,16] Although the disease entity has been called "tropical splenic abscess" and appears to be more common in tropical areas of the world, cases have been reported throughout the world.[22]

Jelliffe,[22] suggested that the primary type of splenic abscess was caused by splenic enlargement due to malaria or schistosomiasis, with subsequent subcapsular hematoma formation produced by friction of the enlarged spleen against the ribs. Others have also written of "splenitis" in Africans due to infarction of "malarial spleens,"[15,16] and cases of splenic abscess reported in the early 1900s were also attributed to malaria.[30] Hemoglobin genotypes were not known in these early cases and there has been little or no evidence to support the view that primary splenic abscess can be attributed to malaria alone. Wallace[40] and Gelfand[17] suggested that thrombosis of the splenic vein and/or artery is important in the development of the primary type of splenic abscess. Thrombophlebitis of other vessels was a common feature in their reports and some authors refer to an entity of tropical primary phlebitis.[12,29] Thrombosis of the splenic veins has been suggested as a factor in the development of secondary abscesses also. Cockshott and Weaver[6] reported thrombosis of the splenic vessels in 2 out of 6 cases of splenic abscess seen in Ibadan. Aortography in 2 of our patients (Cases 8 and 12) showed the splenic artery to be patent and of normal caliber (Fig. 3*B*).

Although infection is "always" present in the metastatic or secondary abscesses, its role in primary abscesses is uncertain and controversial. Reports in the literature contain both "sterile" cases and infected cases. Are the latter just secondary invaders into necrotic splenic tissue or sterile abscesses or, when present, is the infection a primary event in the so-called

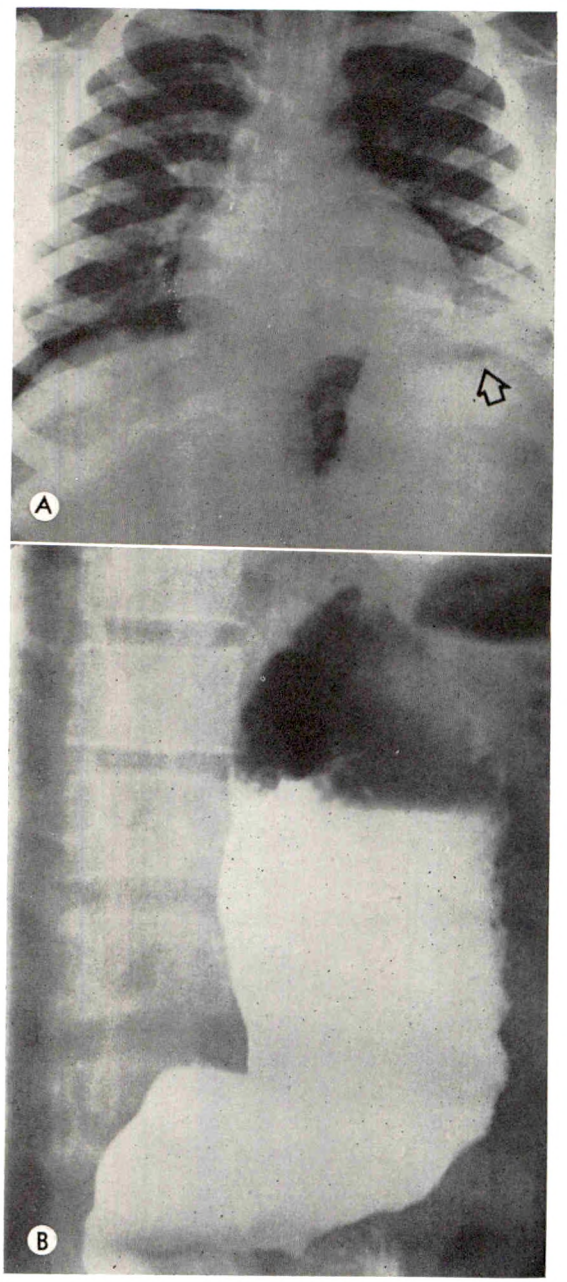


Fig. 7. Case 4. *Type B* splenic abscess in a 21 year old man with Hb SC disease.

(*A*) The left hemidiaphragm is elevated and the gastric air bubble is displaced to the right by the left upper quadrant mass. A small air bubble is present within the mass (arrow). (*B*) Barium in the stomach demonstrates that the higher air-fluid level to the left is not stomach. The air-fluid level within the abscess has a straight-line interface.

primary splenic abscesses; *i.e.*, must infarction precede septic involvement? At present, there are no clear-cut answers to these questions.

GENE FOR HEMOGLOBIN S

Thirteen of the 15 cases (87 per cent) of primary splenic abscess in our series, whose hemoglobin electrophoresis was done, had hemoglobin S (Hb S) and another hemoglobin present; *i.e.*, they had 1 gene for the production of Hb S (Tables I and II). Five of the 6 cases (83 per cent) in Cockshott and Weaver's series[6] from Ibadan also had 1 gene for Hb S. Normally, only about 25 per cent of the Ibadan population are carriers of the S gene.[11] Thus, the number of persons in this series and in Cockshott and Weaver's series[6] who carry a gene for the production of Hb S is highly significant.

The absence of any Hb SS patients (homozygous sickle cell anemia) in these series is not necessarily significant. With the expected frequency of homozygous sickle cell anemia in Ibadan, less than 2 per cent,[11] one would not normally, with unselected conditions, expect to find any patient with Hb SS in our series of 16 cases with known hemoglobin genotypes or Cockshott and Weaver's series[6] of 6 cases with known genotypes. However, one could expect 1 patient with Hb SS in a given group of 13 Ibadan patients with some Hb S shown to be present electrophoretically, but unselected otherwise. The fact that many homozygous sickle cell anemia patients do not survive childhood, and those who do often have small fibrotic spleens, may be related to the absence of such patients in these series.

Beet[2] was probably the first to suggest that the etiology of primary splenic abscess is related to the presence of Hb S. He reported 2 cases, both of which were most probably sickle cell trait (Hb AS) considering his clinical and hematologic findings; hemoglobin electrophoresis was not routinely available at that time. Hemoglobin genotype estimations were not discussed in other early cases attributed to "tropical phlebitis" or malarial infarcts. Cockshott and Weaver[6] suggested that a gene for hemoglobin S is probably a predisposing factor for the development of these abscesses.

In our series, there are also significant hemoglobin genotype differences in relation to the roentgenologic types of abscesses described. Five of the 6 patients with Type A primary abscesses (no gas present) and known genotypes (1 case was unknown) were Hb AS. Six of the 7 patients with Type B abscesses (gas present) and known genotypes (1 case was unknown) were Hb SC. The seventh patient with a Type B abscess and known hemoglobin genotype (Hb AC) had a secondary splenic abscess. Thus, all 6 patients with primary Type B abscesses and known genotypes were Hb SC. There were no Hb SC patients in our series who did not have the pyogaseous type of abscess (Type B). Two patients, one each in the series of Cockshott and Weaver[6] and Frankel *et al.*[14] with Hb SC had pyogaseous abscesses (Type B). Cockshott and Weaver[6] also found this type of abscess in 2 other patients, one with Hb AS, and one with normal adult hemoglobin (Hb AA). Beet's[2] case, presumably Hb AS, had gas present also.

HEMOGLOBIN SC PATIENTS AND TYPE B ABSCESSES

In Ibadan, the common symptomatic types of "sickle cell disease" are Hb SS, Hb SC and Hb S-Thalassemia (Hb S. Thal.). Hb AS is not considered here to be a variety of symptomatic sickle cell disease (see below—Hb AS patients). The almost exclusive occurrence of the pyogaseous type of primary splenic abscess in hemoglobinopathy patients with Hb SC disease would not be expected in a group of patients unselected except for the presence of Hb S; most should be Hb AS (over 95 per cent of Ibadan cases with Hb S are Hb AS). However, in a group of patients

with symptomatic sickle cell disease, most might be expected to have Hb SC disease for several reasons. By the very nature of sickle cell disease, infarctions are common in all organs of the body. They may be small and subclinical or produce the so-called painful or infarctive crises. As already noted, Hb SS patients do not commonly survive beyond childhood in Nigeria and, if they do, they tend to have small, fibrotic spleens, unlikely to undergo massive acute infarction. Splenic abscess has been reported in Hb S. Thal. disease,[18] but this hemoglobinopathy is uncommon in Nigeria; it occurs in less than 0.1 per cent of the population.[11] By contrast, Hb SC disease patients, although making up only about 0.8 per cent of the Ibadan population,[11] usually survive to adulthood and have an enlarged spleen. Thus, if infarcts are important in the etiology of primary splenic abscesses and patients with symptomatic sickle cell hemoglobinopathies are more likely to develop splenic infarcts than normal patients (Hb AA), perhaps for these simple numerical and pathophysiologic reasons alone, most patients with pyogaseous primary splenic abscesses have Hb SC disease, rather than the other varieties of sickle cell disease (Hb SS or Hb S. Thal.). Nongaseous abscesses in Hb AS patients are discussed below.

Patients with Hb SC disease may be more prone than Hb SS patients to develop splenic infarcts for hematologic reasons also; their blood viscosity is greater. At least 2 factors influence this: (a) Hb SC red blood cells are inherently more viscous than Hb SS red blood cells; and (b) severe anemia is present in Hb SS patients and not in Hb SC patients; therefore, the latter's blood is more viscous because it has more erythrocytes per unit volume.[5,7,27,41]

The association of *Salmonella* osteomyelitis in sickle cell patients is well known.[10] The association of Hb SC disease and *Salmonella* organisms in splenic abscesses was only recently recorded by Frankel *et al.*[14] *Salmonella* organisms have been reported in only a few other cases of splenic abscess.[6,18,36] The splenic aspirate of 2 of our cases grew *Salmonella* species (Cases 4 and 10); both were patients with Hb SC disease.

HEMOGLOBIN AS PATIENTS AND TYPE A ABSCESSES

Individuals with Hb AS (sickle cell carriers or trait) do not usually develop sickling *in vivo* because less than 50 per cent of their hemoglobin is Hb S, and their erythrocytes require a much lower oxygen tension to sickle than do the homozygotes. Anemia is not present in the carriers and their red blood cells have a normal life span. For practical purposes, most authors feel that there is no significant morbidity associated with the carrier state. It is, however, believed that certain unusual circumstances, such as flying in unpressurized airplanes or extreme exercise, may produce acute splenic infarctions and other acute clinical manifestations in sickle cell carriers,[9,19,26,34,37] even "sudden death."[23] There have been reports of carriers manifesting a variety of other problems also, such as bone infarcts, hematuria, renal papillary necrosis, inability to concentrate urine, complications with migraine headaches, and increased perinatal mortality.[1,3,8,21,25,31,32,39]

The 6 Hb AS patients in our series gave no history of unusual circumstances or stress preceding the development of their splenic abscesses. Similarly, Beet[2] and Cockshott and Weaver[6] made no mention of unusual precipitating events in relation to their 5 patients with Hb AS and primary splenic abscesses. It is, therefore, important to document this serious condition (subacute and acute primary splenic abscess) as one which can occur in sickle cell carriers, evidently under "normal" circumstances. Perhaps some of the many factors which can promote sickling such as hypoxemia, acidosis, pyrexia, etc., were present, but unnoticed, in these patients.

Why primary splenic abscesses occur in

Hb AS patients remains unknown. Severe malaria will not explain this finding, as Hb AS patients have some specific protection against the disease not present in Hb AA patients.[28] The presence of a large spleen will not account for the development of this condition in Hb AS patients. As children, in malarial areas, they tend to have smaller spleens than persons with other hemoglobin genotypes. Also, abscess formation is not a feature of idiopathic tropical splenomegaly, a condition probably related to chronic malaria and found more commonly in persons without Hb AS.[20,35,38] Perhaps it is the combination of a large spleen, a precipitating illness such as malaria or pneumonia, the presence of some sickle hemoglobin, and other, as yet unknown factors which are required for the development of the nongaseous type of primary splenic abscess. However, if the presence of Hb S alone is the selective factor for the development of primary splenic abscesses without gas, most such cases in Ibadan would be expected to be in Hb AS patients.

Five of our 6 Hb AS patients with primary splenic abscesses developed the Type A abscess, with no gas present. The presence or absence of gas within a splenic abscess is the one feature which distinguishes our Type A abscesses from Type B abscesses. It seems likely that the absence of gas in the Hb AS patients' abscesses, and the presence of gas in all of the abscesses in our Hb SC patients, is related to the different hemoglobins and/or to differences in the states of the spleen in these 2 groups of patients. Two of Cockshott and Weaver's[6] Hb AS patients and Beet's[2] Hb AS patient did have the pyogaseous type of abscess. Cockshott and Weaver[6] have commented on the source of the gas in these abscesses; they believe it is liberated from dying red blood cells and necrotic tissue. Beet[2] stated that the gas obtained by direct aspiration in his case was foul smelling. Three of our 5 patients with Hb AS and Type A abscesses (no gas) had sterile cultures, and 2 had organisms present. Two of the patients with Type B abscesses (gas present) and Hb S had sterile abscesses, and 4 had positive cultures. The exact nature of the gas and mechanism of its production remain unconfirmed and, therefore, any basic difference in the etiologies of these two varieties of primary splenic abscesses remains speculative.

TYPE C ABSCESSES

The 3 patients with known genotypes and Type C abscesses were either Hb AA or Hb AS. One of the Hb AA patients (Case 1) may have had a secondary abscess. This small group of patients with localized and/or generalized bowel distention, fluid levels, and ascites, in addition to their splenic abscesses, is thought to represent primary splenic abscesses with associated perisplenitis and/or peritonitis. The absence of intrasplenic gas in these cases suggests that they began as Type A abscesses. The absence of any Hb SC patients in this group is also in keeping with this suggestion.

These patients did present with a longer average history of disease than those with Type A or Type B abscesses (Table II; 15 weeks compared to 2–4 weeks), and slightly higher white blood cell counts. They had huge abscesses totally replacing the spleen (Cases 18 and 20) and/or proof of abscess rupture (Cases 6, 18 and 19). If the rupture is walled off by loops of small bowel, then a localized ileus occurs. Splenic abscesses in nonsickle cell patients (Cases 1 and 18) can also rupture and produce Type C roentgenologic findings.[14] Perhaps Type B abscesses in Hb SC patients do not develop these complications because a thickened splenic capsule protects them against rupture.

Three patients with Type C abscesses died (50 per cent) while only 1 patient each with Type A and Type B abscesses died (14 per cent and 12.5 per cent, respectively) (Tables I and II).

DIAGNOSIS, ROENTGENOLOGY AND TREATMENT

The marked preponderance of males over females in our series, the dominating clinical features of splenic pain associated with unremitting fevers, and the brownish-red, anchovy-sauce fluid recovered from the abscesses are all findings which have also been reported by previous writers.[6,15,16] Anchovy-sauce aspirate from the spleen is not pathognomonic of an amebic abscess,[40] as it would be if aspirated from the liver. The color is probably due to a large number of recently destroyed erythrocytes. Tympany, succussion splash or a fluid thrill over a tender splenic area is not only pathognomonic of a splenic abscess,[6,40] but it also indicates the gaseous type of abscess (Type B). Three cases (Cases 2, 21 and 22) in this series, 4 of the cases of Cockshott and Weaver[6] and Beet's[2] patient had one of these findings.

Although most left subdiaphragmatic air-fluid levels are in the gastric fundus, this is not always so. An air-fluid level with a clear, distinct, single straight line interface (Fig. 7*B*),[5] an air-fluid level far to the left (Fig. 6), or 2 separate subdiaphragmatic air-fluid levels on the left (Fig. 7) should raise suspicion of the presence of an abscess cavity. A fluid level in the splenic colon may also produce this appearance. Careful evaluation of the plain roentgenograms plus simple contrast medium examinations such as intravenous pyelography, barium meal (Fig. 4*B*; and 7*B*) or barium enema examination usually allows one to make the correct diagnosis. It is important to note that one of our patients (Case 16) had a normal abdominal roentgenogram.

Case 12 with undetermined hemoglobin genotype had splenic calcifications (Fig. 3). There were both irregular amorphous deposits within the spleen and a curvilinear marginal calcification. A variety of splenic calcifications have been reported in patients with the Hbs AA, SS and SC.[24] The calcifications in our patient are probably the result of previous infection and/or infarction.

The treatment of splenic abscess is primarily surgical with drainage of the abscess or, when possible, splenectomy and drainage of the left subphrenic space. Concomitant antibiotic therapy is also mandatory. Delay in surgical management is usually due to uncertainty of the diagnosis. The serious nature of this disease demands early diagnosis. This condition must be considered high on the list of possibilities in patients with hemoglobin S and suggestive clinical and/or roentgenologic features.

SUMMARY

Twenty-two cases of splenic abscess in Nigerians are reviewed; 2 were of the secondary type and 20 were primary splenic abscesses. Roentgenographically, 3 types of abscesses were distinguished: Type A with findings related only to splenomegaly; Type B with splenomegaly and gas within the abscess (pyogaseous abscess); and Type C with findings of localized or generalized peritonitis in addition to splenomegaly.

Fifteen patients with primary abscesses had hemoglobin electrophoresis done; 13 had Hb S present. Almost all patients with Type A abscesses had hemoglobin AS (sickle cell trait) electrophoretically. All patients with primary pyogaseous abscesses (Type B) had hemoglobin SC disease. There were no hemoglobin SS (homozygous sickle cell anemia) patients among those who had hemoglobin electrophoresis done. These findings and associations are discussed. The gene for Hb S is thought to be a significant factor in the etiology of primary splenic abscesses. It is important to recognize that Hb AS persons, who are usually asymptomatic, can develop this serious condition (primary splenic abscess) apparently without any unusual precipitating circumstances.

The clinical features in our cases were similar to previous reports. About 50 per

cent of our cases had sterile cultures. *Salmonella* organisms were grown from 2 abscesses in patients with Hb SC disease.

The roentgenologic and therapeutic aspects of splenic abscesses are briefly discussed.

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POSITIVE CONTRAST PERITONEOGRAPHY: THE ABNORMAL ABDOMEN*

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POSITIVE contrast peritoneography is a relatively new method of visualizing the peritoneal and serosal surfaces of the abdominal cavity which in many applications may prove superior to pneumoperitoneum. Positive contrast media have the advantage of diffusing throughout the abdominal cavity, roentgenographic contrast can be controlled over a wide range, and small structures are easily delineated. The safety of water soluble radiopaque media in the abdominal cavity has been demonstrated in animals and humans.[1-3,5-11,13,14] The performance of positive contrast peritoneography in living subjects has been described and abdominal masses and hernias have been identified by this means.[1,3,5,13,14]

This article describes the roentgenographic appearance of a number of pathologic entities as seen during positive contrast peritoneography. It is a sequel to the author's previous description of the normal abdominal anatomy as visualized by this procedure.[4]

MATERIAL AND METHOD

Materials were derived from a total of 41 positive contrast peritoneograms, of which 19 had demonstrable roentgenographic and gross pathologic abnormalities. Twenty-seven of the 41 studies were performed on cadavers with confirmation of the roentgenographic findings at autopsy. Eleven studies involved accidental spills of large volumes of contrast material into the abdominal cavity, with surgical confirmation of findings. Three living patients underwent peritoneography, with findings confirmed at surgery or by autopsy at a later date. Most studies were performed with 300 cc. of 25 per cent sodium diatrizoate* solution, as packaged for pyelography.

FINDINGS

The pathologic findings to be described are summarized in Table 1.

PERITONEAL CAVITY

Abnormalities of the parietal peritoneum consisted of adhesions, hernias, intraperitoneal masses, and displacement by extraperitoneal masses.

Three forms of adhesions were demonstrated. In 1 case with adhesions in the right lower quadrant following appendectomy, the cecum was adherent to the adjacent parietal peritoneum, giving the appearance of an abnormal mass in the right paracolic gutter (Fig. 1*A*). In 2 patients with extensive adhesions following laparotomy, 1 of whom had recurrent colonic adenocarcinoma, the instilled contrast material was confined to a relatively small

* Hypaque sodium.

TABLE 1

TYPES OF PATHOLOGY DEMONSTRATED DURING POSITIVE CONTRAST PERITONEOGRAPHY AND CONFIRMED AT SURGERY OR AUTOPSY

	No. of Examples*
Adhesions	4
Hernias	2
Retroperitoneal hematomas	5
Primary neoplasms	3
Recurrent or metastatic carcinomas within the abdominal cavity	2
Liver metastases	3
Other liver abnormalities	3

* In a total of 19 abnormal studies.

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From the Cincinnati General Hospital and Sinai Hospital of Detroit (X-Ray Departments of the University of Cincinnati and Wayne State University Colleges of Medicine).

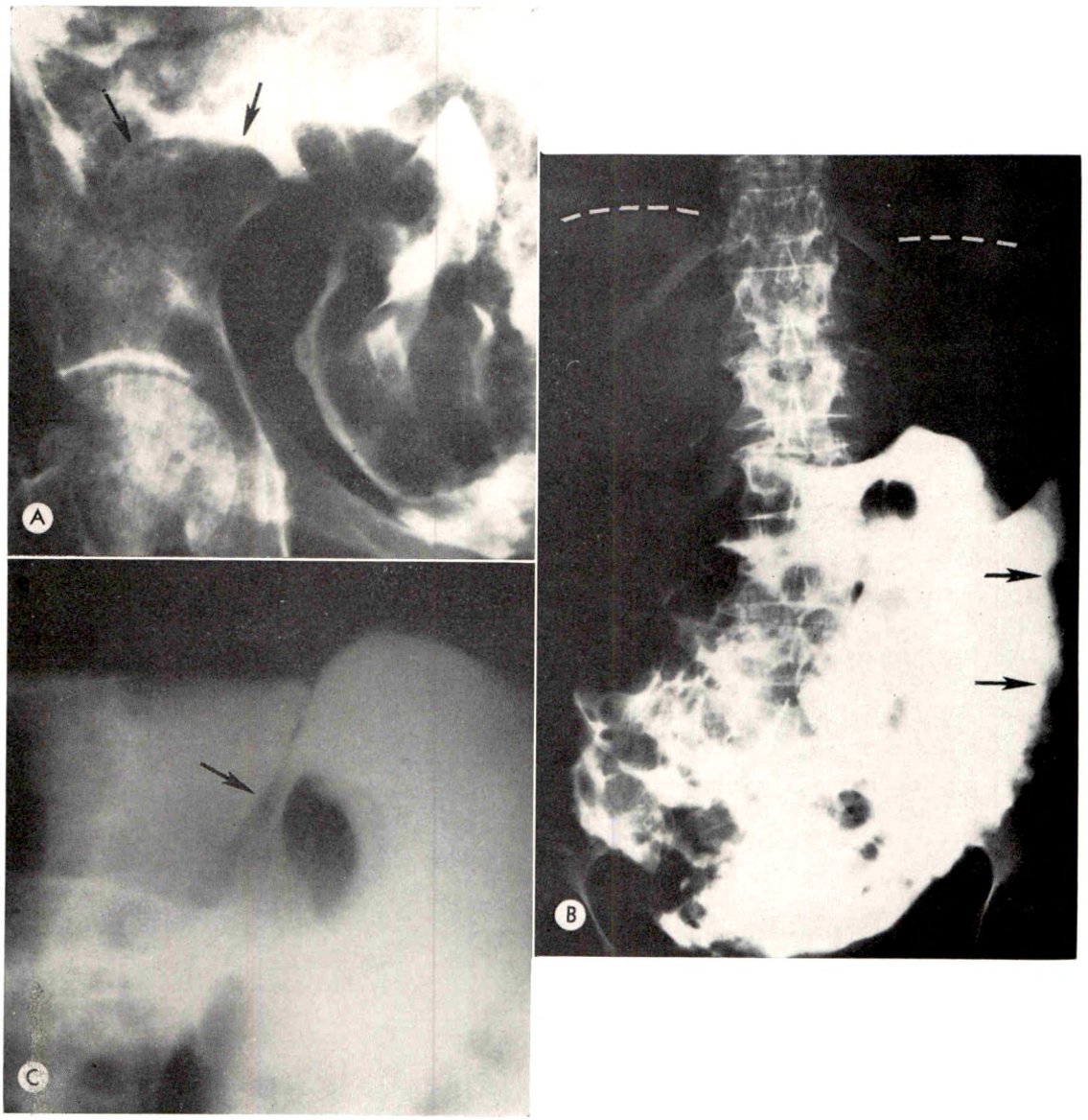


Fig. 1. (*A*) The cecum is bound to the parietal peritoneum by adhesions and interrupts the normally smooth contour of the right paracolic gutter simulating an abnormal mass (arrows). (*B*) Dense adhesions confine the contrast material to the lower abdomen (dashed lines outline the diaphragm). The liver, spleen, stomach and other upper abdominal structures, normally bathed by contrast material, are not visualized. The irregular mass in the left paracolic gutter is recurrent adenocarcinoma of the descending colon (arrows). (*C*) Filmy adhesions (arrow) appear as linear filling defects, similar to mucus shards seen during the barium enema examinations.

portion of the peritoneal cavity and was, in effect, loculated (Fig. 1*B*). This contrasts with the normal condition wherein the entire peritoneal cavity is bathed by contrast medium, and the lateral and anterior portions of the parietal peritoneum are easily visualized and relatively smooth in contour. In 1 patient, filmy adhesions appeared as linear filling defects similar in appearance to mucus shards on a barium enema roentgenogram (Fig. 1*C*).

Two examples of hernias were recorded: an occult hernia of the pelvic floor in an elderly individual (Fig. 2*A*); and a patent

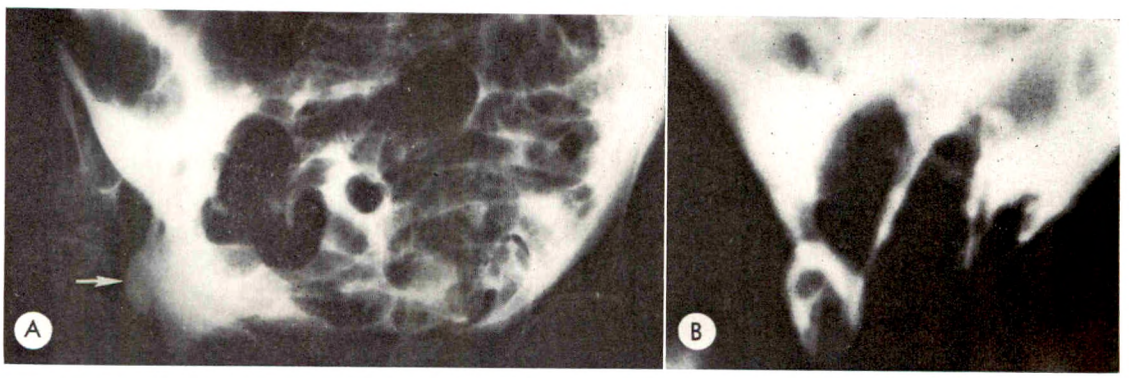


FIG. 2. (*A*) Occult hernia of the right side of the pelvic floor (arrow) in an elderly female, asymptomatic during life. (*B*) Patent processus vaginalis in a stillborn male infant. The testis and spermatic cord are visible within the hernial sac.

processus vaginalis in a stillborn infant (Fig. 2*B*).

In a patient with recurrent colonic carcinoma, intraperitoneal neoplasm was visible as a large, irregular filling defect occupying the left paracolic gutter (Fig. 1*B*). In another patient with metastatic carcinoma of the ovary, small tumor nodules studding the pelvic peritoneum were easily detected (Fig. 5*C*).

In 1 cadaver with a ruptured abdominal aortic aneurysm, extrinsic pressure from a large retroperitoneal hematoma obliterated most of the left side of the abdominal cavity (Fig. 3*A*). Four patients with pelvic fractures had displacement of the pelvic peritoneum by hematomas (Fig. 3*B*).

LIVER AND SPLEEN

The liver and spleen were the most easily identified and evaluated structures within the abdominal cavity. In 3 patients with extensive liver metastases, distinct nodules were visible along the inferior surfaces of the liver (Fig. 4*A*). In contrast, metastatic lesions involving the domed surface of the

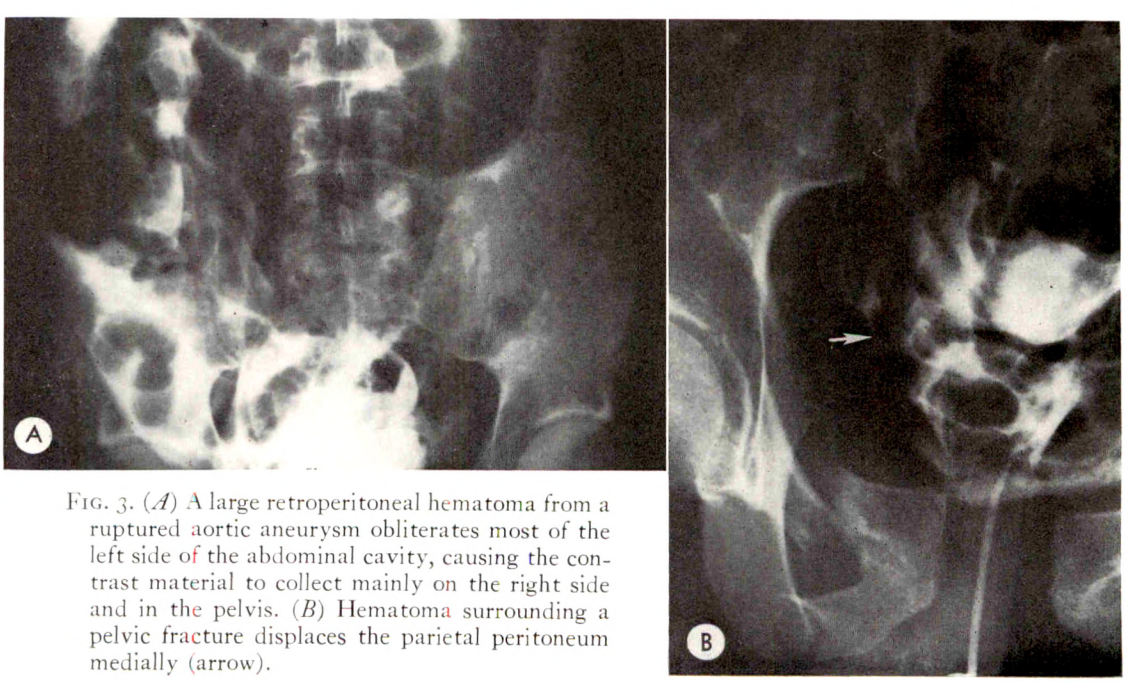


FIG. 3. (*A*) A large retroperitoneal hematoma from a ruptured aortic aneurysm obliterates most of the left side of the abdominal cavity, causing the contrast material to collect mainly on the right side and in the pelvis. (*B*) Hematoma surrounding a pelvic fracture displaces the parietal peritoneum medially (arrow).

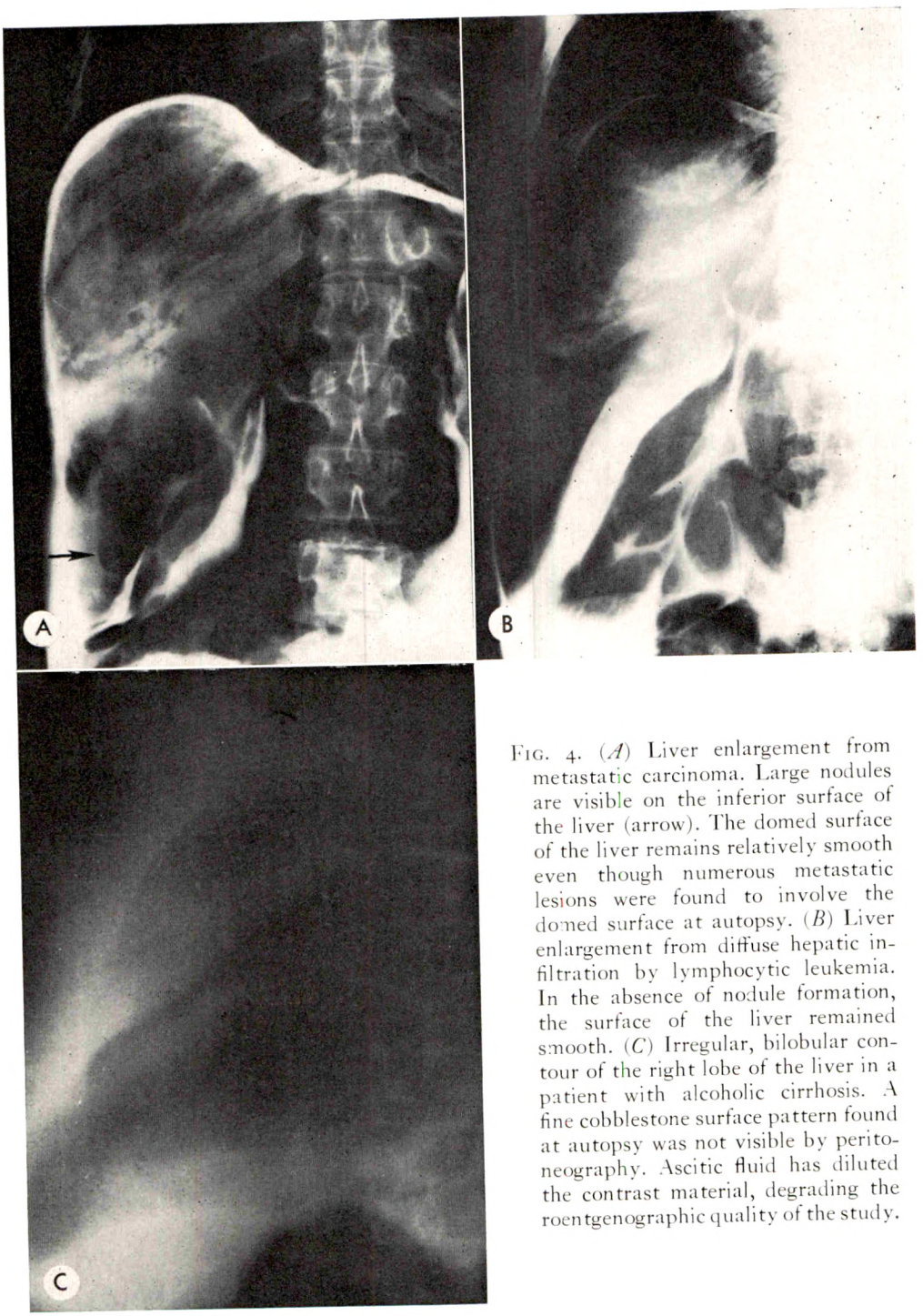


Fig. 4. (*A*) Liver enlargement from metastatic carcinoma. Large nodules are visible on the inferior surface of the liver (arrow). The domed surface of the liver remains relatively smooth even though numerous metastatic lesions were found to involve the domed surface at autopsy. (*B*) Liver enlargement from diffuse hepatic infiltration by lymphocytic leukemia. In the absence of nodule formation, the surface of the liver remained smooth. (*C*) Irregular, bilobular contour of the right lobe of the liver in a patient with alcoholic cirrhosis. A fine cobblestone surface pattern found at autopsy was not visible by peritoneography. Ascitic fluid has diluted the contrast material, degrading the roentgenographic quality of the study.

liver were not as readily detectable, apparently molded flat by pressure of the adjacent abdominal wall and diaphragm. In a cadaver with lymphocytic leukemia and another with alcoholic cirrhosis, hepatic enlargement was readily detected; however, the surface of the liver was smooth, the diseased process having produced no gross nodularity (Fig. 4*B*). In another cadaver with cirrhosis, the irregu-

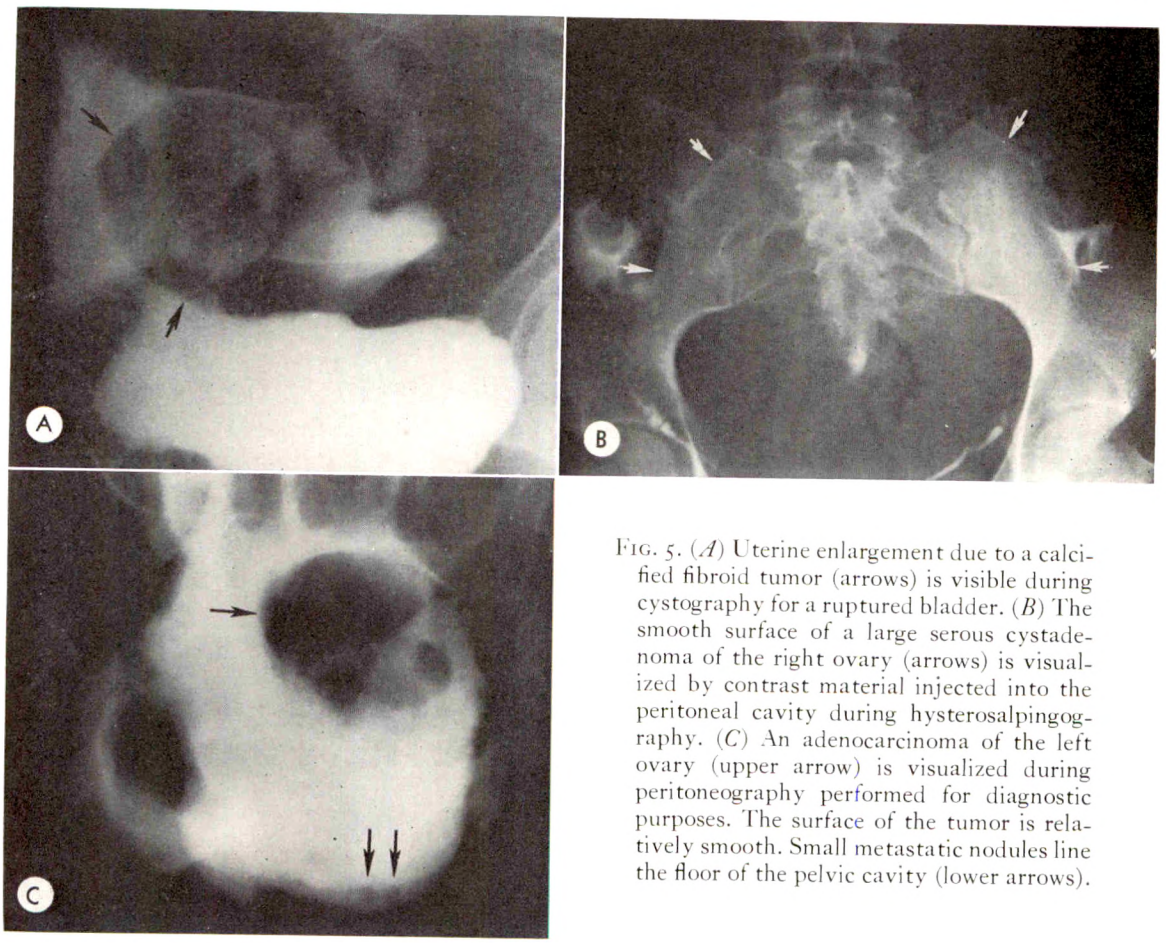


FIG. 5. (*A*) Uterine enlargement due to a calcified fibroid tumor (arrows) is visible during cystography for a ruptured bladder. (*B*) The smooth surface of a large serous cystadenoma of the right ovary (arrows) is visualized by contrast material injected into the peritoneal cavity during hysterosalpingography. (*C*) An adenocarcinoma of the left ovary (upper arrow) is visualized during peritoneography performed for diagnostic purposes. The surface of the tumor is relatively smooth. Small metastatic nodules line the floor of the pelvic cavity (lower arrows).

lar contour of the right lobe of the liver was apparent (Fig. 4*C*). No splenic abnormalities were recorded in this series, but in 1 patient with extensive adhesions in the upper abdomen, both the liver and spleen were totally invisible to peritoneography (Fig. 1*B*).

HOLLOW VISCERA

There were no lesions of the hollow viscera detectable by positive contrast peritoneography. In a living subject with carcinoma of the breast metastatic to the gastric antrum, there was no grossly visible abnormality of the gastric serosa during peritoneography or at autopsy 5 months later. In a cadaver with findings of chronic cholecystitis, the external surface of the gallbladder was also roentgenographically normal. None of the cadavers or living patients had a small bowel or colonic abnormality other than adenomatous polyps.

FEMALE REPRODUCTIVE ORGANS

Three patients had neoplasms of the female reproductive organs. Uterine enlargement from a fibroid tumor was clearly visible in 1 case (Fig. 5*A*). In another case, a large serous cystadenoma of the right ovary was seen as a smooth-surfaced mass filling the entire pelvic cavity (Fig. 5*B*). In the third instance, an adenocarcinoma of the left ovary and its metastases were detected preoperatively during a purposely performed peritoneography (Fig. 5*C*). In contrast to these 3 cases, the normal uterus and ovaries are seldom clearly visualized during positive contrast peritoneography.[4]

MESENTERIES, OMENTA AND LIGAMENTS

The greater and lesser omentum, the hepatic ligaments, the mesenteries, and the various ligaments surrounding the spleen are normally visible in varying degrees during positive contrast peritoneography.[4] In 2 patients with extensive adhesions, they were bound to adjacent organs or to the parietal peritoneum and were for the most part rendered invisible.

DISCUSSION

Although limited in number, these cases demonstrate that a wide variety of pathologic conditions can be detected by positive contrast peritoneography, including adhesions, hernias, benign and malignant neoplasms, metastatic lesions and retroperitoneal masses. Moreover, their roentgenographic appearance during peritoneography closely conforms to the gross pathology found at autopsy or surgery. It was also determined that the major peritoneal compartment and its solid organs are likely areas for investigation by peritoneography, while the hollow organs and lesser sac most likely are not.

On the basis of this limited study, a few tentative and incomplete guidelines are advanced for interpreting positive contrast peritoneograms. Any interruption of the smooth contours of the parietal peritoneum is probably abnormal, and is likely to be a metastatic or recurrent malignancy (Fig. 1*B*), or a normal structure bound down by adhesions (Fig. 1*A*). Dense adhesions tend to loculate the contrast material and may render areas of the abdomen invisible to peritoneography (Fig. 1*B*). In somewhat different fashion, extraperitoneal masses may obliterate portions of the abdominal cavity by displacement (Fig. 3, *A* and *B*). Metastatic tumor nodules in the liver are most readily detectable when they involve its inferior surface (Fig. 4*A*). Metastatic nodules which do not involve the surface of the liver will almost certainly be undetectable by peritoneography. The normal female reproductive organs are not usually seen during positive contrast peritoneography,[4] but are readily visible when enlarged by a sizeable neoplasm (Fig. 5, *A–C*).

Although neoplastic masses can be detected by positive contrast peritoneography, differentiation between benign and malignant tumors may not always be possible. Highly irregular malignant masses (Fig. 1*B*) and metastatic studding of the parietal peritoneum (Fig. 5*C*) are unlikely to be mistaken for benign tumors. On the other hand, a malignacy which has not destroyed the serosal covering of the involved organ probably cannot be differentiated from a benign neoplasm. Also, it has been demonstrated that a normal structure may be mistaken for a tumor within the abdominal cavity if it has been bound to the parietal peritoneum by adhesions (Fig. 1*A*).

It has become apparent that particular techniques yield the best peritoneograms. For optimal radiographic contrast, kilovoltages should be low (70 kv.). Contrast material should be instilled into the abdomen as near the suspected pathology as possible with the injection monitored fluoroscopically. Any ascitic fluid should be withdrawn before the injection. Posteroanterior, anteroposterior and oblique views of the abdomen should be obtained. The effects of gravity and position should be utilized to bring an adequate amount of contrast material into the desired area of the abdomen.

In practice, 75 grams of sodium diatrizoate in 300 cc. of water, as supplied for pyelography, has provided satisfactory roentgenographic contrast and adequate volume. The material was well tolerated during 3 peritoneographic examinations performed on living subjects and was readily absorbed from the peritoneal cavity. Pyelograms were produced within 15 minutes, and 1 hour after instillation there was a visible decrease in the amount of contrast material within the abdomen. Twenty-four hours after the studies, contrast material was no longer visible in the abdominal cavity or urinary tract.

In terms of specific clinical applications,

positive contrast peritoneography is most likely to be helpful in detecting the presence of abnormal masses within or adjacent to the peritoneal cavity, in visualizing the surfaces of the solid organs, in evaluating the presence and extent of adhesions, and in demonstrating the presence of occult hernias. In 3 peritoneographies performed on living subjects, primary malignancy, recurrent neoplasm, and metastatic studding of the peritoneum were demonstrated in the 2 patients in whom these subsequently proved to be present. Three of 4 examples of intraabdominal adhesions were detected by peritoneography prior to being found at surgery or autopsy. All 3 examples of metastatic liver disease were detected roentgenographically before being confirmed anatomically. Other studies have shown the method's ability to detect intraabdominal masses and occult hernias.[1,3,5,13,14]

Positive contrast peritoneography, while useful in a number of situations, is not likely to be performed with great frequency. Radioisotope techniques provide much information about the liver and spleen with less inconvenience to the patient. Barium studies remain the primary means of examining the stomach and bowel. Pneumoperitoneum provides a more reliable method of evaluating the female reproductive organs. Peritoneoscopy permits direct visualization of much of the abdominal cavity as well as the possibility of biopsy. Nevertheless, when pneumoperitoneum is being contemplated, positive contrast peritoneography should be considered as an alternative examination capable of more easily visualizing the entire major peritoneal compartment and its contents.

SUMMARY

This paper describes the roentgenographic appearance of a variety of pathologic conditions as seen during positive contrast peritoneography, and is a sequel to the author's previous description of normal abdominal anatomy as visualized by this procedure.

The material studied includes benign and malignant neoplasm, metastatic lesions, retroperitoneal masses, organ enlargements, adhesions and hernias.

The ease and clarity with which the major peritoneal compartment and its contents can be visualized suggest that positive contrast peritoneography may be preferable to pneumoperitoneum in certain applications.

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DISTRIBUTION OF INTRA-ABDOMINAL MALIGNANT SEEDING: DEPENDENCY ON DYNAMICS OF FLOW OF ASCITIC FLUID*

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NEW YORK, NEW YORK

THE spread of neoplasms within the peritoneal cavity occurs by direct invasion, true embolic metastases, lymphatic extension, and intraperitoneal seeding.[10,16] Direct invasion occurs from locally aggressive contiguous primary tumors or from noncontiguous lesions along mesenteric planes. Hematogeneous metastases may follow discrete vascular routes, both venous and arterial. Similarly, lymphatic extension and permeation follow known anatomic channels.

There is, however, only very limited understanding of the factors contributing to the distribution of intra-abdominal seeded metastases. It has been generally assumed that transcoelomic spread is random or, at least, a function of serosal implantation in the immediate area of a primary neoplasm. The relationship of the circulation of the induced ascitic fluid to the transporting and deposition of malignant cells in the peritoneal cavity has not been previously shown. In an earlier report, the observations on spread of intraperitoneal effusions derived from a limited series of peritoneography were first recorded.[9] Emphasis was placed on the pathways of flow in the upper abdominal recesses and their correlation with the localization of intraperitoneal abscesses remote from their source. Further dynamic observations have detailed the precise flow patterns dominating in the lower abdomen and pelvis.

The purpose of this report is to document: (1) by detailed *in vivo* fluoroscopic and roentgenologic observations that ascitic fluid is not in a static state, but pursues a continual dynamic flow in the lower abdomen and pelvis along distinct pathways. These pathways are determined by peritoneal reflections and recesses, as well as the forces of gravity and negative subdiaphragmatic pressure; and (2) that the sites of lodgment and growth of intraperitoneal seeded metastases, shown in an analysis of a large proved series, follow the spread of the ascitic fluid.

MATERIAL AND METHOD

The dynamic pathways of flow of intraperitoneal fluid *in vivo* were studied in 35 hospitalized patients by the technique of peritoneography.[11] The patients ranged in age from 28 to 74 years, and were comprised of 22 males and 13 females. None had previous abdominal operations. The patient is positioned supine on the roentgenographic table. After local anesthesia, transabdominal needle puncture using a Rochester needle* is accomplished in the left supraumbilical area. Briefly, the technique consists of the intraperitoneal injection of 1,200–1,500 ml. of nitrous oxide followed by 150 to 200 ml. of 60 per cent meglumine diatrizoate† diluted with 50 to 75 ml. of normal saline. The presence of intraperitoneal gas enhances the positive contrast and helps its natural dispersion. Image amplified fluoroscopic observations detailed the patterns of flow of the intraperitoneal fluid and these were confirmed by roentgenograms of the abdomen. In several patients, the pathways of flow following successive 50 ml. increments of the contrast solution were fluoroscopically observed. Routine filming consisted of a

* Jelco Intravenous Placement Unit, Jelco Laboratories, Raritan, N. J.

† Renografin-60, E. R. Squibb & Sons, New York.

* From the Department of Radiology, The New York Hospital and Memorial Hospital for Cancer and Allied Diseases, Cornell University Medical Center, New York, New York.

series of 10 projections: frontal and both obliques in the prone Trendelenburg (20–30°); then posteroanterior, anteroposterior, and right and left lateral with the table horizontal; and then frontal and both obliques in the supine Trendelenburg (20–30°). In the early stages of the investigation, decubitus and cross-table lateral views were also obtained and contributed to an understanding of the pathways of flow. Following the procedure, the plugged plastic cannula left *in situ* was opened to permit as much of the gas as possible to escape. Both the water-soluble contrast medium and any residual gas are spontaneously absorbed by the peritoneal surfaces over the next few hours. Since renografin 60 is hypertonic, even the relatively small amount used transiently induces some ascitic fluid. This was shown by progressive dilution of the contrast medium throughout the study.

A series of 34 cases of intraperitoneal malignant seeding, proved at operation or autopsy, from The New York Hospital and Memorial Hospital for Cancer and Allied Diseases, was reviewed. The roentgenologic and pathologic features were carefully analyzed, particularly in terms of the primary source and the distribution of secondary growths.

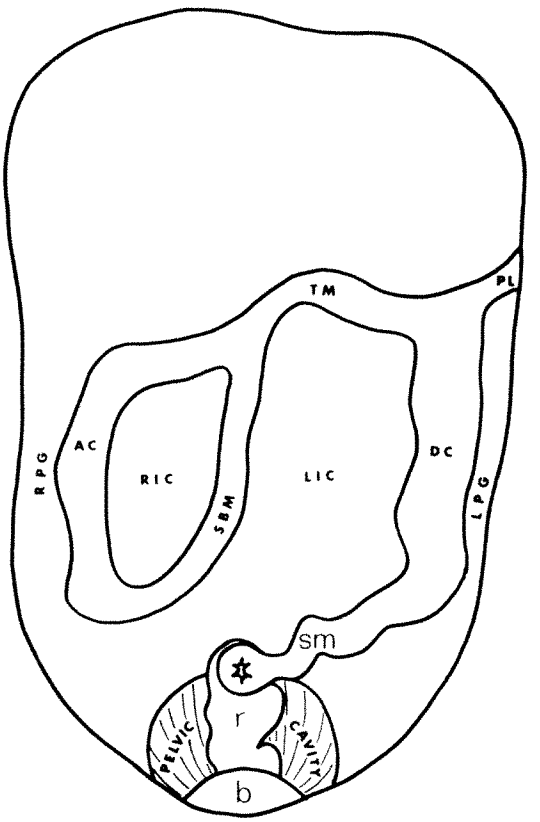


Fig. 1. Posterior peritoneal reflections and intra-abdominal spaces. TM = transverse mesocolon; PL = phrenicocolic ligament; SBM = small bowel mesentery; AC = attachment of ascending colon; DC = attachment of descending colon; SM = sigmoid mesocolon; r = rectum; b = urinary bladder; RIC = right infracolic space; LIC = left infracolic space; RPG = right paracolic gutter; LPG = left paracolic gutter.

ANATOMIC CONSIDERATIONS

The peritoneal cavity is subdivided by peritoneal reflections and mesenteric attachments into several compartments and recesses (Fig. 1). Most of these are anatomically continuous, either directly or indirectly.

The major barrier dividing the abdominal cavity is the *transverse mesocolon*. Below this, the obliquely oriented *small bowel mesentery* divides the inframesocolic space into 2 compartments of unequal size: the *right* and *left infracolic spaces*. The small bowel mesentery itself is of a characteristically ruffled nature. It can be likened to a series of fan-like extensions from the root of the mesentery. It is this feature that contributes to the curvature of the loops of small bowel. Multiple intraperitoneal recesses are thus formed within the reflections of the small bowel mesentery. Its main axis, nevertheless, is directed toward the right lower quadrant in relation to the terminal ileum and cecum. The right infracolic space terminates at their junction. The left infracolic space is anatomically open to the pelvis to the right of the midline; toward the left, it is restricted from continuity with the pelvic cavity by the sigmoid mesocolon.

The *right* and *left paracolic gutters* are lateral to the attachments of the peritoneal reflections of the ascending and descending colon. They represent potential

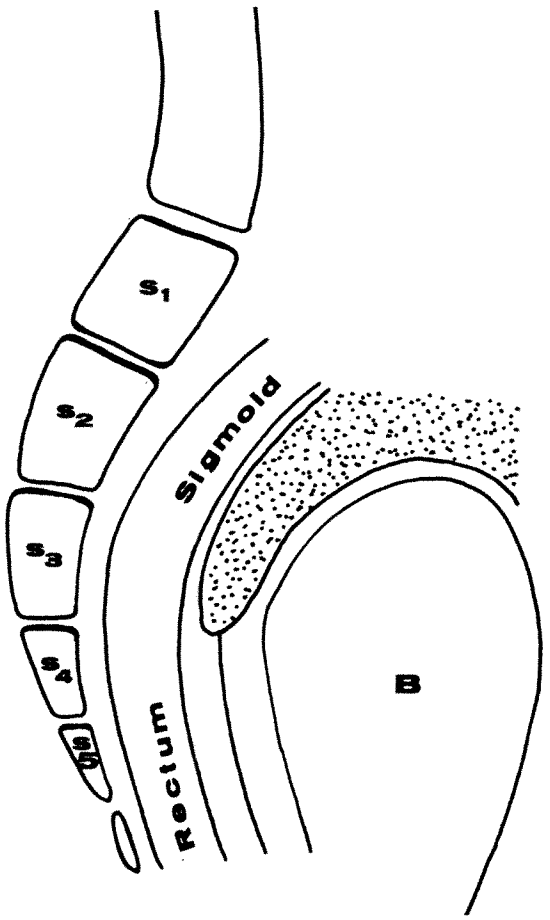


FIG. 2. Sagittal diagram of relationships of pouch of Douglas, the lower continuation of the peritoneal cavity between the rectosigmoid and the urinary bladder (B).

communications between the lower abdomen and pelvis below, with the supramesocolic area above. On the left, however, the phrenicocolic ligament partially separates the paracolic gutter from the perisplenic (left subphrenic) space. This ligament extends from the splenic flexure of the colon to the diaphragm at the level of the eleventh rib.[8]

In the supine position, the *pelvis* is the most dependent portion of the peritoneal cavity. Its compartments include the midline cul-de-sac or *pouch of Douglas* (rectovaginal pouch in the female and rectovesical pouch in the male) and the lateral paravesical recesses. The lower extension of the peritoneal reflections comprising the pouch of Douglas projects generally at the level of the lower second to upper fourth sacral segment (Fig. 2). This variability is determined by the developmental fixation of the peritoneum to Denonvilliers' fascia (rectovaginal or rectovesical septum) and the degree of distention of the urinary bladder and rectum.[7] The level is also a useful landmark to demarcate the junction between the rectum and the sigmoid colon. The ventral surface of the rectosigmoid junction thus faces the pouch of Douglas.

RESULTS

DYNAMIC ROENTGENOGRAPHIC OBSERVATIONS

Fluid in the inframesocolic compartment preferentially seeks the pelvic cavity. From the left infracolic space, flow is direct along the right side of the rectum. Some fluid is temporarily arrested along the superior plane of the sigmoid mesocolon but gradually channels into the pelvis. From the right infracolic space, spread occurs in a series of minor cascades or rivulets along the recesses of the small bowel mesentery. It is not until a pool is formed at the apex, at the termination of the ileum with the cecum, that some begins to overflow into the pelvis. The pouch of Douglas is first filled, and then symmetrically the lateral paravesical recesses (Fig. 3).

From this point the fluid ascends both paracolic gutters. Passage up the shallower left one is slow and weak, and cephalad extension is limited by the phrenicocolic ligament. The major flow from the pelvis is up the right paracolic gutter (Fig. 4). This then progresses to the right subhepatic and subphrenic spaces. Direct passage across the midline to the left subphrenic space is prevented by the falciform ligament.

The right paracolic gutter serves also as the main communication from the upper to the lower abdominal compartments. Freely movable fluid collecting in the right upper quadrant also continues to be redirected downward along this channel to the pelvis.

Four predominant sites are therefore

clearly identified as the preferential, repeated, or arrested flow of intraperitoneal fluid: (1) the pelvic cavity, particularly the pouch of Douglas; (2) the right lower quadrant at the termination of the small bowel mesentery; (3) the superior aspect of the sigmoid mesocolon; and (4) the right paracolic gutter. These pathways are illustrated in Figure 5.

Analysis of Clinical Cases of Intraperitoneal Malignant Seeding

Table 1 summarizes the major anatomic features of intraperitoneal seeding in this series. The primary malignancies arose in the ovary, pancreas, stomach and colon. It is apparent that, regardless of the primary origin of the neoplasm, 4 discrete sites of seeded metastases were encountered:

(1) *Pouch of Douglas.* Seeding at this site was the most common, occurring in 56 per cent. On barium enema study, this is revealed by a characteristic pattern of fixed parallel folds or a nodular indentation upon the ventral aspect of the rectosigmoid junction (Fig. 6). This change reflects the coalescence of deposits with a dense fibrous reaction.

(2) *Right lower quadrant.* Seeded deposits in the lower end of the right infracolic space along the root of the small bowel mesentery were clinically identifiable by their displacement of distal ileal loops, perhaps with pressure effects also upon the medial contour of the cecum and ascending colon. At times, multiple growths oriented along this axis within the terminal recesses of the mesentery may displace small bowel loops in a characteristic scalloped-crescent pattern (Fig. 7). Extensive desmoplastic reaction may fix and angulate ileal loops toward the small bowel mesentery (Fig. 8). This localization in the right lower quadrant was present in 41 per cent of the cases of intraperitoneal seeding.

(3) *Sigmoid colon.* Lodgment and growth of deposits arrested along the barrier of the sigmoid mesocolon in the left lower quadrant resulted in characteristic changes localized to the superior border

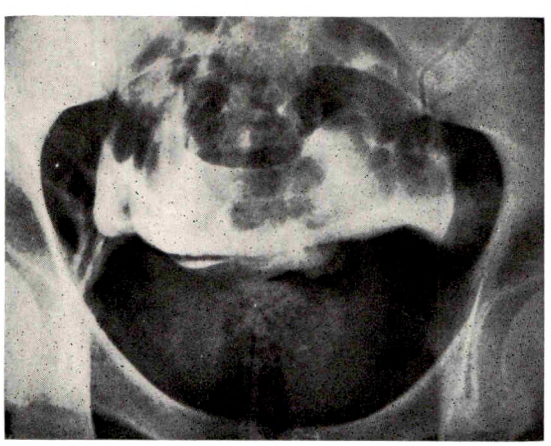


Fig. 3. Supine position. Intraperitoneal opaque contrast material first gravitates to the pelvic cavity, filling the pouch of Douglas and the 2 paravesical recesses.

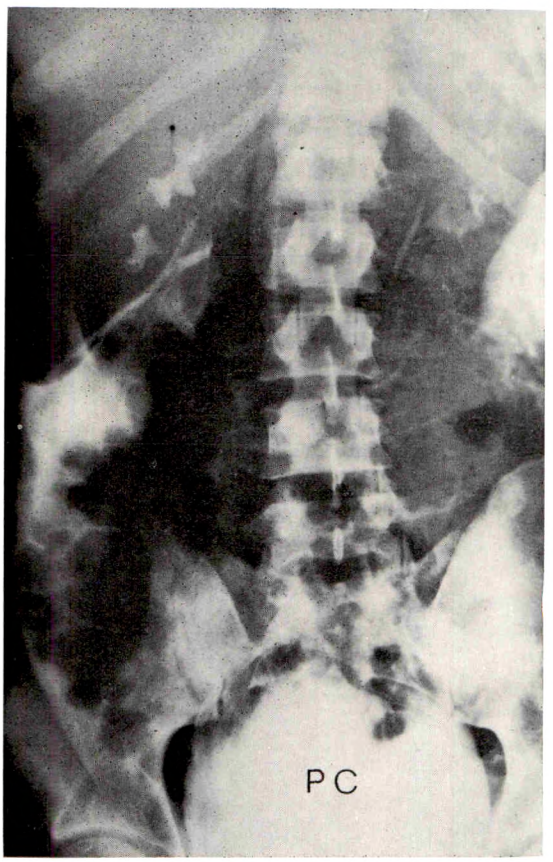


Fig. 4. Supine position. Intraperitoneal opaque contrast fluid fills the pelvic cavity (PC) and then extends cephalad along the right paracolic gutter.

TABLE I

ANALYSIS OF 34 CASES OF INTRAPERITONEAL MALIGNANT SEEDING

Primary Carcinoma		Seeded Site			
Site	No. of Cases	Pouch of Douglas	Lower Small Bowel Mesentery	Sigmoid Mesocolon	Right Paracolic Gutter
Ovary	15	9	7	1	4
Pancreas	8	5	2	3	
Stomach	7	4	3	2	2
Colon	4	1	2	1	
Total	34	19	14	7	6

of the sigmoid colon. The associated desmoplastic reaction caused tethering of the mucosal folds. These lose their normally perpendicular axis to the lumen of the bowel and become angulated, often toward a common point in the mesentery at the site of the secondary lesion (Fig. 9). This localization occurred in 21 per cent of the cases.

(4) *Right paracolic gutter.* Deposition and growth in this peritoneal recess, particularly its inferior portion, were shown by a mass lateral and posterior to the cecum and proximal ascending colon. Tethering of mucosal folds or angulated fixation of a small bowel loop in this area may occur as

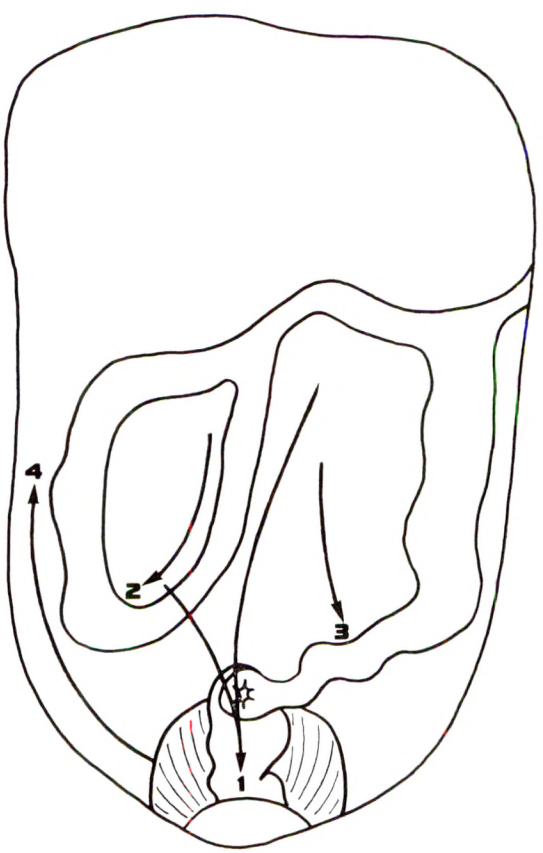


FIG. 5. Diagram of the pathways of flow of intraperitoneal fluid and the 4 predominant sites.

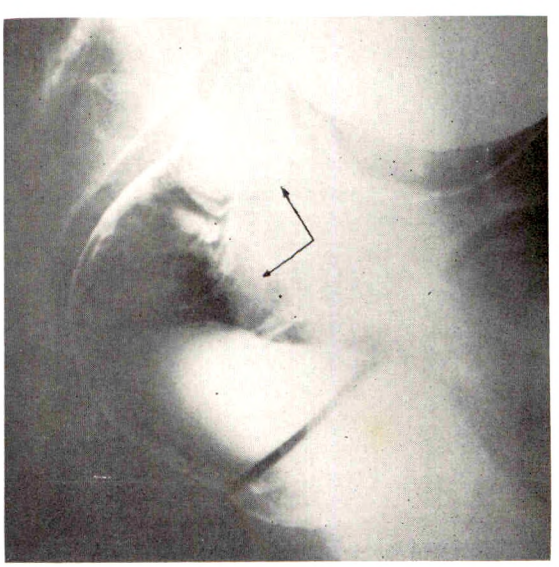


FIG. 6. Seeded metastasis in the pouch of Douglas results in a nodular indentation (arrows) upon the rectosigmoid junction anteriorly. (The primary in this case is a carcinoma of the cecum.)

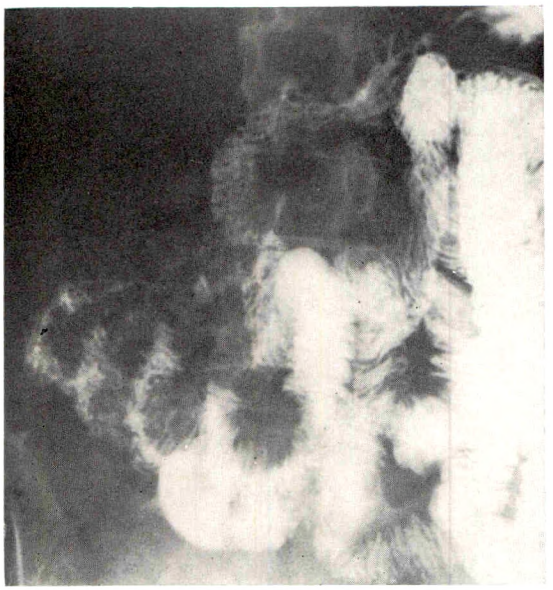


Fig. 7. Scalloped displacement of distal ileal loops in the right lower quadrant from multiple seeded deposits arising from carcinoma of the ovary. (Reproduced with permission from *Radiology*.[10])

a consequence of an associated fibrous reaction (Fig. 10). This localization occurred in 18 per cent of the cases.

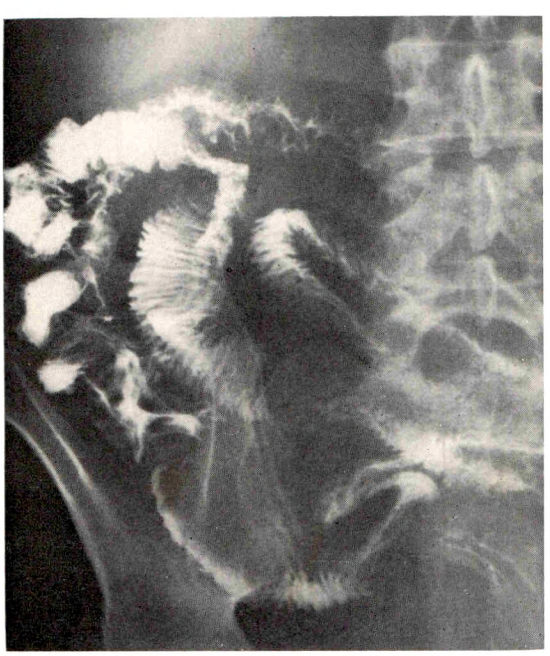


Fig. 8. Intraperitoneal seeding from carcinoma of the pancreas fixes and angulates ileal loops. There is considerable fibrous reaction.

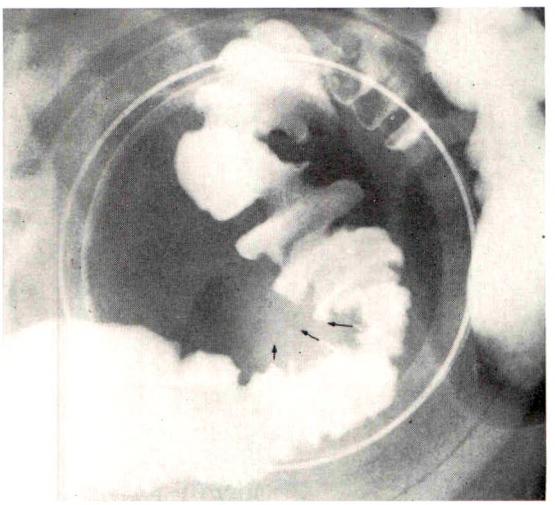


Fig. 9. Intraperitoneal seeding in the sigmoid mesocolon from carcinoma of the pancreas. The desmoplastic reaction causes tethering of the mucosal folds, angulated toward a common site in the mesentery (arrows).

Multiple sites of seeded deposition were somewhat more common than a solitary focus. Seeding in sites in addition to these

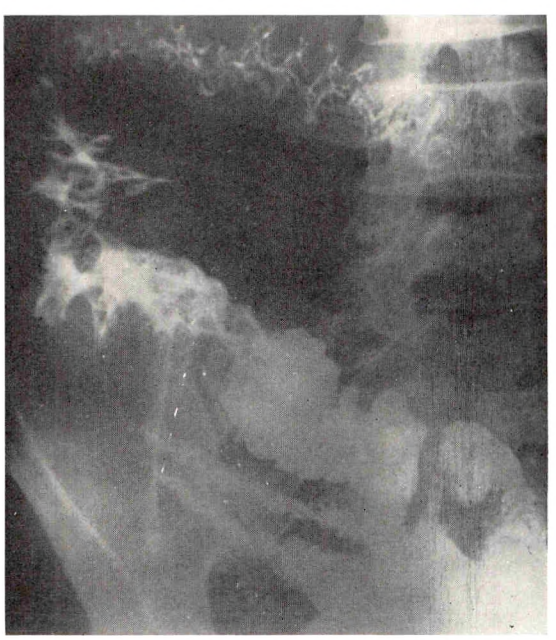


Fig. 10. Intraperitoneal seeding in the right paracolic gutter from gastric carcinoma. Nodular masses displace the ascending colon anteriorly and medially and cause angulated fixation of a small bowel loop. (Reproduced with permission from *Radiology*.[10])

4 foci occurred only 6 times in the 34 patients. These included 3 instances of metastases to the transverse and descending colon in 3 cases of ovarian carcinoma, and 2 instances of metastases to the jejunum and 1 to the splenic flexure of the colon in 2 cases of primary carcinoma of the colon. These disparate sites may have been related to adhesions from previous abdominal operations.

DISCUSSION

Fluoroscopic and roentgen observations clearly and consistently demonstrate that intraperitoneal fluid, rather than being static, continually follows a circulation through the abdomen. Its pathways of distribution and sequential spread are dependent on mesenteric reflections and peritoneal recesses. The transverse mesocolon, small bowel mesentery, sigmoid mesocolon, and the peritoneal attachments of the ascending and descending colon serve as watersheds directing the flow of intraperitoneal fluid. The force of gravity operates to pool peritoneal fluid in dependent peritoneal recesses. Most notable of these is the pelvic cavity, particularly the pouch of Douglas. This is the most caudal and posterior extension of the peritoneal cavity. Other conspicuous dependent recesses in the inframesocolic portion of the abdomen, particularly in the supine position, are the pockets along the small bowel mesentery, the apex of the right infracolic space, the trough along the cephalad margin of the sigmoid mesocolon, and the right paracolic gutter.

Egress from the pelvis upward is not a function simply of overflow. Fluid surmounts the sacral promontory and flank muscles to extend upward, whether the patient is horizontal or erect. In an effort to understand the spread of infection, Autio[1] first documented the intraperitoneal extension of radiographic contrast medium into the upper abdominal recesses even in the erect position. Ten cc. of contrast medium was introduced into the ileocecal, paracecal and paraduodenal regions of 38 patients in connection with appendectomy or cholecystectomy. It was found that the contrast medium moved both down into the pelvis and up into the subphrenic space, *via* the 2-way avenue of the right paracolic gutter. In addition to the anatomic pathways and action of gravity, variations in intraperitoneal pressure also determine the distribution of peritoneal fluid. The hydrostatic pressure of the contents of the abdominal cavity together with the flexibility of a portion of the abdominal walls determine, for the most part, the pressure within the abdominal cavity. Overholt[12] demonstrated in animals that the hydrostatic pressure is lower in the subdiaphragmatic region than elsewhere in the abdomen and that the pressure varies with respiration. The intraperitoneal pressure in the upper abdomen is subatmospheric and decreases further during inspiration. This negative subdiaphragmatic pressure and its relation to breathing is maintained in the horizontal or erect position. This is explained by the outward movement of the ribs during inspiration which enlarges the space in the upper abdomen more than it is decreased by the descent of the diaphragm. Salkin[14] subsequently confirmed these observations in humans, noting in a series of 50 cases that most showed an intraperitoneal pressure from 0 to −30 mm. water and that the pressure is less in the epigastrium than in the hypogastrium. Drye[6] recorded that in the upright position pressure in the lower abdomen is almost 3 times as great as in the supine position. These pressure differences with positional and respiratory variations have been confirmed by others.[2,13]

Surprisingly few observations have been made in the literature regarding the distribution of intraperitoneal seeding. Interest has apparently centered only on the most clinically evident site, *i.e.*, Blumer's shelf which represents a coalescence of deposits with a dense fibrous reaction in the pouch of Douglas.[7] First described in 1909,[4] it was not until 1939 that Bacon[2] published a series of 22 cases of "extra-

rectal metastatic malignancy simulating primary carcinoma of the rectum." In 1941, Buie *et al.*[5] reported a 5 year series from the Mayo Clinic consisting of 254 cases of secondary shelf tumors. They noted that the most common causes of extrarectal masses among women were lesions of the genital system, and among men lesions of the gastrointestinal tract. In 1955, Hultborn and associates[7] concluded that the factor essential to the development of a shelf tumor is pathologic fixation of the uppermost part of the rectogenital septum (Denonvilliers' fascia). Observations on the distribution of intraperitoneal seeded malignancies other than the pouch of Douglas are notably lacking. Willis' classic work on the spread of neoplasms[16] does not substantially further understanding of transcoelomic spread.

It is accepted that in breaking through into the peritoneal cavity, a primary neoplasm or even its intra-abdominal lymph node metastases can shed cells into the ascitic fluid induced. Our observations indicate that it then seeds deposits along the pathways of spread and localization of intraperitoneal fluid. The commonest sites of lodgment and growth of seeded metastases clearly correlate with the patterns of flow of ascitic fluid. Four specific sites are evident: (1) the pouch of Douglas at the rectosigmoid level; (2) the right lower quadrant at the lower end of the small bowel mesentery; (3) the left lower quadrant along the superior border of the sigmoid mesocolon and colon; and (4) the right paracolic gutter lateral to the cecum and ascending colon. The degree of ascites need not be great. Theander *et al.*[15] found characteristic changes in the rectosigmoid to be roentgenologically identifiable in 10 per cent of cases of ascites secondary to peritoneal carcinomatosis. In our material, the finding was present in 56 per cent of those cases with involvement of the peritoneal cavity at single or multiple sites, regardless of the degree of ascites. Localization in the right lower quadrant was clearly identifiable in 41 per cent, in the sigmoid mesocolon in 21 per cent, and in the right paracolic gutter in 18 per cent.

The fact that deposition and growth of secondarily seeded neoplasms in the abdomen is dependent upon the natural flow of ascites within the peritoneal recesses is not only of diagnostic significance but may also have therapeutic implications. Further efforts in the control of flow of ascitic fluid might be developed to localize seeded deposits for more effective radiotherapy or chemotherapy.

SUMMARY AND CONCLUSIONS

The distribution of intraperitoneal malignant seeding clearly correlates with the pathways of flow of ascitic fluid.

Four predominant sites of seeded metastases are identified: (1) the pouch of Douglas at the rectosigmoid level; (2) the right lower quadrant at the lower end of the small bowel mesentery; (3) the left lower quadrant along the superior border of the sigmoid mesocolon and colon; and (4) the right paracolic gutter lateral to the cecum and ascending colon. It is at these sites that the preferential, repeated or arrested flow of intraperitoneal fluid occurs.

It is concluded that deposition and growth of secondarily seeded neoplasms in the abdomen are dependent upon the natural flow of ascites within the peritoneal recesses.

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Representatives on the American Board of Radiology: Justin J. Stein, Los Angeles, Calif.; Ralph M. Scott, Louisville, Ky.; E. Richard King, Richmond, Va.

Liaison Representatives on the National Council on Radiation and Measurements: Marvin M. D. Williams, Rochester, Minn.; Morris J. Wizenberg, Baltimore, Md.

Representative on the Board of Chancellors of the American College of Radiology: Juan A. del Regato.

Fifty-sixth Annual Meeting: Surfer Hotel, Maui, Hawaii, April 21–25, 1974.

EDITORIALS

THE CONTINUUM PRINCIPLE IN DIAGNOSTIC ROENTGENOLOGY

LIKE Molière's character who was unaware he was speaking prose, the radiologist has been using the continuum principle since his first days in radiology: for a given anatomic part or region, there is a characteristic direction and rate of change of contour or of density as one proceeds from one point to another. For example, the interpedicular distance of the vertebral bodies presents a characteristic direction and rate of change as one proceeds from neck to sacrum; these characteristics of direction and rate of change in this example lend themselves nicely to quantitative graphic portrayal. Any deviation from the expected requires explanation.

Most continua, however, cannot be quantitated: a vertical diameter of a lumbodorsal vertebral body smaller than that of the vertebra above must be explained.

Another widely used continuum consists of the progressive anterior migration of the planes of the apophyseal joints as one proceeds from the upper lumbar to the lumbosacral level; any deviation in this orderly progression must be explained.

Other continua show no significant change in direction of contour (hence, no rate of change); a sudden bulge in the left paraspinal soft-tissue stripe is abnormal and must be explained.

I have found it helpful, and entertaining, to "devise" other continua, and, regarding them as continua with normal expected patterns, I have found that any deviations become strikingly salient and at times are highly significant. A few examples suffice:

1. *Young man with unexplained back pain.* Normally, the thickness of the intervertebral disks increases as one proceeds from the upper to the lower lumbar reaches. This patient's disk between the second and third lumbar vertebrae was barely perceptibly thinner than the one just above. The radiologic suspicion of early tuberculous spondylitis was eventually confirmed. This continuum of the lumbar intervertebral disks becomes less helpful the older the patient because of the frequent presence of degenerative changes.

2. *Middle-aged man with known coronary arteriosclerosis.* For several months the shadow merging into the posterior aspect of the ventricular mass in the lateral view of the chest had been considered part of the descending thoracic aorta. Appreciation of the continuum of the descending thoracic aorta in the lateral view indicates that normally there is no hiatus between the proximal and distal descending thoracic aorta (as demonstrated by this patient), but rather, a progressive decrease or constant maintenance of the density of the aorta, according to the patient's age, as one proceeds from arch to diaphragm. Hence, this shadow was not esophagus, an opinion confirmed by angiography, but most likely a ventricular aneurysm with thrombus, or a pericardial cyst.

3. *Child with clinical diagnosis of pneumonia in the right base.* The lungs roentgenologically were clear except for a little disturbance in the right retrophrenic continuum. In the properly exposed posteroanterior study of the chest, the lung behind the right (and left) hemidiaphragm presents as a gradually increasing density traversed only by vascular shadows as one proceeds distally to the pleural reflection. Any disturbance of this continuum must be explained, and the common explanation of

this common finding is pneumonitis. Further evidence for such pneumonitis will often be found in the disturbance of the continuum of the thoracic spine in the lateral view. Normally, the thoracic spine appears to become less dense as one proceeds toward the diaphragm because of diminishing muscle mass. With a posterior gutter pneumonitis, as suggested by disturbance in the retrophrenic continuum in the posteroanterior view, an abnormal increase in density will be noticed in the vertebral segments just above the diaphragm, as well as obliteration of the posterior-most segment of the involved hemidiaphragm (the positive silhouette sign).

4. *Middle-aged woman with symptoms compatible with gallbladder disease.* The first oral cholecystogram disclosed a concentration of the contrast medium at the lower limits of normal, but a barely perceptible diminution of density as one approached the fundus—a reversal of the findings in the normal gallbladder with the patient upright. Additional study permitted the definition of many small stones lying in the fundus.

Continua abound, and just await the resourceful radiologist's formulation and utilization.

Christian V. Cimmino, M.D., F.F.R.

Mary Washington Hospital
Fredericksburg, Virginia

HOW RADIOLOGY BECAME ESTABLISHED IN UTAH

ESTABLISHMENT of x-ray facilities was somewhat later in Utah than in other parts of the United States, and somewhat disorderly. Someone, in 1902, purchased an x-ray machine; I have not been able to learn the name of the individual, or under whose direction it was operated.

The second machine was purchased, late in 1902, by Dr. Augustus C. Behle. It was a huge static machine and took from 5 to 10 minutes to make an exposure.[1] At about the same time machines were purchased by other physicians, among them Doctors A. J. Hosmer, E. E. Root and S. H. Pinkerton. Dr. Pinkerton did most of his work at St. Mark's Hospital and, at his suggestion, Dr. J. C. Landenberger studied up on x-ray work. The machine was installed at St. Mark's Hospital and the staff was allowed to use it. St. Mark's Hospital, therefore, appears to have been the first hospital in Utah to have x-ray equipment. It is worthy of note that it also was the first hospital to be established in Utah and the first to graduate a three year class of nurses.

About 1906 Doctors Pinkerton and Landenberger moved to the newly-completed Judge Mercy Hospital, left St. Mark's Hospital, and took the equipment with them.

St. Mark's Hospital purchased its own equipment in 1907 and Dr. Warren Benjamin was given charge of the department. He graduated from the University and Bellevue Hospital Medical College, New York, in 1900 and died at age 56 of heart disease, April 27, 1927. At the suggestion of Dr. Augustus Behle, who travelled extensively in medical centers in Europe, he was persuaded to make a fluoroscopic examination of the stomach using a bismuth and buttermilk preparation. He quickly became disenchanted with the procedure when the patient vomited the mixture.

Louis A. Thody[2] gives the most factual and detailed account of early radiology in Utah. He was trained as an electrician in the Woolwich Arsenal, England, and came to Utah, as a convert to the Church of Jesus Christ of Latter Day Saints, in 1907. Because of his training in electricity, he was placed in charge of electrical equipment in the W. H. Groves Latter Day Saints Hospital, and, as part of those facilities, of the x-ray apparatus that had many problems, not the least of which was failure to produce satisfactory results.

The equipment consisted of a 10 inch coil and an electrolytic interrupter with a D'Arsonval high frequency resonator and 2 Leyden jars. There were 8 or 10 gas tubes of various makes. The tube stand consisted of a wooden pole set in a metal base on casters and the tube was attached to the post with a sliding wooden clamp. The department was located in the basement adjoining the corridor leading to the laundry and garbage depository. The walls were painted black and the light source was a single chandelier and a small window at ground level. There was no dark room; the plates were sent to a downtown photographer for development. When an x-ray examination was required, one of the 2 interns at the Hospital obtained the key to the department and, with the attending physician and patient, waited for the appearance of Mr. Thody. Then the plates were sent downtown for development. This procedure was totally unacceptable, so Mr. Thody established a "dark room," where processing was carried out in enamel trays with closely guarded secrets of chemical formulae, developing time, etc. Ultimately, he used an electrical "rocking table," called a "titulator," which saved much time and effort on the part of the technician.

It was necessary to have a number of tubes, each selected for a particular type of examination. Tubes were classified as soft, medium soft, medium hard and hard. A common method for testing tubes was to fluoroscope the operator's hand. A soft tube showed the hand entirely black and

[1] BEHLE, W. H. Biography of Augustus C. Behle with an Account of the Early History of St. Mark's Hospital. Edwards Bros. Inc., Ann Arbor, Mich., 1948.

[2] THODY, L. A. Personal communication.

was useless for examination. A medium soft tube showed the fleshy parts light green, the bones dark gray, and a metal ring black. A medium hard tube showed the bones light gray and a hard tube showed the bones lighter—the photography was thin and faded as compared with metal. The fluorescence of the tube was also used to indicate its condition. A soft tube gave a mixed blue and green color; a medium soft tube, a uniform dark green; a medium hard tube, a pale gray; and a hard tube, a yellowish to green tint, frequently also showing a reverse current. Mr. Thody notes that several static machines in downtown offices were idle (circa 1908–10), because a few x-ray burns had been reported and one serious injury occurred when a University janitor used his bare hands all day to demonstrate the mysteries of fluoroscopy.

The second physician-radiologist in Utah was Dr. Charles Wesley Stewart who graduated in medicine from Denver College of Medicine in 1900 and died in Burbank, California on March 29, 1927 at the age of 83. In 1918 he was associated with Dr. Albert Soiland, who was then serving on active status with the Naval Reserve. Dr. Stewart established a general practice in Sugarhouse, a suburb of Salt Lake City, and subsequently established and equipped an x-ray office "up town," at the same time continuing his private general practice. Later he restricted his practice to x-ray work and became director of the x-ray department of Holy Cross Hospital. As radiologist of Holy Cross Hospital, he was succeeded by Dr. James P. Kerby who, during World War I, had been serving with the U. S. Medical Corps at Fort Douglas Military Reservation, Salt Lake City. Dr. Kerby was the first formally trained radiologist in Utah, having studied from 1917–18 at the Military School of Roentgenology, Johns Hopkins Hospital, Baltimore, Maryland. He died in January 1952 after a career which embraced private practice in 5 Salt Lake City hospitals, a private office and Veterans' Administration out-patient department.

The first Snook machine was installed in Judge Mercy Hospital, under direction of Doctors Pinkerton and Landenberger, and, subsequently, similar equipment was put in use in Latter Day Saints Hospital and the Salt Lake Clinic—all under the instigation of Mr. Thody.

Looking back to the early years of radiology in Utah, it becomes apparent that Louis A. Thody, erstwhile electrical engineer, was largely responsible for pioneering and developing the specialty. When physicians in the community questioned his ability to function in the capacity of technician/interpreter of x-ray images (circa 1915 there were no specialists in radiology), he studied with Drs. Russell Carman at the Mayo Clinic, Henry Pancoast and W. R. Manges. As a result he became, although a "layman," an astute evaluator of the different shades of gray portrayed originally on the glass plate, and subsequently on the film. When it became apparent that radiology was becoming a specialty reserved for physicians, he formed an association with Q. B. Coray, M.D., who returned to Utah after completing a residency in radiology at Washington University, St. Louis, in 1926. He finally retired in 1946. In February 1911, the Salt Lake County Medical Society dedicated its program to "X-Ray Night." The participants on the program were Doctors Warren Benjamin, St. Mark's Hospital, E. Viko, Holy Cross Hospital and Louis A. Thody, Latter Day Saints Hospital. In recognition of his services, Mr. Thody was elected an Honorary Member of the Utah State Medical Association and, a few years later, of the Salt Lake County Medical Society.

Since the late 1930s, radiology has become a field for diplomates of the American Board of Radiology, whose names, at this time of writing have become legion and, lest some worthy of mention be omitted, none will be named.

Angus K. Wilson, M.D.

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NEWS ITEMS

AMERICAN SOCIETY OF NEURORADIOLOGY

The American Society of Neuroradiology Annual Meeting was held in Boston in April 1973.

The following are the officers elected for the coming year: *President*, T. Hans Newton, M.D., San Francisco, California; *President-Elect*, H. L. Baker, Jr., M.D., Rochester, Minnesota; *Vice-President*, William Hanafee, M.D., Los Angeles, California; *Treasurer*, John Allcock, M.D., London, Ontario, Canada; and *Secretary*, David O. Davis, M.D., Washington, D. C.

MOUNT ZION HOSPITAL AND MEDICAL CENTER

The San Francisco Cancer Symposium of 1973, presented by the Claire Zellerbach Saroni Tumor Institute of Mount Zion Hospital and Medical Center, San Francisco, California, will be held in the Stanford Ballroom, at the Stanford Court Hotel, November 16–17, 1973, from 8:30 A.M. to 5:00 P.M.

The subject is "Current Concepts in Breast Cancer and Tumor Immunology."

The Program Chairman is Joseph R. Castro, M.D., Chief, Department of Radiation Oncology, Mount Zion Hospital and Medical Center; Clinical Professor of Radiation Oncology, University of California Medical Center, San Francisco.

Deadline for advanced registration is November 8, 1973.

For further information please contact: Department of Continuing Education, Mount Zion Hospital and Medical Center, P.O. Box 7921, San Francisco, California 94120.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE

The 18th Annual meeting of The American Institute of Ultrasound in Medicine will be held in Ann Arbor, Michigan, October 14–18, 1973.

The program will include a full day of educational lectures and video tape in all disciplines of medicine.

For further information, please contact: Dr. Michael A. Wainstock, Towsley Center, University of Michigan Medical Center, Ann Arbor, Michigan 48104.

DIAGNOSTIC ULTRASOUND PHYSICIANS TRAINING COURSE

The purpose of this program is to provide a basic indoctrination for physicians interested in establishing an active program of diagnostic ultrasound in their own hospitals.

Seminar presentations, demonstrations and work-shops will embrace all of the 4 types of information display used in diagnosis. Some of the speakers will be: Gilbert Baum, M.D., Barry Goldberg, M.D., Stuart Gottlieb, M.D., Raymond Gramiak, M.D., Morris Kotler, M.D., Marc Lapayowker, M.D., George Leopold, M.D., J. Stauffer Lehman, M.D., William McKinney, M.D., Gordon Perlmutter, M.D., Bernard Siegel, M.D., Renate Soulen, M.D., William Steel, M.D., Marvin Ziskin, M.D. and others.

The dates of these courses are: September 17–21, 1973; December 10–14, 1973; March 25–29, 1974 and May 20–24, 1974, at Episcopal Hospital, Philadelphia, Pennsylvania, Section of Diagnostic Ultrasound, Department of Radiology.

Enrollment is limited to a maximum of 15 physicians for each course.

For further information please write to: Barry B. Goldberg, M.D., Program Director, Section of Diagnostic Ultrasound, Department of Radiology, Episcopal Hospital, Front Street and Lehigh Avenue, Philadelphia, Pennsylvania 19125.

SIXTH L. H. GRAY CONFERENCE

The organization is now underway of the 6th L. H. Gray Conference, on "The Initial Shapes of Cell Survival Curves: Clinical and Theoretical Implications."

The Conference will take place at Bed-

ford College, Regent's Park, London, England, September, 16–21, 1974.

It is hoped that the meeting will be of interest to radiotherapists as well as to radiobiologists.

The organizers have prepared a list of topics which they envisage as the subject matter, and they plan to arrange the program to some extent in accordance with the availability of relevant results.

The list may be obtained from Dr. Tikvah Alper, Medical Research Council, Experimental Radiopathology Unit, Hammersmith Hospital, London, W12 OHS, England.

AMERICAN OSTEOPATHIC COLLEGE OF RADIOLOGY

The 1974 Annual Scientific Meeting of the American Osteopathic College of Radiology will be held at the Century Plaza Hotel, Los Angeles, California, October 7–10, 1974.

The Program Chairman—1974 is Lawrence Jordon, D.O., 803 Delene Road, Jenkintown, Pennsylvania 19046.

For further information please contact: Charles Ballinger, D.O., Suite 21, Ponce American Building, 4601 Ponce de Leon Boulevard, Coral Gables, Florida 33146.

LEO G. RIGLER LECTURE, TEL-AVIV UNIVERSITY, TEL-AVIV, ISRAEL

The 13th Annual Leo G. Rigler Lecture, of the Tel-Aviv University will be given in Tel-Aviv, Israel, on October 23, 1973 by Dr. John Caffey on "The Shaken Babe: Radiographic Changes in the Skeleton and Central Nervous System." This will be followed on October 24th by a symposium on Bone Dysplasias. On this occasion the inauguration of the Leo G. Rigler Teaching Archive will take place.

For further information write Dr. Samuel Schorr, Ichilov Hospital, Tel-Aviv, Israel.

BOOK REVIEWS

ATLAS OF ROENTGENOGRAPHIC MEASUREMENT. Third edition. By Lee B. Lusted, M.D., Professor of Radiology, The University of Chicago; and Theodore E. Keats, M.D., Professor and Chairman, Department of Radiology, University of Virginia School of Medicine. Cloth. Pp. 300, with 250 figures. Price, about $16.00. Year Book Medical Publishers, 35 East Wacker Drive, Chicago, Ill. 60601, 1972.

Credit should be given to the authors for continuing to update their valuable atlas, the first edition of which appeared in 1959. In general, the format of this book is the same as the previous editions. The order of chapters has not been changed, but the material has been significantly updated and many new tables have been included. Whenever available, newer, more accurate, or more detailed material has replaced data which appeared in older editions.

The book itself represents an accumulation of many tables and statistical material published in the medical literature of recent years. The borderlines of normal are not always remembered. The authors undertook the effort to compile the pertinent and more important data which represent the wide range in size and form of normal structures. Combining this important, but usually unaccessible, material into a single volume resulted in an excellent source of reference.

The book is divided into 9 chapters which cover the central nervous system, the neck, skeletal system, respiratory system, cardiovascular system, gastrointestinal system, spleen and adrenal glands, urinary tract and pelvimetry, and fetometry. By far the longest chapter is the one dealing with the skeletal system. It comprises over half the book. Because of the many diseases and syndromes which may affect the skeletal system, one is particularly grateful to be able to refer with ease to tables and illustrations which might help in the recognition of these frequently somewhat obscure entities. It would have been nice to include tables and diagrams describing the different more common fractures and their nomenclature. Possibly such material could be added in future editions.

This is a reference book which is intended to be used when there is doubt in the interpretation of roentgenograms. Each topic is discussed on a separate page and references are listed. The interested reader may refer to the original work.

It is a book which should be available in all radiology departments and offices and will prove of immense value to the individuals using it.

P. RUBEN KOEHLER, M.D.

THE ESSENTIALS OF ROENTGEN INTERPRETATION. Third edition. By Lester W. Paul, M.D., Professor of Radiology, the University of Wisconsin Medical School; and John H. Juhl, M.D., Professor of Radiology and Chairman of the Department of Radiology, the University of Wisconsin Medical School. Cloth. Pp. 1,144, with 1,323 illustrations. Price, $35.00. Harper & Row, Publishers, Inc., 2350 Virginia Avenue, Hagerstown, Md. 21740, 1972.

The authors of a book detailing the basic features of diagnosis in clinical radiology are faced with a formidable task. It must fill the gap between the short elementary text introducing a student to the clinical contributions of the specialty of radiology and the multivolume or reference work of a particular area of diagnostic radiology. It must be concise and yet not superficial, comprehensive and yet not laboriously detailed. It must emphasize the differential features of diagnosis without forsaking an appreciation of normal variants.

This work, now in its third edition, fulfills these goals admirably. Sections devoted to the osseous system (10 chapters), the brain and spinal cord (2 chapters), the abdomen and gastrointestinal tract (7 chapters), the urinary and female genital tracts (2 chapters), the chest (11 chapters), and the face, mouth and jaws (3 chapters) have been updated and revised with the addition of new material covering advances in the last decade. Balance is achieved through a strikingly consistent approach. Entities as diverse as fracture and the mucopolysaccharidases, or duodenal ulcer and disaccharidase deficiency are presented clearly. The frequent inclusion of the incidence of signs or features in various diseases gives the reader an idea of their diagnostic reliability. Most of the 1,323 illustrations clearly show the pathologic features, although some could be improved with

labelling. References have been carefully selected, citing both classic articles and recent contributions. A 40 page index provides immediate access to the breadth of material covered.

Such a work imposes by its very nature a limitation on the discussions of clinical and pathologic aspects to the information necessary to clarify the roentgen observations. Understandably, not all entities are illustrated. However, several major conditions could profit immeasurably from being illustrated. The description of lobar and segmental atelectasis, for example, deserves more than 2 reproductions.

A critical reader of special interests may find areas of lapses or incomplete coverage. But what is, after all, to be considered "essential" is a matter of judgment and experience, and the authors have contributed notably in this regard. It is recognized that the changing nature of radiology has been determined to a large degree by the valuable diagnostic information provided by special procedures. Brief but concise descriptions of the applications and diagnostic potential of these advances in the specialty of radiology are also included as essential information.

This comprehensive book is equally suited to the radiology resident, both at the beginning and as a review survey at the end of his training period, and to the clinician and surgeon in training or practice as a basic reference and review source.

Morton A. Meyers, M.D.

BOOKS RECEIVED

The Radiology of Joint Disease. By D. M. Forrester, M.D., Associate Professor of Radiology, University of Southern California School of Medicine, Los Angeles County-University of Southern California Medical Center, Los Angeles, Calif.; Consultant Physician in Rheumatology, Long Beach Veterans Administration Hospital, Long Beach, Calif.; and John W. Nesson, M.D., Instructor in Radiology, University of Southern California School of Medicine, Los Angeles County-University of Southern California Medical Center, Los Angeles, Calif. Volume 2 in the Series, Saunders Monographs in Clinical Radiology. Cloth. Pp. 474, with 554 illustrations. Price, $22.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

The Portal Venous System. By Charles Marks, M.D., M.S., Ph.D., F.R.C.P., F.R.C.S., F.A.C.S., F.C.C.P., F.A.C.C., Professor of Surgery, Louisiana State University School of Medicine; Attending Surgeon, Charity Hospital, Touro Infirmary and Hotel Dieu Hospital; Consultant Cardiovascular Surgeon, East Jefferson and Methodist Hospitals, New Orleans, La.; Former Hunterian Professor, Royal College of Surgeons of England. With an introduction by Isodore Cohn, Jr., M.D., D.Sc., F.A.C.S. Cloth. Pp. 137, with some illustrations. Price, $12.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

The Use of X-Ray Techniques in Forensic Investigations. By Daniel Graham, F.S.R., Group Superintendent Radiographer to the Victoria Infirmary of Glasgow; Honorary Consultant Radiographer to Glasgow Police. Cloth. Pp. 142, with many illustrations. Price, $13.50. The Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, exclusive U. S. agents, 1973.

Atlas of Hand Radiographs. By Philip Jacobs, T.D., D.M.R.D., F.R.C.P., F.F.R., Consultant Radiologist, Royal Orthopaedic Hospital, Birmingham, Birmingham General Hospital, Birmingham Accident Hospital, and Warwickshire Orthopaedic Hospital for Children; Clinical Lecturer in Radiology, University of Birmingham, England. Cloth. Pp. 246, with 300 illustrations. Price, $24.50. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1973.

Hospitals—a Systems Approach. By Raymon D. Garrett. Cloth. Pp. 171, with some figures. Price, $12.50. Auerbach Publishers Inc., 121 North Broad Street, Philadelphia, Pa. 19107, 1973.

Planning a Diagnostic Radiology Department: Basic Considerations. By William G. Terry, DSR, SX-RayT, Associate Member, Faculty of Radiologists (Honorary), and J. W. McLaren, F.R.C.P., F.R.C.P.Ed., F.F.R., D.M.R.E., Sometime Physician in Charge of Diagnostic Radiology, St. Thomas' Hospital, London; Lately Examiner in Radiology, University of Edinburgh; University of London, Faculty of Radiologists and Conjoint Board. Cloth. Pp. 207, with some illustrations. Price, $22.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

Exercises in Diagnostic Radiology. Volume 6: Nuclear Radiology. By A. Everette James, Jr., Sc.M., M.D., Associate Professor of Radiology and Director, Radiological Research, Department of Radiology, The Johns Hopkins Hospital, Baltimore, Md.; and Lucy Frank Squire, M.D., Professor of Radiology, Downstate Medical Center, Brooklyn, N. Y.; Consultant in Radiology, Mas-

sachusetts General Hospital, Boston, Mass. Paper. Pp. 218, with 261 illustrations. Price, $5.95. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

Soft Tissue Roentgenography in Diagnosis of Thyroid Cancer: Detection of Psammoma Bodies by Spot-Tangential Projection. By Masayoshi Akisada, M.D., Chief, Radiology Department, Mitsui Memorial Hospital, Tokyo; Assistant Professor, Department of Radiology, Faculty of Medicine, University of Tokyo, Tokyo; and Yoshihide Fujimoto, M.D., Assistant Professor, Second Department of Surgery, Faculty of Medicine, University of Tokyo, Tokyo. Cloth. Pp. 138, with 47 figures. Igaku Shoin Ltd., 5-29-11 Hongo Bunkyo-Ku, Tokyo, Japan, 1973.

Health Practice Research and Formalized Managerial Methods. By F. Grundy, M.D., M.R.C.P., D.P.H., Assistant Director-General, World Health Organization, 1961–1965; and W. A. Reinke, Professor, Department of International Health, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Md. Public Health Papers No. 51. Paper. Pp. 193. Price, $3.00. The American Public Health Association, Inc., 1015 Eighteenth Street N.W., Washington, D. C. 20036, 1973.

Mercury, Mercurials and Mercaptans. Edited by Morton W. Miller and Thomas W. Clarkson, Department of Radiation Biology and Biophysics, School of Medicine and Dentistry, The University of Rochester, Rochester, N. Y. Cloth. Pp. 386, with some figures. Price, $19.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

A Text-Book of X-Ray Diagnosis: by British Authors. Volume III. Fourth edition. Edited by S. Cochrane Shanks, C.B.E., M.D., F.R.C.P., F.F.R., Consulting Radiologist, University College Hospital, London; and Sir Peter Kerley, K.C.V.O., C.B.E., M.D., F.R.C.P., F.F.R., D.M.R.E., Consulting Radiologist, Westminster Hospital and National Heart Hospital, London. Cloth. Pp. 728, with 526 illustrations. Price, $30.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

Golden's Diagnostic Radiology. Laurence L. Robbins, M.D., Editor. Section 15: Pediatric Radiology. Second edition. By Lawrence A. Davis, M.D., Professor of Radiology, The University of Louisville Medical School; Department of Radiology, The Children's Hospital, Louisville, Ky.; and Loretta T. Shearer, M.D., Assistant Professor of Radiology, The University of Louisville Medical School; Associate Radiologist, The Children's Hospital, Louisville, Ky. Cloth. Pp. 293, with many figures. Price, $27.50. The Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, exclusive U. S. agents, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Queen Elizabeth Hotel, Montreal, P. Q., Canada, September 25–28, 1973.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Surfer Hotel, Maui, Hawaii, April 21–25, 1974.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 25–30, 1973.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Roosevelt Hotel, New Orleans, La., April 1–6, 1974.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Chicago, Ill., June 15–19, 1974.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel; New York, N. Y., June 10–14, 1974, New York Hilton Hotel; and Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers in June 1974.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1974 is September 30, 1973.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Chairman, Dr. Merrill A. Bender, Suite 2402, 211 E. 43rd St., New York, N. Y. 10017.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, D. John Wright, Ph.D., Department of Radiology, Temple Univ. Health Sciences Center, Philadelphia, Pa. 19140. Annual Meeting.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting: Royal Sonesta, New Orleans, La., Oct. 24–28, 1973.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting: Ann Arbor, Mich., Oct. 14–18, 1973.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907. Meeting: Feb. 16–21, 1975.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Meeting in June, 1975 in Edinburgh, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Richard H. Greenspan, Dept. of Radiol., Yale University School of Medicine, 333 Cedar St., New Haven, Conn. 06510. Twenty-second Annual Meeting, New York, N. Y., May 9–11, 1974. Dr. Norman E. Chase is in charge of arrangements, New York Medical Center, New York, N. Y. 10016.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Charles Masters, Maj. MC, USAF Hospital, Wiesbaden, APO New York 09220.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Giacomo Pantaleo, 69-13 Forest Ave., Brooklyn, N. Y. 11227. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Ru-Kan Lin, 191 North St., Buffalo, N. Y. 14201. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. Lohn L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P.O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer, Dr. William H. Roush, Radiology Service, Veterans Administration Hosp., Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Lee Radford, 6011 Harry Hines, Suite 900, Dallas, Tex. 75235. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Fall meeting at Cloister, Sea Island, Ga., Sept. 15–16, 1973.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Stanley I. Worton, 1400 N.W. 10th Ave., Miami, Fla. 33136. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Raymond W. Brust, Jr., St. Francis Hosp., 2260 Liliha St., Honolulu, Hawaii 96817. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Shirley D. Vickers, Catholic Medical Center of Brooklyn and Queens, 88-25 153rd St., Jamaica, N. Y. 11432. Annual Meeting: Houston, Texas, June 16–20, 1974.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional

Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Gabriel H. Wilson, UCLA Center for Medical Sciences, Los Angeles, Calif. 90024. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert T. Cook, Southern Baptist Hosp., 2700 Napoleon Ave., New Orleans, La. 70115.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. D. S. O'Brien, Dept. of Radiol., Anne Arundel General Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Joseph Ferrucci, Jr., Massachusetts General Hosp., Boston, Mass. 02114.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. A. G. Fleurquin, Pontiac General Hosp., Pontiac, Mich. 48053. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Howard L. Copas, 827 South 131st St., Omaha, Neb. 68154. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89520.

New England Roentgen Ray Society
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting: May 10, 1974.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 25–27, 1974. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 S. University Dr., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 2100 Rockwood Dr., Sacramento, Calif. 95825. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald W. Philips, 2305 Washington, Milwaukie, Ore. 97222. Meets on second Wesnesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., E. Penn and Wister St., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
President-Secretary, Dr. Arch W. Templeton, Univ. of Kansas Med. Ctr., Kansas City, Kan. 66103. Meets 5 times a year on given dates.

Radiological Society of Louisiana, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Hanson, 116 Nayatt Rd., West Barrington, R. I. 02806.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 1974.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. Robert M. Maurer, Santa Rosa Medical Center, San Antonio, Tex. 78207. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President, James R. Collins, 340 Fourth Ave., Suite 15, Chula Vista, Calif. 92010. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, Mary's Help Hospital, 1900 Sullivan Ave., Daly City, Calif. 94015.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Chairman, Dr. Henry W. Wiggins, Jr., Mercy Hosp., 2537 South Prairie Ave., Chicago, Ill. 60616. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Annual meeting: Queen Elizabeth Hotel, Montreal, P. Q., Canada, September 23–24, 1973.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting.

South Bay Radiological Society
Secretary, Dr. Virgil H. Voss, 900 Kiely Blvd., Santa Clara, Calif. 95051. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. John Byfield, 1000 West Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Carl T. Stubblefield, Bedford Country Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neil, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205. Annual Meeting: La Mansion Hotel, San Antonio, Tex., March 21–23, 1974.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada. Annual meeting.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John F. Stucki, Department of Radiology, McKay-Dee Hosp., 3939 Harrison Blvd., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 8952 Bellfonte Rd., Richmond, Va. 23229.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 9110 Seven Locks Rd., Bethesda, Md. 20014.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Isaiah J. Seligman, 16 Guion Pl., New Rochelle, N. Y. 10801. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, Gundersen Clinic, LaCrosse, Wis. 54601. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Francisco Miranbell S., Apartado 3352, San José, Costa Rica. Thirteenth Central American Congress of Radiology, San José, Costa Rica, Dec. 4–8, 1973.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. T. Kerr, 32 Welbeck St., London, W1M/7PG, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, 1555 Summerhill Ave., Montreal 109, Que., Canada. Thirty-seventh Annual Meeting, Regency Hyatt House, Vancouver, B. C., April 21–26, 1974.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

SOCIÉTÉ CANADIENNE-FRANÇAISE DE RADIOLOGIE
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

THE HOSPITAL PHYSICISTS' ASSOCIATION
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

THE ROYAL AUSTRALASIAN COLLEGE OF RADIOLOGISTS
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

SOUTH AMERICA

COLÉGIO BRASILEIRO DE RADIOLOGIA
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Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

SOCIEDAD ARGENTINA DE RADIOLOGÍA
Secretary-General, Dr. Tomás J. Gutiérrez F., Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. Tomas Strauszer, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

SOCIEDAD COLOMBIANA DE RADIOLOGIA
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

SOCIEDAD ECUATORIANA DE RADIOLOGÍA
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SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

SOCIEDAD PERUANA DE RADIOLOGIA
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

SOCIEDAD DE RADIOLOGÍA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

SOCIEDAD DE RADIOLOGÍA DEL NORDESTE ARGENTINO
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SOCIEDAD DE RADIOLOGÍA DE LA PLATA
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SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

SOCIEDADE DE RADIOLOGÍA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

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Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

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Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

SOCIEDAD SALTEÑA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

SOCIEDAD VENEZOLANA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

CONTINENTAL EUROPE

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SOCIÉTÉ ROYALE BELGE DE RADIOLOGIE
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

SOCIÉTÉ EUROPÉENNE DE RADIOLOGIE PÉDIATRIQUE
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Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

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Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

SOCIÉTÉ FRANÇAISE DE NEURORADIOLOGIE
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting: Salpetrière, Paris, Dec. 1, 1973.

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Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

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Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa 1-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

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Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Japanese American Radiological Society
Secretary, Thomas J. Imray, Maj. MC, Chief, Radiol. Dep., US Army Hosp., Camp Zana, Japan, APO San Francisco, Calif. 96343. Meets 3rd Thursday, monthly at various locations in Tokyo.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

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Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Head

Willich, E., Sellier, W., and Weigel, W. Die intrakraniellen Verkalkungen des Kindesalters. (Intracranial calcifications in childhood.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, June, 1972, *116*, 735–750. (Address: Priv.-Doz. Dr. E. Willich, Universitätskinderklinik, Hofmeisterweg 1–9, 69 Heidelberg, Germany.)

A review of 34,941 skull roentgenograms of children of 3 pediatric hospitals revealed 293 (0.83 per cent) cases with intracranial calcifications. The most common causes were: toxoplasmosis (22 per cent); infectious processes other than toxoplasmosis (16 per cent); Sturge-Weber syndrome (7.8 per cent); intracranial hemorrhage (7.5 per cent); tumors (7.2 per cent); and tuberous sclerosis (4 per cent). Less common causes were perinatal brain damage, convulsive disorders, vascular calcification, neurofibromatosis, and pseudohypoparathyrodism. No cause of the intracranial calcification was found in about one-fourth of the cases.

The calcifications can be divided into the following 4 groups: (1) intracranial calcifications that are diagnostic alone or in conjunction with other skull roentgenographic findings (long standing subdural hematoma, craniopharyngioma, and Sturge-Weber syndrome); (2) intracranial calcifications that allow a diagnosis with the knowledge of additional clinical findings (tuberous sclerosis, toxoplasmosis, cytomegalic inclusion disease, listeriosis, and tuberculosis); (3) intracranial calcifications that can be clarified only with supplementary studies such as cerebral angiography, pneumoencephalography, skeletal roentgenography or electroencephalography (tumors, intracerebral bleeding, seizures, and pseudohypoparathyroidism); and (4) intracranial calcifications which remain nondiagnostic after extensive roentgenographic work-up.—*Peter F. Winter, M.D.*

Whatmore, W. J. Sincipital encephalomeningoceles. *Brit. J. Surg.*, April, 1973, *60*, 261–270. (From: Department of Surgical Neurology, The Royal Infirmary and Western General Hospital, Edinburgh, Scotland.)

Encephalomeningoceles are congenital herniations of cerebral tissue through the skull. They are usually composed of a meningeal sac containing brain tissue, cerebrospinal fluid or both and are covered externally by skin and soft tissue. They usually pass through congenital defects in the skull and lie in the midline of the cranium between the occiput and the nasofrontal region.

Cranial encephalomeningoceles occur much less frequently than spinal encephalomeningoceles. In the Western world the occipital site is more common, while in certain countries of the East the anterior encephalocele is more frequent.

Anterior encephalomeningoceles consist of 2 types: (1) sincipital—near the root of the nose; and (2) basal—within the nose, pharynx or orbit. The sincipital form is subdivided into nasofrontal, nasoethmoid and naso-orbital varieties.

The author reports a detailed analysis of 30 cases of sincipital encephalomeningoceles studied in Burma. The age of the patients was from a few days to 39 years. There were 19 males and 11 females. The age of the parents at the patients' birth was probably significant, as 36 parents were over 30 years of age and 13 parents were over 40 years. A swelling at birth was present in 29 of the 30 cases. In 25 of the cases the lesion was progressively enlarging.

The most common group was the nasofrontal form of sincipital encephalomeningocele (21 cases), followed by the nasoethmoid (8 cases) and naso-orbital (1 case) varieties.

The lesions varied from a slow growing small, hard nasofrontal or nasoethmoid mass to a rapidly growing massive tumor which assumed an "elephant-trunk" form and produced visual, breathing and feeding disorders. Some tumors were cystic, translucent and fluctuant. The swellings were nontender on palpation, but would often become tense with coughing or straining. None of the cases could be reduced by compression of the mass. A skull defect could sometimes be palpated at the base of the lesion.

Roentgenograms of the skull including tomograms were useful to show the presence, size, shape and location of a skull defect. A circular or elliptical bone defect from 1.5 to 2.5 cm. in diameter was found in the 11 patients who had an intracranial operative procedure. In 8 of the cases the bone defect was in the midline at/or adjacent to the foramen cecum and in 3 cases the defect was lateral to the anterior cribriform plate.

Lumbar air encephalography was done to reveal accompanying hydrocephalus. Air did not enter the encephalomeningocele. No direct injections into the sac were attempted.

Surgery is the only effective treatment for suitable lesions and should be done as early as feasible. Intracranial surgical procedures are described by the author.

The pertinent literature concerning etiology, pathogenesis and embryogenesis of sincipital encephalomeningocele is reviewed.—*Paul M. Dassel, M.D.*

Larson, Steven M., Johnston, Gerald S., Ommaya, Ayub K., Jones, A. Eric, and di Chiro, Giovanni. The radionuclide ventriculogram. *J.A.M.A.*, May, 1973, *224*, 853–857. (Address: Gerald S. Johnston, M.D., 9000 Rockville Pike, Bethesda, Md. 20014.)

A review of 70 radionuclide ventriculograms in 47 patients is presented. The studies were performed in patients with implanted ventriculostomy tubes or cerebrospinal fluid (CSF) shunts. Early studies were performed with radioiodinated serum albumin (RISA). More recent studies are performed with high specific activity serum albumin labeled with technetium 99m (Tc^{99m} HSA). Sequential gamma camera views were obtained.

An explanation of the different types of shunts and their physical features is presented. The indications for these studies as well as the general findings are presented in both narrative and tabular form.

The indications include: (1) shunt function; (2) cerebrospinal fluid dynamics; (3) distribution of intrathecal medications; and (4) tumor progression.

The findings under these general headings include: (1) patent or obstructed shunts and the location of the obstruction; (2) dilatation of ventricles and localization of third ventricle rhinorrhea; (3) obstructions to CSF pathways; and (4) tumor masses and encroachments or obstruction of ventricular system.

One bias that is pointed out by the authors is that the patient population included very few children, so that obstructive hydrocephalus which is a relatively common indication for CSF shunting in children is represented only by a small number of patients.

Some general conclusions about CSF dynamics are made in the nonobstructive group of patients even though this is not a "normal" population: (1) injection into one lateral ventricle will fill the contralateral ventricle; (2) a significant fraction of intraventricular nuclide descends into the subarachnoid space indicating that the entire subarachnoid space is a distribution space for the medications; and (3) a time interval of approximately 2 hours is required for equal distribution between the ventricles and the basilar cisterns, which is consistent with other assessments of normal clearance half-life from the ventricles.—*Donald P. Harrington, M.D.*

Neck and Chest

Powell, David, Murray, Timothy M., Pollard, James J., Cope, Oliver, Wang, Chiu-An, and Potts, John T., Jr. Parathyroid localization using venous catheterization and radioimmunoassay. *A.M.A. Arch. Int. Med.*, May, 1973, *131*, 645–648. (Address: David Powell, M.B., Department of Biochemistry, Trinity College, Dublin 2, Ireland.)

The results of preoperative localization of parathyroid secreting sources utilizing large vessel venous sampling and radioimmunoassay for parathyroid hormone in hyperparathyroid patients are reported in 50 cases.

The difficulty of diagnosing hyperparathyroidism as well as the localization of the causes such as adenoma or hyperplasia are pointed out. Patient selection, catheterization procedure, and hormone assay techniques are described.

A patient with a left lower lobe parathyroid adenoma is used to illustrate the venous sampling findings; these indicated a higher level of PTH in the proximal left innominate. The results were confirmed surgically in that the parathyroid adenoma was removed from the left lower gland.

The over-all findings in 50 cases showed localization of abnormal PTH-secreting tissues achieved in 28 (56 per cent). No false-positives were found.

Surgical or postmortem findings are given in 38 of 50 patients. Three patient summaries are given to indicate the usefulness of the procedure. These include the diagnosis of hyperparathyroidism in hypercalcemic patients as well as the localization of the source of high PTH secretion.

Reference is also made to other works where selective catheterization of thyroidal veins has produced higher localizing figures.—*Donald P. Harrington, M.D.*

Skucas, J., Rossi, Plinio, Brinker, Ray A., and Eberle, Robert C. Angiographic differentiation of neurogenic tumors at the carotid artery bifurcation. *Surg., Gynec. & Obst.*, July, 1973, *137*, 35–39. (From: Departments of Radiology and Surgery, St. Vincent's Hospital and Medical Center, New York, N. Y.)

Masses in the lateral portion of the neck may be caused by aneurysm, enlarged lymph nodes, metastasis or extension of carcinomas, branchial cysts, or neurogenic tumors, particularly chemodectoma or neurilemmoma. Angiographic differentiation of the last 2 is specific because of their characteristic vascular patterns.

Chemodectoma is sometimes called carotid body tumor, and is usually unilateral, although unsuspected contralateral tumors are occasionally encountered, particularly when there is a family history of chemodectoma. The tumor is usually a slowly growing painless mass. Malignancy is established by the clinical course rather than by the microscopic appearance of the tumor.

The tumor commonly originates between the internal and external carotid arteries, and separation of the vessels is shown frequently in both frontal and lateral projections. Both arteries are usually displaced laterally. Part of the blood supply may arise from other adjacent vessels, even though the feeding vessel is usually the external carotid artery. The tumor vessels are irregular, mottled, and poorly defined, with the production of a densely staining, nonhomogeneous, relatively well circumscribed tumor, with no early venous filling. The tumor may encase either the

internal or external carotid artery, but does not produce occlusion.

Chemodectomas may arise at other sites in the neck, but will produce the same characteristic vascular pattern.

Neurilemmomas arise from nerve sheaths, and are sometimes called schwannomas. They are usually solitary; although they may arise in any portion of the body, there is a predilection for the neck. They usually present as a painless mass. If they arise from an appropriate position in relation to the internal and external carotid arteries, there may be similar displacement and separation of the 2, but this is less likely than with a chemodectoma. The principal differentiation occurs with the vascularity of the tumors. Neurilemmomas are much less vascular, even though some tumor vessels may be present. There is no definite capillary stain, as there is with chemodectoma. In none of the neurilemmomas did the capillary vascularity approach that of even the least vascular chemodectoma. There is no hypertrophy of the feeding vessels in neurilemmomas.

Angiographic examination thus will permit differentiation of these 2 neurogenic tumors, as well as distinguishing them from other neoplasms in this location.—*E. Frederick Lang, M.D.*

Halpern, S., Alazraki, N., Littenberg, R., Hurwitz, S., Green, J., Kunsa, J., and Ashburn, W. ^{131}I thyroid uptakes: capsule versus liquid. *J. Nuclear Med.*, July, 1973, *14*, 507–510. (Address: Samuel E. Halpern, Division of Nuclear Medicine, University Hospital of San Diego County, 225 West Dickinson Street, San Diego, Calif. 92103.)

Eleven volunteers had iodine 131 uptakes on 2 occasions: one after receiving a capsule of iodine 131 NaI, the other after receiving a dose of iodine 131 NaI liquid.

All uptakes were determined in the same manner, by the same examiner, at 9 intervals from 15 minutes after a dose to 24 hours. Differences in uptake were noted in all subjects, at almost all measurements.

The study performed following administration of liquid NaI was usually higher, by 5–10 per cent, than that following capsule administration. This finding is possibly explained by the observation that digestion of the capsule produces products that are labeled with iodine 131, and may be unavailable for absorption.—*William H. Strauss, M.D.*

Kimura, Eiichi, Ichikawa, Akira, Takahashi, Koichi, and Kusachi, Nobunori. Abnormal respiration and unusual movements of the diaphragm in patients with neurocirculatory asthenia. *Am. J. M. Sc.*, March, 1973, *265*, 205–214. (Address: Eiichi Kimura, M.D., Nippon Medical School, 1-1-5 Sendagi, Bunkyo, Tokyo 113, Japan.)

Respiratory abnormalities in neurocirculatory asthenia (NCA) were analyzed by spirometry and cineroentgenography of diaphragmatic movement. Spirometric analysis revealed irregular intervals of the respiratory cycle, nonuniform amplitude of respiration, nonlinearity of the level of the respiratory curve, and a sighing pattern in NCA. The acute type of NCA showed an increase in the amplitude of respiration as well. Cineroentgenograms revealed irregular periodicity, nonuniform magnitude of the diaphragmatic movement, superimposition of smaller movements on the larger movements, chaotic and disorganized movement of the diaphragm and fluctuation in baseline level of the diaphragmatic movement.

The abnormal movements of the diaphragm as shown in the present study may have unfavorable effects on the respiration and hemodynamics as well as a potential role in the origin of the gastrointestinal symptoms of patients with NCA.

It is proposed that the abnormal diaphragmatic movement may be the primary disorder in NCA and may cause subjective complaints.—*David Morse, M.D.*

Gonzalez, Leopold, and Van Ordstrand, Howard S. Wegener's granulomatosis: review of 11 cases. *Radiology*, May, 1973, *107*, 295–300. (Address: Leopold Gonzalez, M.D., Department of Radiology, Cleveland Clinic, Cleveland, Ohio 44106.)

Three types of necrotizing angiitis are categorized according to histologic criteria. These are polyarteritis nodosa, hypersensitivity angiitis, and Wegener's granulomatosis. The authors discuss in detail their findings in 11 patients with Wegener's granulomatosis.

The etiology of Wegener's granulomatosis is unknown. It is thought that Wegener's granulomatosis is an autoimmune disease with the initial insult being an inflammatory or infectious process. There is then a secondary autoimmune reaction to the involved destroyed tissue. The authors' series consisted of 8 men and 3 women. Age range was from 25 to 59 years. The most common occurrence is in the 4th and 5th decades. Teenage occurrence is unusual, although it has been reported.

Clinically the involvement is usually in the upper respiratory tract, lungs and kidneys. The authors' series showed 6 patients with cavitary pulmonary nodules. The appearance of the nodules is variable with the cavitation either central or eccentric and with the walls of the cavity usually thick and shaggy, although there were some cavitary lesions with smooth walls. No calcification was identified in any of the patients.

Case reports of 4 patients are described in some detail.

Differential diagnosis of pulmonary nodules discussed by the authors includes polyarteritis nodosa (variable miliary), allergic granulomatosis, pyemic abscesses (multiple lesions rarely larger than 5 cm.), coccidioidomycosis (thin-walled cavities), hematogeneous metastases (lower lobe predilection), rheumatoid nodules (lung periphery), and Hodgkin's disease (air bronchograms present).—*J. P. Eberts, M.D.*

Inkley, Scott R., and MacIntyre, William J. Measurement of regional area gas exchange by perfusion and clearance of ^{133}Xe from the lung. *J. Nuclear Med.*, July, 1973, *14*, 490–495. (Address: Scott R. Inkley, Department of Medicine, Case Western Reserve University, Cleveland, Ohio 44106.)

The lung's performance as a gas exchange organ can be measured by following the distribution of injected xenon 133 in the lung, and evaluating its subsequent washout. The initial distribution of injected xenon is a measure of the perfusion and its subsequent diffusion into the alveolar space, the subsequent washout is a measure of ventilation.

The examination is performed by placing the patient upright against the detector of a scintillation camera, and recording the distribution of activity in a 1600 channel analyzer. The data are analyzed in a computer, where the counts at equilibrium following intravenous injection are determined (perfusion); the slope of the half time of washout from each is determined (ventilation) and the ventilation and perfusion are multiplied by each other to give a third parameter, the regional area gas exchange (RAGE).

The information is displayed as a series of 3 dimensional images, where height is proportional to RAGE. Three patients had 5 studies by both differential bronchospirometry and RAGE. A good correlation was found in all.—*William H. Strauss, M.D.*

Bosnjakovic, Vladimir B., Bennett, Leslie R., Greenfield, Larry D., and Vincent, William R. Dual-isotope method for diagnosis of intracardiac shunts. *J. Nuclear Med.*, July, 1973, *14*, 514–521. (Address: Leslie R. Bennett, Nuclear Medicine Division, University of California, Center for the Health Sciences, Los Angeles, Calif. 90024.)

Isotope angiocardiography using 2 tracers has been evaluated in 25 patients, 7 without shunts, 4 with right to left shunts, and 14 with left to right shunts.

The 2 tracers were employed to identify the cardiac chambers of the right heart separately from those of the left. Xenon in saline was used to identify the chambers of the right heart, and technetium labelled sulfur colloid to identify the chambers of both the right and left heart.

All patients were examined in the modified left anterior oblique position (LAO with 10–15° of caudad tilt). This position was felt to offer the best separation of all 4 cardiac chambers.

Information from the passage of each tracer through the heart was recorded and time activity curves from regions of interest in the heart analyzed. The curves were then evaluated for the presence of shunting.

The method correctly detected the presence of shunting in the 4 patients with right to left shunts, and in 11 of 14 patients with left to right shunts. The 7 normal patients were correctly identified as having no shunts.

These preliminary findings suggest that the method permits screening of patients for hemodynamically significant intracardiac shunts.—*William H. Strauss, M.D.*

Pinet, F., Amiel, M., Clermont, A., Rubet, A., and Froment, J.-C. Les dissections aortiques et leur bilan angiographique: à propos de 21 observations. (Aortic dissections and their angiographic study: report of 21 cases.) *J. de radiol., d'électrol. et de méd. nucléaire*, Nov., 1972, *53*, 787–792. (Address: Pr F. Pinet, Service de Radiologie, Hôpital Cardio-vasculaire et Pneumologique, 59, boulevard Pinel, 69003 Lyon, France.)

The authors used direct aortic angiography in the study of suspected dissecting aneurysms. Fifteen of the cases were in the emergency stage.

The necessity of a careful examination is stressed from the points of view of treatment (especially surgical) and prognosis. The tolerance of the patients is good, even in emergency cases, when the angiography is performed carefully. The catheter tip should be surveyed fluoroscopically as it progresses in the aorta and test injections should be performed to choose the level, as well as the start of injections.

The tip of the catheter is in the aortic lumen if the test injection causes a rapid diffusion of the contrast medium, whereas it is in the false lumen of the dissecting aneurysm if the diffusion is slow and cloudy.

The percutaneous femoral route is the usual approach, unless no femoral pulse is present or the aortic lumen is unattainable, in which case angiography is carried on through right humeral catheterization.

Angiography is performed at numerous levels, depending on the site, as well as extent of the lesion. Both oblique views are important, especially in helicoidal and posterior types of dissecting aneurysm; 35 mm. cineradiography is indispensable in the diagnosis of difficult cases such as aortic insufficiency.—*Jirair N. Sarian, M.D.*

Abdomen

Drut, Ricardo, Gimenez, Pedro O., and Vestfrid, Mario A. Laceraciones cardioesofágicas en pacientes comatosos; presentación de 3 casos. (Esophagogastric junction lacerations in comatose patients; report of 3 cases.) *Prensa méd. argent.*, Dec., 1972, *59*, 1827–1829. (From: Cátedra de Patologia I, Facultad de Medicina de La Plata, Argentina.)

This article reports 3 autopsy studied cases of mucosal lacerations occurring in comatose patients. The coma was due to: diabetes with meningitis; cerebral hemorrhage; and anoxia secondary to externally treated cardiac arrest.

It is considered that this Mallory-Weiss type lesion is due to a sudden increase in intraabdominal pressure (secondary to external cardiac massage, severe cough, nausea, vomiting, hiccups, or convulsions).

These mucosal tears produce hemorrhage of variable degree.—*Julian B. Alvarez, M.D.*

Turner, Robert, Lipshutz, William, Miller, Wallace, Rittenberg, Gerald, Schumacher, H. Ralph, and Cohen, Sidney. Esophageal dysfunction in collagen disease. *Am. J. M. Sc.*, March, 1973, *265*, 191–199. (Address: Robert Turner, M.D., Department of Medicine, Bowman Gray School of Medicine, Winston-Salem, N. C. 27103.)

A prospective cineradiographic and manometric study of pharyngeal and esophageal dysfunction was performed in 54 patients with rigidly defined scleroderma, polymyositis, systemic lupus erythematosus, and rheumatoid arthritis. Decreased or absent distal esophageal peristalsis was demonstrated by cineradiography in 55 per cent and manometry in 73 per cent of 22 patients with scleroderma, and 13 and 3 per cent, respectively, of 32 patients with other collagen diseases. This finding showed a highly significant correlation with clinically diagnosed scleroderma. The manometric abnormality was seen less frequently in patients with Raynaud's phenomenon unrelated to scleroderma. This difference was statistically significant.

When clearly defined criteria for the diagnosis of the skeletal muscle dysfunction of polymyositis were used, no false positive diagnoses were made and, of the 10 polymyositis patients, only 2 were diagnosed by manometry and 1 by cineradiography as having this disease.

This study thus demonstrated that: (1) distal esophageal smooth muscle dysfunction was relatively specific for clinical scleroderma; (2) significant pharyngeal and esophageal skeletal muscle dysfunction was an infrequent finding limited to polymyositis; and (3) manometry was somewhat more accurate and specific than cineradiography in diagnosing these changes.—*David Morse, M.D.*

Beaudin, D. J., DaCosta, L. R., Prentice, R. S. A., and Beck, I. T. Duodenoscopy in the diagnosis of upper gastrointestinal disease. *Canad. M.A.J.*, March, 1973, *108*, 565–569. (Address: I. T. Beck, M.D., Hôtel Dieu Hospital, Kingston, Ontario, Canada.)

Examination of 143 patients with 2 flexible fiberoptic duodenoscopes was carried out without complications. These were not volunteers, but symptomatic patients undergoing examination for upper gastrointestinal symptoms. The findings were correlated with both radiologic findings and histologic changes. Particular attention was given to those patients in whom radiographic or clinical diagnosis was changed as a result of the findings at duodenoscopy.

The instruments used were a Japanese side viewing scope and an American forward viewing scope. Both instruments were equipped for photography and biopsy. The forward viewing scope allowed visualization of the pyloric canal, the duodenal cap, and the proximal part of the duodenal loop, with the papilla of Vater rarely seen. The side viewing scope allowed viewing of the duodenal loop and papilla, but was not adequate for viewing of the pyloric canal.

The diagnosis of ulcers and polyps was easily made and agreed upon by 2 observers, but duodenitis was a problem. In 59 per cent of the examinations there was complete agreement between the roentgenographic and endoscopic diagnoses.

Five chronic duodenal ulcers were found in patients with negative roentgenograms and 8 cases of duodenitis were discovered. Duodenoscopy also seemed to exclude pathologic changes diagnosed roentgenographically in 20 other patients.—*Richard E. Kinzer, M.D.*

Ament, Marvin E., and Ochs, Hans D. Gastrointestinal manifestations of chronic granulomatous disease. *New England J. Med.*, Feb., 1973, *288*, 382–387. (Address: Marvin E. Ament, M.D., Department of Medicine, RG-20, University of Washington, Seattle, Wash. 98195.)

Chronic granulomatous disease is an inherited illness in which phagocytic cells are unable to kill certain bacteria which cause bacterial infections. A hydrogen peroxide-myeloperoxidose-halide system within the cell has been shown to be important for the killing of certain bacteria.

Granulocytes in patients with this disorder are not able to destroy *Staphylococci, Serratica marcescens* and gram-negative *enterococci*. These bacteria pro-

duce abscesses and granulomata throughout the body. Because diarrhea and perianal abscesses were observed in 20 per cent of these patients, the authors undertook to study the gastrointestinal tracts of their patients. Fecal fat excretion, bacterial cultures of intestinal contents, and small bowel and rectal biopsies were studied. Gastrointestinal studies were carried out on each patient.

All patients studied had gastrointestinal tract involvement. One had findings simulating Crohn's disease and was treated with good result with salicylazosulfapyridine.—*Richard E. Kinzer, M.D.*

SANE, SHASHIKANT M., and GIRDANY, BERTRAM R. Total aganglionosis coli: clinical and roentgenographic manifestations. *Radiology*, May, 1973, *107*, 397–404. (Address: Shashikant M. Sane, M.D., Department of Radiology, Children's Health Center, Inc., 2525 Chicago Avenue, Minneapolis, Minn. 55404.)

In total aganglionosis coli (TAC), the entire colon and often a portion of the distal small bowel demonstrate a complete absence of ganglion cells. The mortality rate in this condition is high; therefore, early diagnosis and surgical treatment are important.

The authors report 13 cases of total aganglionosis coli out of a series of 202 infants with Hirschsprung's disease seen over a 20 year interval at the Children's Hospital of Pittsburgh. Diagnosis was confirmed in every case by microscopic examination.

To analyze the pertinent clinical and roentgenographic features of the entity, the authors combine their 13 cases with 128 previously reported in the literature for a total of 141. Not all the cases in the literature had both clinical and roentgenographic findings reviewed; therefore, the percentages are not necessarily a portion of all 141 cases.

The most common presenting symptoms, occurring in 73 per cent, were abdominal distention and vomiting. Delay in passing meconium or constipation presented in 30 per cent. Vomiting or abdominal distention alone, alternating diarrhea and constipation, or diarrhea alone, were less common. The disease occurred mostly in early infancy, with 81 per cent of the patients being less than 1 month old at the time of diagnosis.

The plain roentgenographic findings were reviewed in 72 cases in the literature. In only 4 of these was the bowel gas pattern normal. In the others, there was moderate to marked small bowel distention (with or without large bowel), showing air-fluid levels on erect roentgenograms. In 76 cases, the findings of contrast enema examination were reported. Of these, 38 per cent had microcolon, meaning any colon with small caliber lumen. In 22 per cent, the colon was considered normal. In 21 per cent, contrast material was retained over a prolonged period. In 15 per cent, there was an apparent transition zone which was actually well below the zone of true transition.

The clinical and roentgenographic findings in the authors' cases correlated closely with those reported in the literature.

The incidence of congenital anomalies in patients with TAC is slightly higher than in normals. In patients with TAC, there is a greater familial incidence of Hirschsprung's disease than in other patients with Hirschsprung's disease.

The mortality rate is high. Of the 141 cases in the literature, follow-up was available in 117, and only 32 per cent were alive. In the authors' series, 5 of 10 cases seen since 1955 have survived. Treatment consists of surgical resection of all the aganglionic bowel.

A full term infant with signs of small bowel obstruction, especially with a familial history of Hirschsprung's disease, should alert physicians to the possibility of TAC.

The roentgenographic studies are not diagnostic. Surgical exploration and biopsy are necessary for the diagnosis.—*J. M. Stoebner, M.D.*

DARKE, S. G., PARKS, A. G., GROGONO, J. L., and POLLOCK, D. J. Adenocarcinoma and Crohn's disease: a report of 2 cases and analysis of the literature. *Brit. J. Surg.*, March, 1973, *60*, 169–175. (From: The London Hospital, London, England.)

Incidence of carcinoma without regard to duration of ulcerative colitis or to age group is about 4 per cent. Death from cancer may be 10–30 times more frequent in patients with ulcerative colitis than in the general population.

The authors have attempted to show that there is also a small, but significant risk of carcinoma in Crohn's disease. Perrett and Truelove, writing in May 1968 issue of the *British Medical Journal*, note a frequency of 3.7 per cent in Crohn's disease. They indicate that this is considerably higher than the chance incidence of carcinoma.

Two cases are reported in this article: 1 carcinoma occurring in the terminal ileum; and 1 in the rectum. The captions on the illustrations are transposed, but the cases presented are rather convincing in spite of the fact that Case II might actually be ulcerative colitis rather than Crohn's disease.

Carcinoma in Crohn's disease is difficult to diagnose, especially when one is dealing only with small bowel disease.

The conclusion is that the risk of carcinoma developing in Crohn's disease is present and one should be on the look-out for it.—*Richard E. Kinzer, M.D.*

GALLOWAY, SHELBY J., CASARELLA, WILLIAM J., and SEAMAN, WILLIAM B. The nonoperative treatment of retained stones in the common bile duct. *Surg., Gynec. & Obst.*, July, 1973, *137*, 55–58. (From: Department of Radiology, Columbia-Presbyterian Medical Center, New York, N. Y.)

In the past 20 years, after the routine use of operative cholangiography was introduced, the incidence of retained stones after cholecystectomy, as determined by T-tube cholangiography, has decreased from about 10 to 3 or 4 per cent. Since spontaneous passage of a retained stone is rare, and since most patients are or will become symptomatic in the presence of retained ductal stones, a safe, nonoperative approach seems desirable, to avoid reexploration, with its attendant risk.

In 1969 the first report of successful expulsion of a stone by manipulation through a T-tube tract appeared, and more recently, similar reports have been published. This communication describes 7 patients in whom manipulation was attempted.

After T-tube cholangiography disclosed the presence of one or more retained stones, the tube was permitted to remain in place for 3 to 6 weeks, to assure formation of a fibrous tract along the drainage tube. After this interval, and after a confirmatory cholangiogram, the manipulation was carried out in the X-Ray Department, under fluoroscopic control. The T-tube was removed, usually after passage of a guide wire, and a catheter was introduced along the fibrous tract into the common duct. Warm saline was used for irrigation, and contrast material was injected intermittently for visualization. Routine premedication has not been employed, but analgesics or anticholinergics may be useful. Occasionally the first attempt was unsuccessful; in this case the catheter was left in place for drainage, and the attempt continued the next day.

Different techniques have been found necessary and useful. Direct pressure on the stone with the end of the catheter may be successful, if the stone is fortuitously situated. Small angiographic catheters may be used instead of a larger, soft rubber catheter. A Dormia stone basket was successfully employed through a rubber catheter in 1 patient. Forceful flushing aids in positioning the stones for other maneuvers.

In 5 of the 7 patients, satisfactory, uncomplicated removal was accomplished. One of the others presented with too small a fibrous tract along the T-tube. In high risk patients, anticipation of this possibility, and use of a sleeve of polyethylene tubing around the T-tube would permit manipulative nonoperative removal more readily if this became necessary. In the other patient, the retained stone was 1.5 cm. in diameter, and it could not be removed because of its size.

The patients have been followed for 6 months to 2 years after this procedure, and there is nothing to suggest a complication, such as a stricture or residual stones.—*E. Frederick Lang, M.D.*

duPriest, Robert W., Jr., Haines, James E., Rösch, Josef, and Krippaehne, William W. A comparison of scintiscans and arteriograms for identifying metastatic intrahepatic tumors. *Surg., Gynec. & Obst.*, May, 1973, *136*, 705–710. (From: Departments of Surgery, Radiology [Division of Cardiovascular Radiology], and Clinical Pathology [Division of Nuclear Medicine], University of Oregon Medical School, Portland, Oregon.)

Scintiscans and selective arteriograms were obtained in 57 patients with suspected metastatic intrahepatic tumors. All of the cases with a single exception were histologically proven.

The first question asked in this series was: Do focal defects suggest metastatic intrahepatic tumors? In this series of 57 scans there were 10 true positive scans and 33 true negative scans. Of the 57 patients, 15 had in fact metastatic intrahepatic tumors. There were then 5 false positives and 9 false negative scans. The over-all accuracy was 75.4 per cent.

The second question asked was: Can the scan be used as a screening method? Forty scans were evaluated as to being normal, mildly abnormal, moderately abnormal or definitely abnormal. If the cases which were moderately abnormal and definitely abnormal were considered positive, there were 14 true positive and 6 true negative scans, but 19 false positive and 1 false negative scans. The accuracy was 50 per cent.

The accuracy of arteriography was determined in 57 arteriograms. There were 18 true positive and 37 true negative with 1 false positive and 1 false negative. The over-all accuracy was 96.5 per cent.

Comparisons were made between the authors' data and previously published series of liver scans and arteriography. The accuracy obtained by scans in other series was higher than in the present series, while the arteriographic detection rates were lower in reported series, when compared to the series under consideration.

The conclusion from this series is that scanning as a screening method is good with only one false negative but many false positives, and that arteriography is the most accurate single test. The evidence further indicates that there is no 100 per cent accurate test and that a combination of tests must be used, and their use must be judged in each individual case.—*Donald P. Harrington, M.D.*

Robins, Jon M., Bookstein, Joseph J., Oberman, Harold A., and Fajans, Stefan S. Selective angiography in localizing islet-cell tumors of the pancreas: a further appraisal. *Radiology*, March, 1973, *106*, 525–528. (From: Departments of Radiology, Pathology, and Internal Medicine, The University of Michigan Medical Center, Ann Arbor, Mich.)

Widely divergent statistics on the incidence of positive angiograms in islet cell tumor of the pancreas have appeared in the recent literature. In-

dividual series and general reviews of the subject claim positive studies in 38 to 100 per cent of cases, with most recent reports claiming conservative estimates of 60 to 70 per cent. The authors present a timely review of their series, confirming their original conservative prediction of the incidence of positive angiograms in cases of insulin-producing islet cell tumors of the pancreas.

Twenty-eight of 33 patients with clinical and laboratory evidence of hyperinsulinism have undergone pancreatic angiography at the University of Michigan Medical Center since 1965. Twenty-seven of the 28 patients underwent celiotomy and islet cell tumors were found and confirmed in 26 patients. One patient was not subjected to celiotomy because of angiographic evidence of liver metastases. Twenty-one patients had single adenomas and 5 had multiple adenomas, with the number of adenomas in each of 5 patients being 2, 4, 2, 5, and 6. Therefore, the base data consist of 40 histologically confirmed adenomas in 26 patients studied angiographically. Thirty-six tumors were histologically benign. The data were then analyzed for size, degree of vascularity, location, and type of examination and correlated with the frequency of angiographic detection.

The results indicate that 15 of 40 tumors were localized by preoperative angiography (38 per cent) and at least 1 tumor was correctly localized in 14 of 26 patients (54 per cent). These lesions were recognized angiographically by a pronounced, more or less discrete, disproportionate opacification during the parenchymal phase, persisting only 2 to 6 seconds. There was a clear correlation between angiographic localization and tumor size. Of 9 tumors 2 cm. or more in diameter, 7 were localized; of 15 that were 1 to 2 cm. in diameter, 5 were localized; of 7 which were 0.35 to 0.99 cm. in diameter, 3 were localized; of 9 less than 0.3 cm. in diameter, none was localized. Areas of adenomatosis, present in 3 patients, were not angiographically demonstrated. The data also indicate correlation between the degree of vascularity seen histologically and the frequency of angiographic localization. Location of the tumor is also important angiographically, since of 15 tumors in the pancreatic head 9 were angiographically visualized, while only 3 of 8 were seen in the body and 3 of 17 in the tail of the pancreas.

Therefore, tumor size, histologic vascularity and location within the pancreas are very important angiographic detection parameters, although location within the pancreas is likely a reflection of size since a progressive decrease in average tumor size was noted in the head, body and tail of the pancreas. Of 24 tumors over 1 cm. in diameter 12 were demonstrated, while in 16 less than 1 cm. in diameter only 3 were localized.

The authors emphasize that a tumor should be demonstrated in 2 projections, and the displacement of the lesion relative to known anterior posterior arteries can be used to localize the depth of the lesion within the pancreas. Evanescent blushes seen in one projection only must be interpreted with caution since false positive areas can be produced both by lymph nodes and small accessory spleens. Also, localized postoperative pancreatitis can produce discrete hypervascularity identical to an islet cell tumor.—*Edward B. Best, M.D.*

LEIGH, PETER G. A. Ruptured spleen: a radiological pitfall and a suggested modification. *Brit. J. Surg.*, April, 1973, *60*, 289–291. (From: Department of Surgery, University of Witwatersrand and General Hospital, Johannesburg, South Africa.)

The author suggests a modification of the Schorr and Danon method of detecting a space-occupying peri- or parasplenic mass, particularly in reference to rupture of the spleen. A distance greater than 3 cm. between the greater curvature of the barium opacified stomach and the lateral abdominal wall, as measured to the inner rib margin of the lower ribs, on a roentgenogram made in the left lateral decubitus position is abnormal according to Schorr and Danon (Schorr, S. and Danon J., AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *99*, p. 616).

A case is reported of a false positive diagnosis of delayed rupture of the spleen utilizing the Schorr and Danon technique because of interposition of a heavy omentum between the greater gastric curvature and the lateral abdominal wall; however, the correct diagnosis of absence of splenic rupture was ascertained by roentgenographic examination of the patient in the right lateral decubitus position with the stomach distended by gas. The thick omentum fell away and the gas distended stomach rose toward the left lateral abdominal wall in the right lateral decubitus position. An interposed fully loaded colon or a fluid-filled loop of small intestine could conceivably also produce a false-positive finding.

In another case in which delayed rupture of the spleen was suspected, the right lateral decubitus position was utilized after distending the stomach with gas and an operative procedure was avoided since this demonstrated unequivocally that there was no space-occupying lesion in the splenic area. This modified technique was used in several young patients who were suspected of having a splenic or other visceral injury in the acute phase.

The author proposes routine use of the right lateral decubitus position with the stomach distended with air prior to the Schorr and Danon method in evaluation for rupture of the spleen, and possibly as the only technique in cases in which it may be inadvisable to give barium orally. Adequate gaseous distention of the stomach is essential to avoid equivocal findings.

This method may be useful in diagnosis of disease of the spleen and in following progress of patients after treatment.—*Paul M. Dassel, M.D.*

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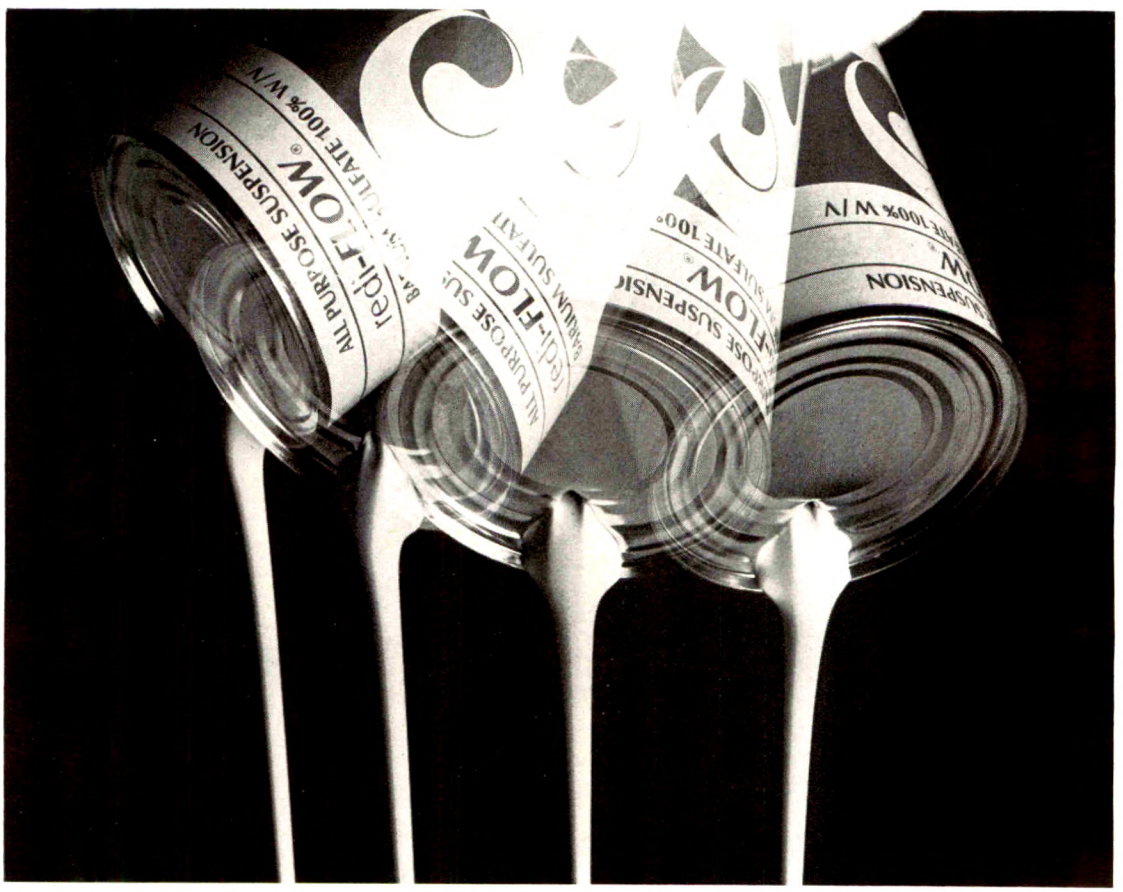
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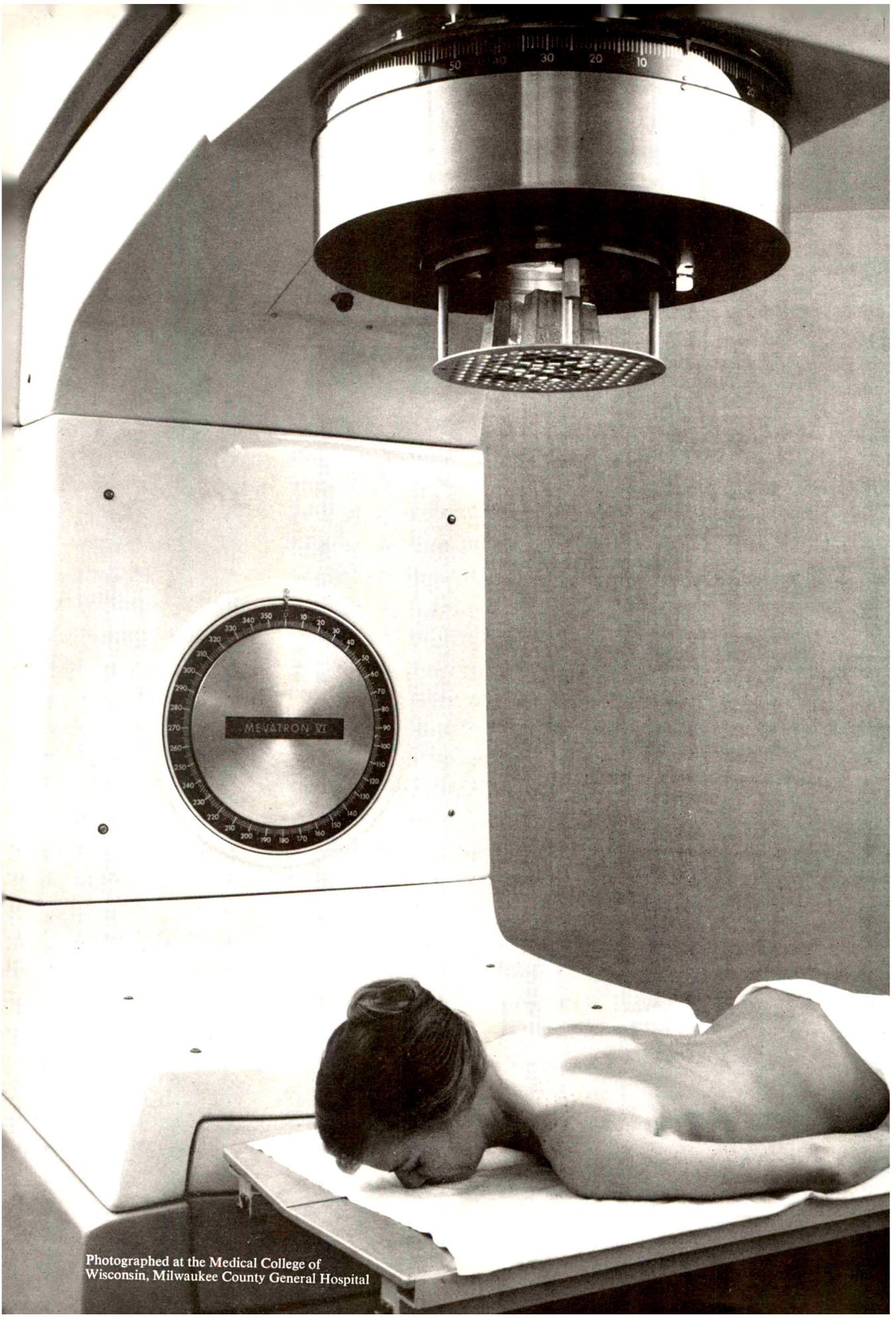
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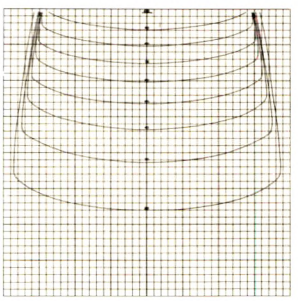
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The new TherX universal radiation therapy simulator is in a class by itself in roviding the highest degree of precision, versatility, and convenience. A unique feature is the 12-1/2" image intensifier-television system for viewing large treatment fields. Using diagnostic x-rays, TherX provides means for pinpointing tumors, confirming entry and exit ports, and verifying beam coverage at every phase of the treatment. Thus, the vital radiation beam/patient relationship can be studied



and trial setups made under conditions comparable to actual treatment. TherX operation is controlled at the patient's side, or remotely with console readout of all significant treatment factors. TherX is equally valuable as a teaching tool, or as an aid to the most sophisticated radiotherapist in developing comprehensive treatment plans for Co^{60}, linear accelerators, or in brachytherapy planning. Picker Corporation, 595 Miner Road, Cleveland, Ohio 44143. Images for health.

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Photo: Courtesy Boston Hospital for Women, and Joint Center for Radiation Therapy, Harvard Medical School



Now-one radiopaque for only one procedure

specifically designed for accurate well-tolerated retrograde cystourethrography

Specifically packaged. 250 ml. in calibrated 500 ml. bottle for dilution with sterile water or 5 per cent dextrose solution.

Complete in a single unit. For greater dilution capacity—calibrated bottle conveniently suited to 1 to 1 dilution. For flexible dosage range—concentration is easily adjusted over a wider range.

Well tolerated. No irritation or other undesirable effects have been reported following its administration in clinical investigations.*

*Data on file at Sterling-Winthrop Research Institute.

the specific for retrograde cystourethrography

New Hypaque-Cysto®

brand of

meglumine diatrizoate, USP

a logical addition to the expanding Hypaque® product line

New Hypaque-Cysto®

brand of
meglumine diatrizoate, USP

Sterile Aqueous Solution
For Retrograde Cystourethrography

Description: Hypaque-Cysto is a radiopaque medium which contains approximately 141 mg. organically-bound iodine per ml. It is a 30 per cent (weight/volume) sterile aqueous solution of the meglumine salt of 3,5-diacetamido-2,4,6-triiodobenzoic acid ($C_{18}H_{26}I_3N_3O_9$). The viscosity is 1.94 cps at 25° C. and 1.42 cps at 37° C. The pH is adjusted with hydrochloric acid or meglumine solution. Hypaque-Cysto is clear and colorless to pale yellow. It should be protected from strong light. The solution is relatively thermostable and may be autoclaved. Calcium disodium edetate 1:10,000 has been added as a sequestering stabilizing agent.

Action: Hypaque-Cysto is a diagnostic radiopaque medium which provides visualization of the lower urinary tract.

Indication: Hypaque-Cysto is indicated for retrograde cystourethrography.

Adverse Reactions: No irritation or other undesirable effects have been reported following the administration of Hypaque-Cysto in clinical investigations.

Dosage and Administration: After the bladder is emptied, Hypaque-Cysto is gently instilled without force, often beyond the first desire to micturate, but not beyond the point of urgency or mild discomfort. The volume required to fill the bladder to slightly less than capacity may vary from patient to patient.

Bladder capacity in normal adults is generally 200 to 300 ml., and rarely, up to 600 ml. Capacity at birth is 20 to 50 ml., and increases about 400 per cent in the first year. In children 3 to 5 years old, bladder capacity is 150 to 180 ml. In children older than 8 years, it is in the low adult range.

In disease, bladder capacity in adults may vary from 50 ml. in neurogenic "reflux" bladder to over 1000 ml. in autonomous bladder or chronic lower urinary tract obstruction.

Repeat examination may be required to detect reflux, or in function studies.

The concentration varies with technique and equipment used. Hypaque-Cysto may be diluted with sterile water or 5 per cent dextrose solution, as indicated in the following table.

To Make		Add	Final Solution Contains
Final conc.	Final volume	Sterile water or 5% dextrose solution	Iodine
30 %	250 ml.	—	141 mg./ml.
25 %	300 ml.	50 ml.	118 mg./ml.
21.4%	350 ml.	100 ml.	101 mg./ml.
20 %	375 ml.	125 ml.	94 mg./ml.
18.8%	400 ml.	150 ml.	88 mg./ml.
16.7%	450 ml.	200 ml.	78 mg./ml.
15 %	500 ml.	250 ml.	71 mg./ml.

How Supplied: Calibrated 500 ml. dilution bottles containing 250 ml. Hypaque-Cysto; rubber stoppered, with inner removable seal and screw neck.

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New York, N.Y. 10016

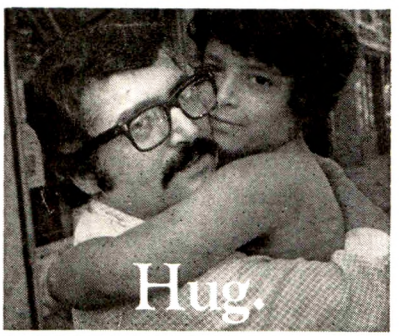


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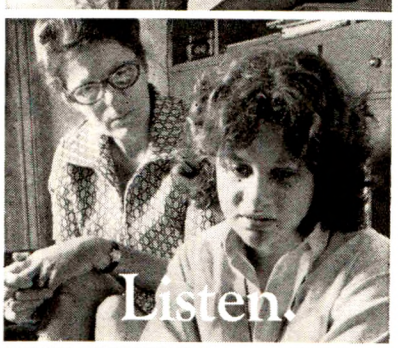
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If you're locked into the concept of a central darkroom for the time, we still can help. With the Independent II—a new, conventional processor that delivers 90% of your top capacity for 50% of the investment.

These two new film processors are another major American technological accomplishment. They are two of many new Profexray units, each built to a key concept that will speed a procedure, overthrow an outmoded way of doing things, innovate changes that can *revolutionize* your departmental efficiency.

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CROSS-FOGGING?

"Cross-fogging" is a term commonly used to describe the increased film fogging which occurs in simultaneous biplane angiography due to the fact that the film in each plane is hit by scattered radiation from two exposures. Cross-fogging can be reduced by using low kV, tight collimation, good grids or air gap between object and film. In addition, if you have a biplane SCHONANDER AOT-R installation, you can eliminate cross-fogging entirely by alternate program-loading of the film magazines. This technique gives you up to 15 films in each plane at exposure rates up to 3 exp./sec. in each plane. Thanks to the long film compression time in the AOT-R changer, ample exposure times are available even though the changers have to operate at 6 exp./sec. in order to give three alternate film pairs per second.



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Whole body scanning that provides diagnostic information with push button control.



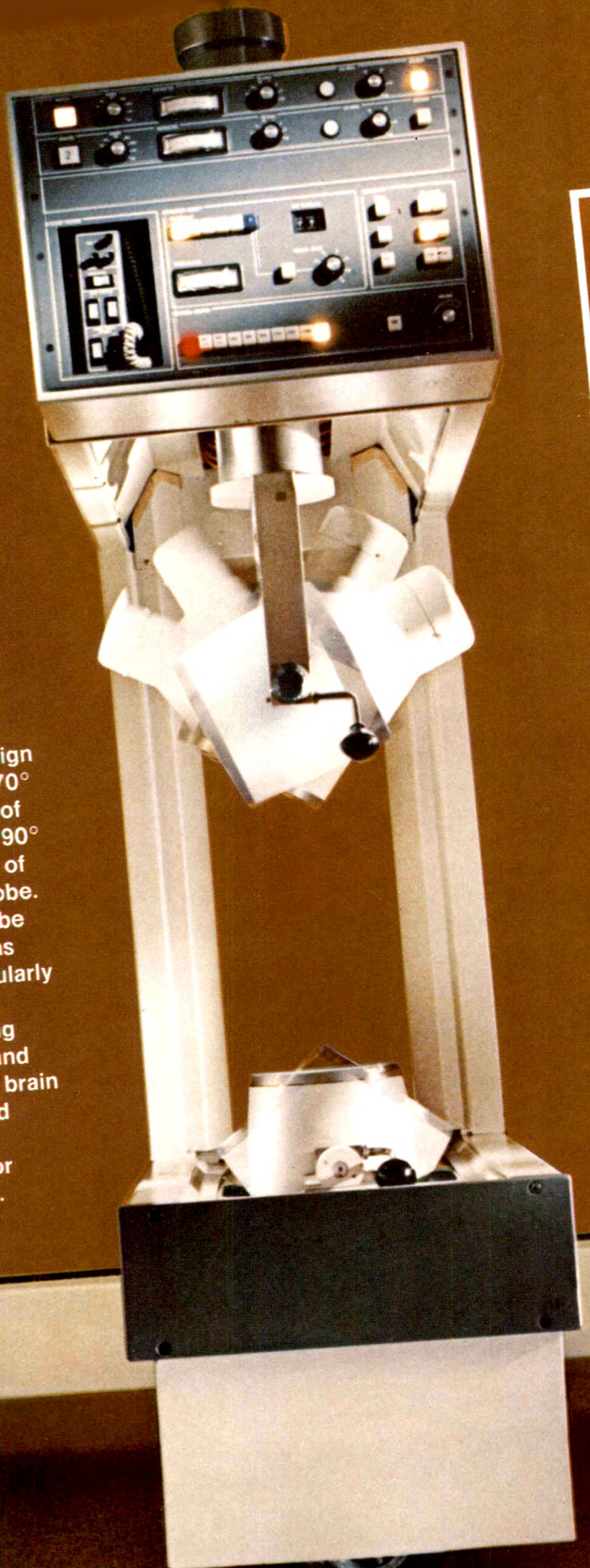
Situation: a vertex scan of the brain is required, but the patient's condition prevents propping up his head.

Situation: a skeletal survey is desired for early detection of bone metastases.

Situation: a suspected embolism in the lungs must be located, with an absolute minimum of patient movement.

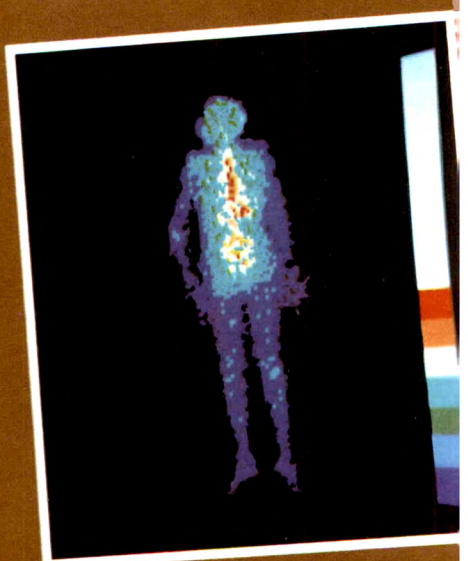
Each situation poses a diagnostic problem. The informational capability you need to solve them, and many more such problems, is automatically available with the push button programming of the General Electric Whole Body Scanner.

The GE Whole Body Scanner provides images head-to-toe, or anywhere between. Like this...



Unique design provides 270° angulation of top probe; 90° angulation of bottom probe. These probe angulations are particularly useful in performing anterior and posterior brain scans and facilitate collimator changes.





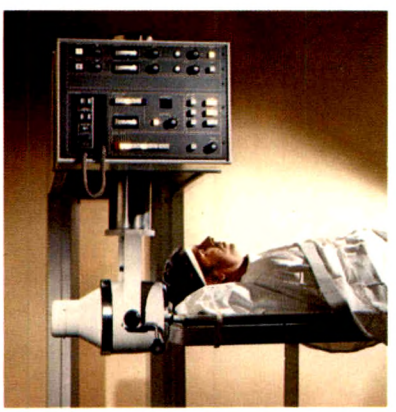
View scans in full count, fully functional color or in black and white with the addition of GE's Videodisplay and Processing Unit. Images, in eight distinct colors or shades of gray, are viewed on a TV monitor; can be manipulated, long after the patient leaves the department, to enhance desired details. Ask for full information.

Probe and table motions add new diagnostic technics; minimize patient movement.

More information. Less chance of technic error. The General Electric Whole Body Scanner combines the features that assure these diagnostic advantages. Automatically.

For whole body scans, the unit travels up to 80 inches longitudinally, 24 inches laterally. Push button settings let you select a full size photodisplay image or minified to 2:1, 3:1, 4:1 or 5:1. In addition to whole body scans, up to four images can be displayed on one 14 x 17 inch film. Location is push button programmed.

The scanner's two probes and three scanning directions permit scans of the entire lung, top and bottom; cover the total isoresponse curve without turning the patient. The top probe angulates 270°, and has a powered 12 inch vertical travel. With optional vertical plane scanning the patient can be seated upright. These probe angulations also permit a



vertex view of the brain while the patient reclines normally, without propping his head in an uncomfortable position.

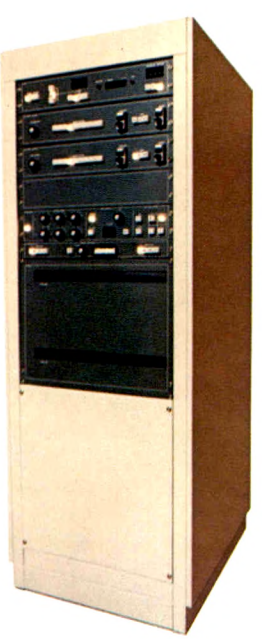
The GE Whole Body Scanner can also perform any other scanning technic possible with a single-probe unit; even lets you start with one probe, then upgrade to two as needs and the budget dictate.

For added procedural versatility, the patient can be placed on GE's mobile table, an accessory to the scanner, while controls are set at the electronics console. The table top raises and lowers.

With the patient in position, simply press the PHA button, locate the hot spot, set the line spacing and press the desired information density button . . . and scanning speed is displayed automatically. No calculations. Patient count information, during the procedure, can be read at the gantry face without leaving the patient's side; also, with the digital scaler at the electronics console.

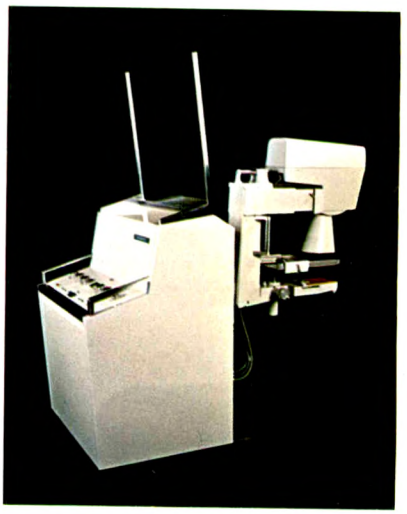
Let the GE Whole Body Scanner add this information depth to your department. Talk soon with your GE medical systems representative.

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Mammographic System Expands Function of Any Radiographic Room

The totally integrated MMX™ system combines, in one unit, everything needed for mammography to maximize diagnostic information. Simplifies preparation; saves set-up time; permits seated or supine views; provides positive compression of any size breast.

High Resolution Thermography for Diagnosis and Screening

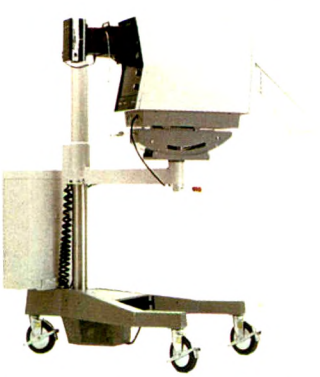
The Spectrotherm 1000 thermographic imaging system, marketed by General Electric, combines speed, simplicity and reliability for diagnosis and screening in the areas of breast cancer, incipient stroke, vascular diseases, arthritis and a variety of other medical applications.

The system features an infrared scanner, electronics and high resolution image display in a single, compact instrument. This integrated design permits the operator to view both the patient and the thermal image while operating the controls, and provides the ease of direct focusing.

A wide angle scan of the patient is taken in 2 seconds and imaged on a high resolution display consisting of 525 lines (with 600 elements per line). Directly below the thermal image is a graphic temperature profile providing accurate measurement of temperature variation for any given line of the image.

The Spectrotherm 1000 provides both automatic and manual brightness control to assure good images with minimal adjustment; ability to "freeze" the image on the display for on-screen evaluation; and a 0.2° C temperature sensitivity.

The thermal image, converted to a standard video signal, permits remote viewing on standard TV monitors and storage on video tape. The image can also be recorded with the system's basic camera, capable of using Polaroid pack film, 4 x 5 sheet film or 70 mm roll film.

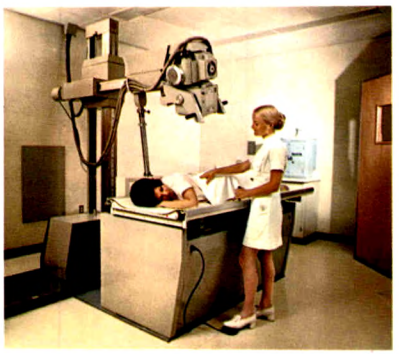


The system is mounted on a versatile stand and includes a lightweight, clip-on mirror for over-bed use.

Hospital Cites Advantages of R&T Procedures Room

One recently completed 137-room hospital installed a General Electric Maxitome™ dual procedures room as a back-up system, then promptly upgraded it to preferred status as the system's full capability became apparent.

The facility's chief technologist explained why: "The biggest thing about Maxitome is its ability to do routine radiographic procedures in addition to multi-directional tomography." As a result, he said, the hospital is doing many procedures with it that hadn't originally been counted on.



For Bucky-quality conventional radiography, Maxitome features collimation that's automatic or manual, or controlled from the remote station. And, manual masking is integrated into the table. Multiple film formats up to 9 on 1. The system also permits off-table technics with GE's vertical Bucky; and the tube unit can be angulated for oblique technics.

"The machine has capabilities we haven't even tapped yet."
—Chief Technologist

For tomography and zonography, the chief technologist said, "Maxitome's results, when compared with straight linear tomography, are just outstanding. We see so much more detail."

One reason is because Maxitome permits linear tomography in four different directions, in addition to pluridirectional patterns. This permits 90° angle sweeps across the area of interest to prevent shadows and avoid the common linear streaking on the long axis of the anatomy.

Maxitome provides 11 tomographic patterns in all, controlled with the turn of a dial. The desired plane can be adjusted in 1 mm increments, from 0-240 mm above the floating table top. Tomographic section thickness adjusts between 1 mm and 5 mm and the source-image distance is fixed at 43 inches.

Time-saving System Simplifies Reporting Procedure for Radiologists

One 420-bed hospital with "growing pains" recently sought relief for handling mounting radiological reporting loads.

The problem: more than 75,000 examinations now conducted yearly, and an annual load of 180,000 projected in three years. The solution: Raport™, radiological reporting system by General Electric.

Radiologists found the Raport system to be a means to speed the reporting procedure per examination and to reduce administrative time too.

"The Raport system should simplify everything for the clinician."
—Chief of Radiology

Proof is their significant decrease in report turn-around time. It's down from as long as 48 hours with the conventional dictation system to less than a minute with the Raport system, plus another two to four hours for distribution.

Key to this dramatic time saving is the Raport system's series of mark sense forms, covering examinations of areas of the anatomy. Like the radiographs they represent, mark sense forms segment the body into various anatomical combinations. The forms permit the radiologist to indicate the existence, location and type of pathology diagnosed by making a series of small pencil marks next to appropriate symbols on the form.

After indicating his diagnosis on the form, the radiologist is ready for another examination. Meanwhile, the form is "read" by an optical scanner, and its information is forwarded to the system's control unit. Here the data is decoded, correlated with pre-stored diagnostic phrases, constructed into proper sentences and sent to the automatic typewriter



for printing. Included on the report are patient and physician information, the radiologist report in text and the summary impression. Dictated addenda can also be incorporated for more complex diagnoses.

According to the hospital's chief of radiology, "The finished reports are very 'meaty', very little verbosity. They report what they have to say in three or four sentences, and they give an impression on every case. I think it should simplify everything for the clinician."

General Electric Medical Systems
Milwaukee, Toronto, Liege

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Service and Financing plans round out total performance concept

Total performance from General Electric incorporates service plans like Planned Maintenance Service (PMS). With a choice of options, PMS involves regular and complete scheduled inspections of all aspects of the systems covered, with emphasis on preventive care to avoid future problems. The program cost can be budgeted in advance, to protect against increases in the price of parts and labor during the term of the contract.

GE's Maxiservice® program not only covers full service needs, it also incorporates true-lease provisions, covers all parts and labor, emergency calls—even insurance. At a fixed monthly fee. Equipment is paid for out of revenue generated through usage. And, it's possible to upgrade equipment at any time, with GE assuming the risk of placing returned hardware. A full range of other Mind-Easer financing plans match specific requirements.

GE sales and service representatives are the men who make these service and financing programs work as an integral part of GE's total performance concept. They are prepared to make total performance a reality in any hospital across the country.

General Electric
Medical Systems,
Milwaukee, Toronto, Liege

Product Purchasing Guides:

Comprehensive product/ supplies catalogs

Two General Electric catalogs featuring x-ray supplies and nuclear medicine accessories provide a complete guide to purchasing, including proper ordering information.

The X-ray Supply catalog features film and processing supplies, together with items involved in their handling, storage and viewing. Other supplies and accessories required in radiography are also included.

The Nuclear Medicine and Non-imaging instrumentation catalog contains a product listing sufficiently broad to equip and maintain the nuclear laboratory. Described is an extensive range of diagnostic instrument systems, protective equipment, phantoms, tables and other nuclear supplies.

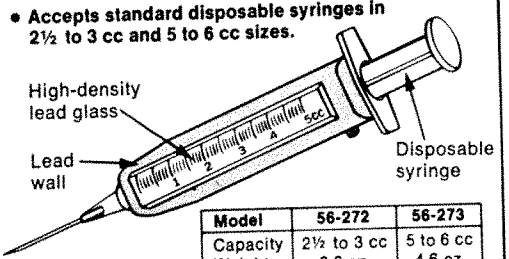
For your copy of the X-ray Supply and/or Nuclear Medicine catalogs, contact your General Electric representative.



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Weight	3.2 oz.	4.6 oz.
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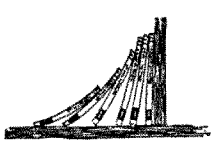
*U.S. Patent 3,596,659

For more details, ask for Bulletin 453-C

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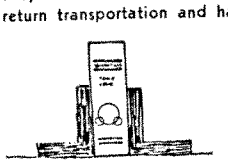
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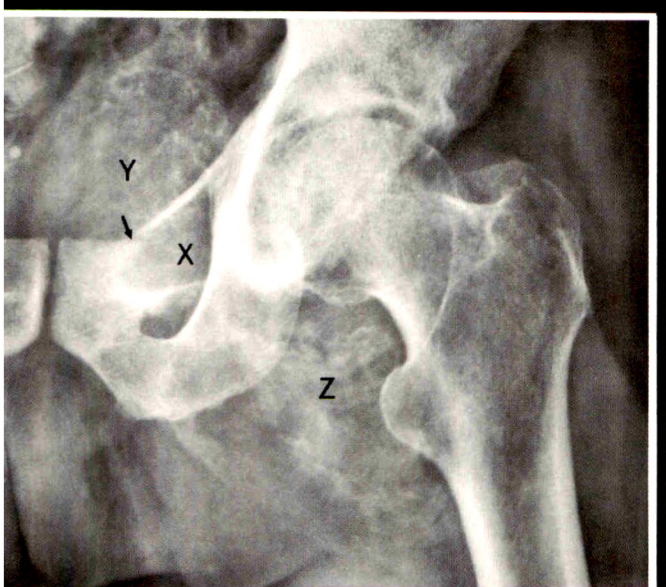
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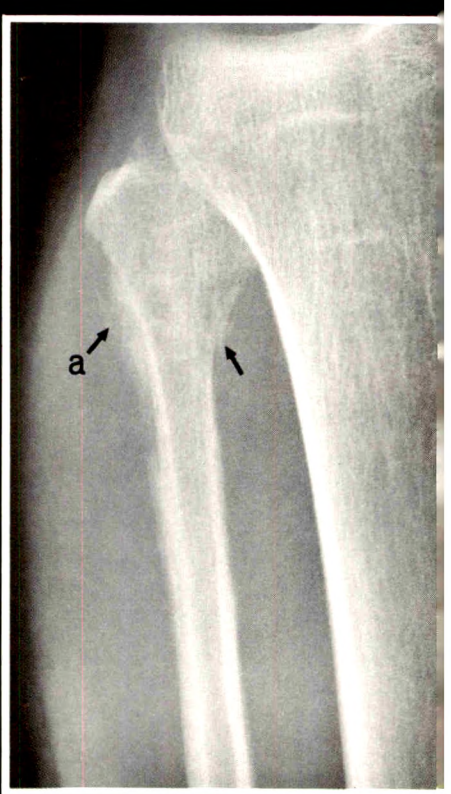
Primary Fibrosarcoma of Bone

Fibrosarcoma: The malevolent mimic! As varied as are its degrees of aggression, so too are its roentgen manifestations!

Classically, these tumors are centrally placed and usually fail to produce much new bone. More common in adults, they favor long bones—especially the lower limbs. They have a tendency to expand bone and thin the cortex, which it frequently violates to invade the perifocal soft tissues. Classic though these roentgen findings may be, fibrosarcomas have a penchant for assuming the trappings of entirely different tumors. Witness the following, all of which are verified:

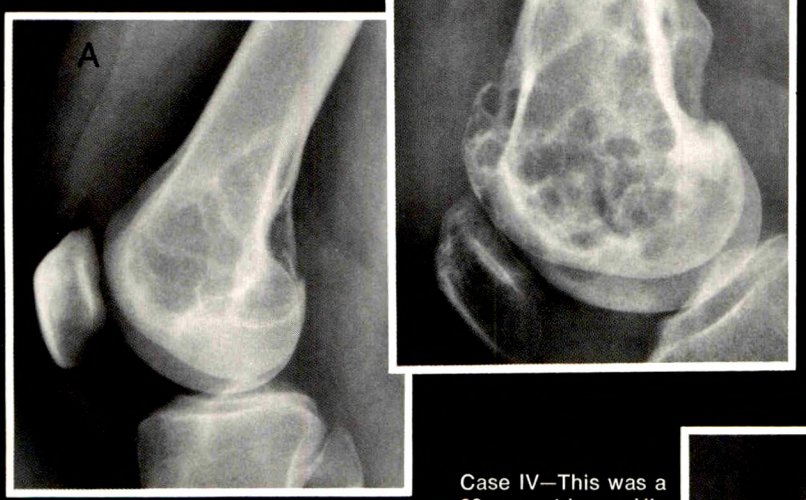


Case I—A 53-year-old man had this huge extraosseous mass in his right groin. Lytic at its site of origin in the pubis (x), which was fractured (arrow), its extensions into the pelvic (y) and groin (z) contained clouds of amorphous calcific debris not unlike that seen in chondrosarcoma. Certainly, there was reason to believe the lesion to be a chondrosarcoma arising in the pubis. Yet it proved to be a primary fibrosarcoma.

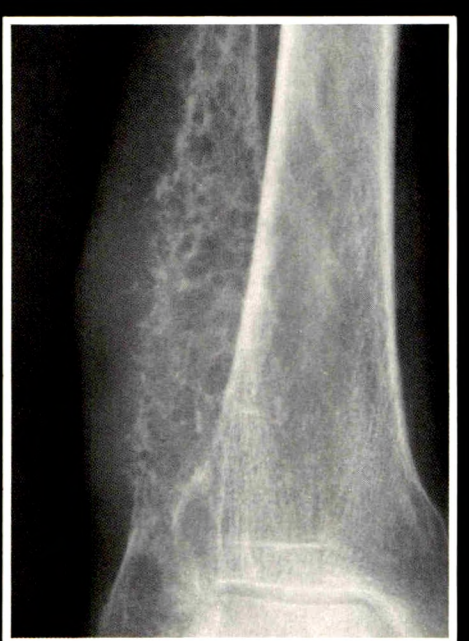


Case II—This young man of 18 years had pain and swelling for several months. His fibular lesion was medullary in origin and destroyed both spongiosa and cortex with equal avidity. The tumor had breached the bony cortex and had invaded perifocal soft tissues. In addition to the pathologic fracture (arrow), there also was considerable amorphous periosteal new bone deposited along its lateral margin (a). In a young adult, would you not have called this tumor an osteosarcoma? It, too, was a primary

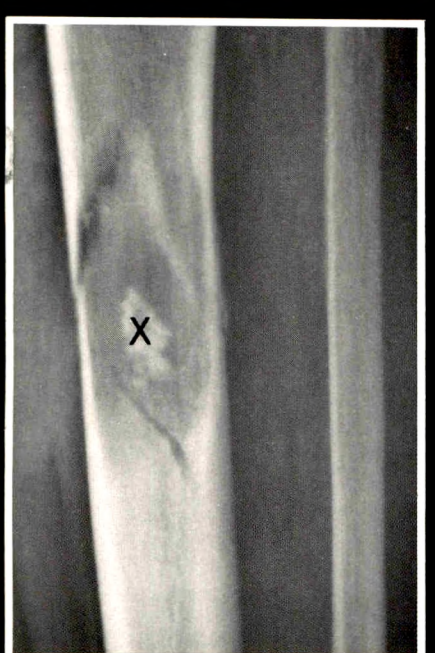
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Case III—This was a 30-year-old man who was first thought clinically to have a giant-cell tumor (Fig. A). The postoperative diagnosis at that time was benign nonosteogenic fibroma. But, 29 months later (Fig. B) the lesion had changed dramatically. Now the distal femur had been further destroyed and was more cystic. However, a degree of marginal sclerosis was still evident, and soft-tissue exposures revealed a large extraosseous tumor mass. Certainly, one would have called this a "malignant giant-cell tumor." It was not; it was another primary fibrosarcoma!



Case IV—This was a 69-year-old man. His distal fibula had been replaced by an expanding bone-destroying tumor, which had left in its wake this peculiar septate and honeycomb abnormal bone. No semblance of normal spongiosa or cortex remained. Its perifocal soft-tissue extensions (arrows) were abundantly clear. Rather than an angiomatous tumor, which logic and experience would have dictated, this also proved to be a fibrosarcoma.



Case V—A woman, 65 years old, complained of a pathologic fracture. The proximal half of the tibia had been invaded by a centrally located osteolytic lesion within the center of which lay flocculent calcific debris (x) not unlike that seen in chondrosarcoma. Noteworthy was the total absence of new bone formation. One might have considered this an enchondroma that had undergone chondrosarcomatous degeneration. Instead, it proved to be a primary fibrosarcoma.

Primary fibrosarcomas in bone are uncommon. To make the diagnosis radiologically, one must bear it in mind constantly, particularly when the tumor appears in the lower limb of an adult with atypical clinical or roentgen findings.

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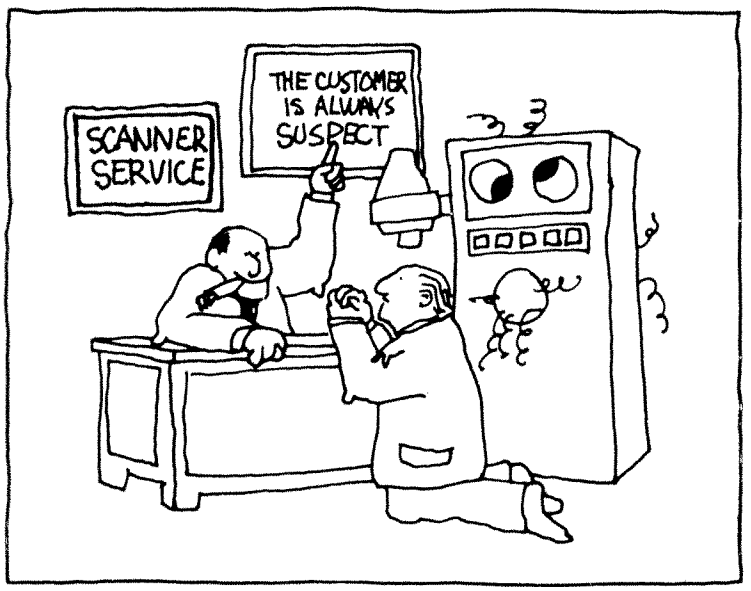
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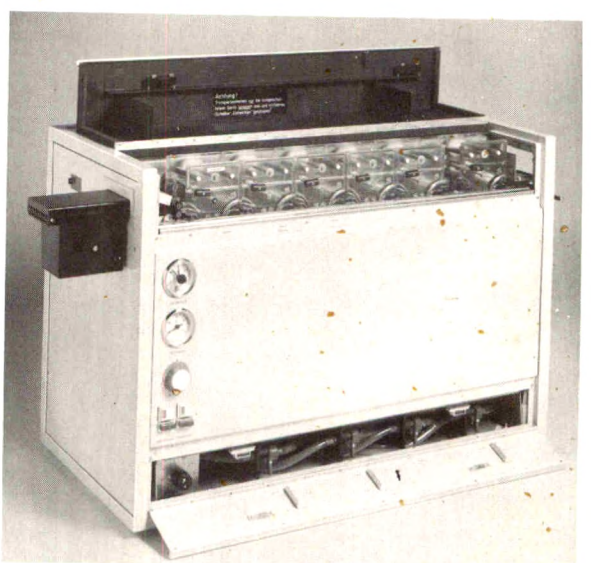
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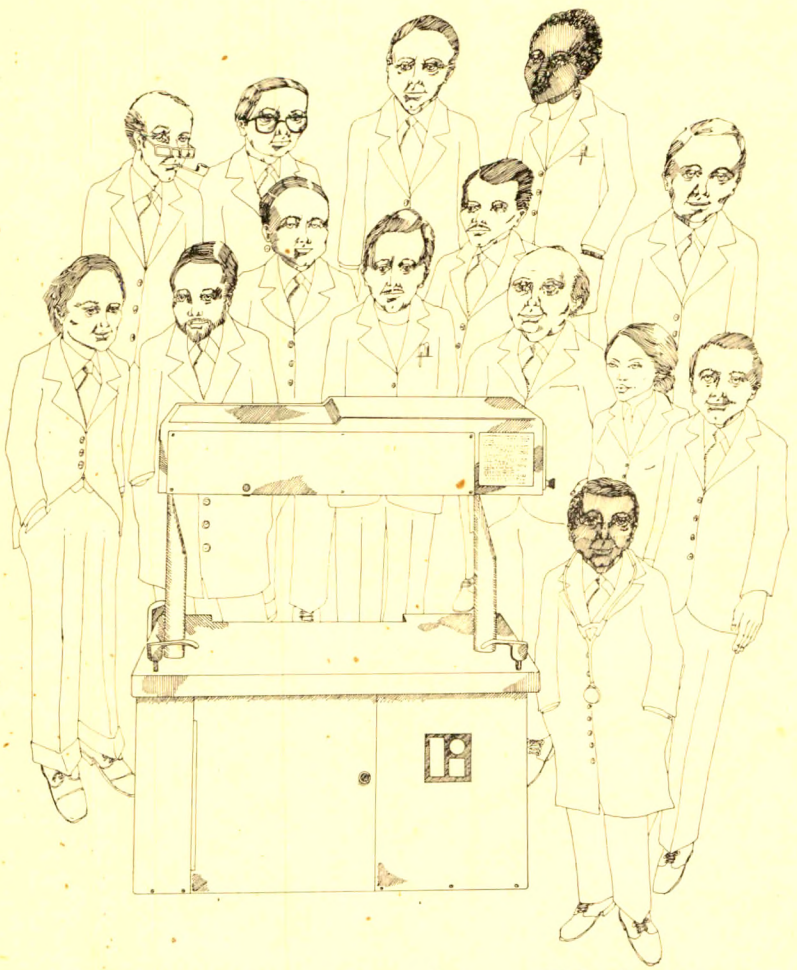


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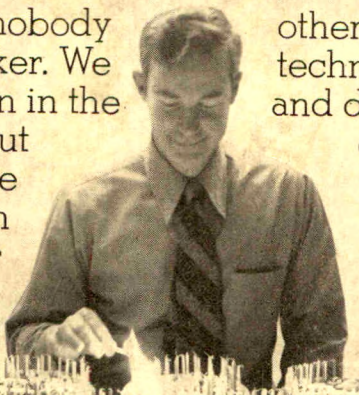
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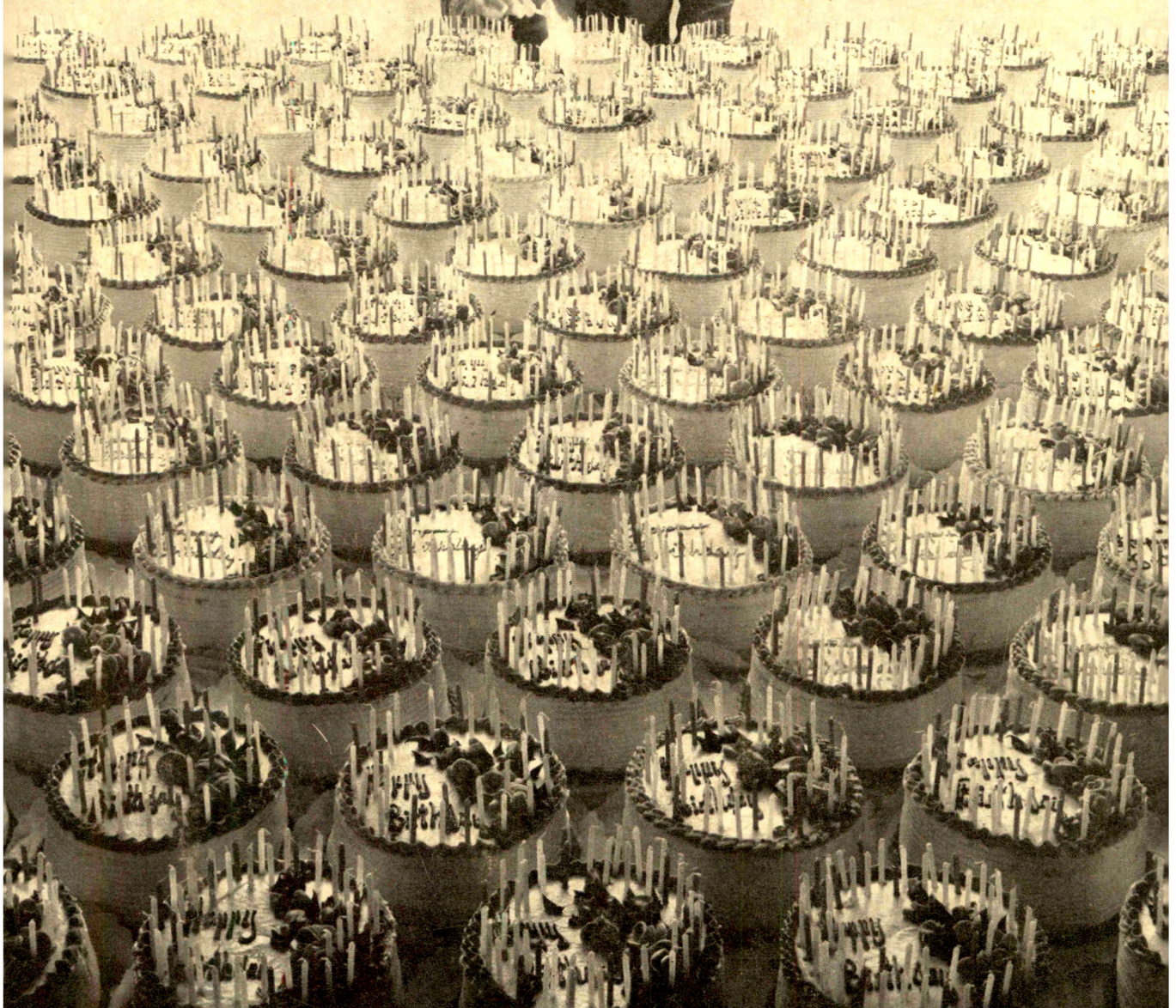
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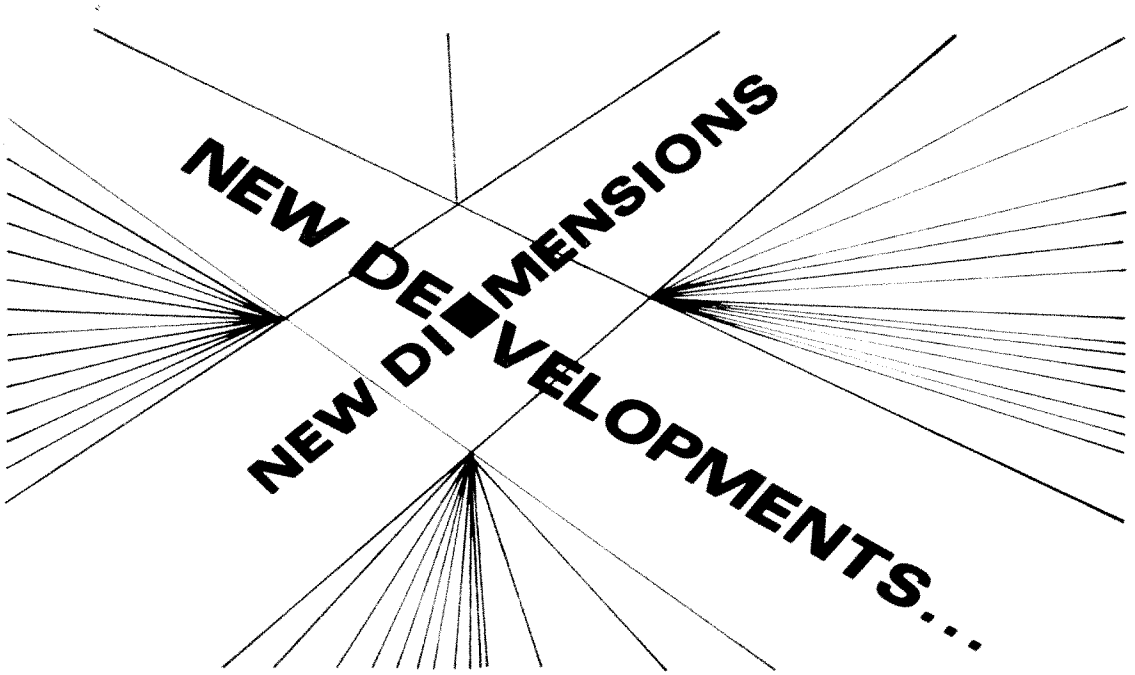
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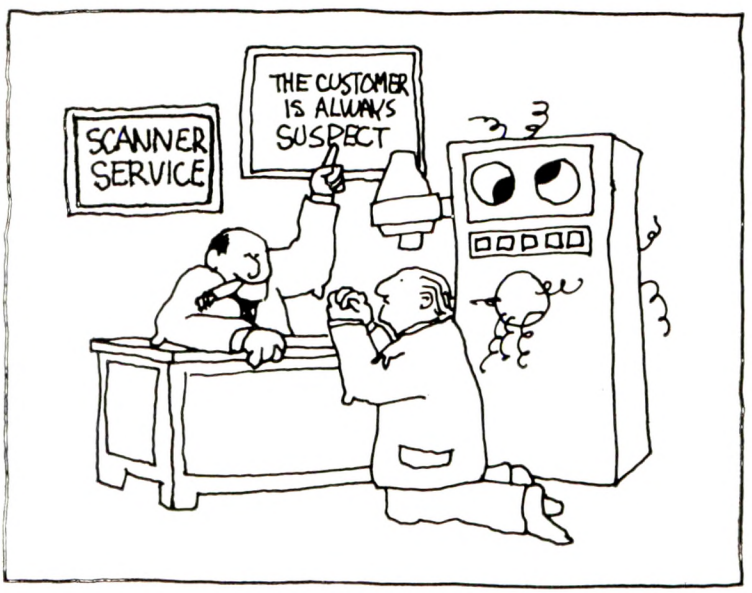
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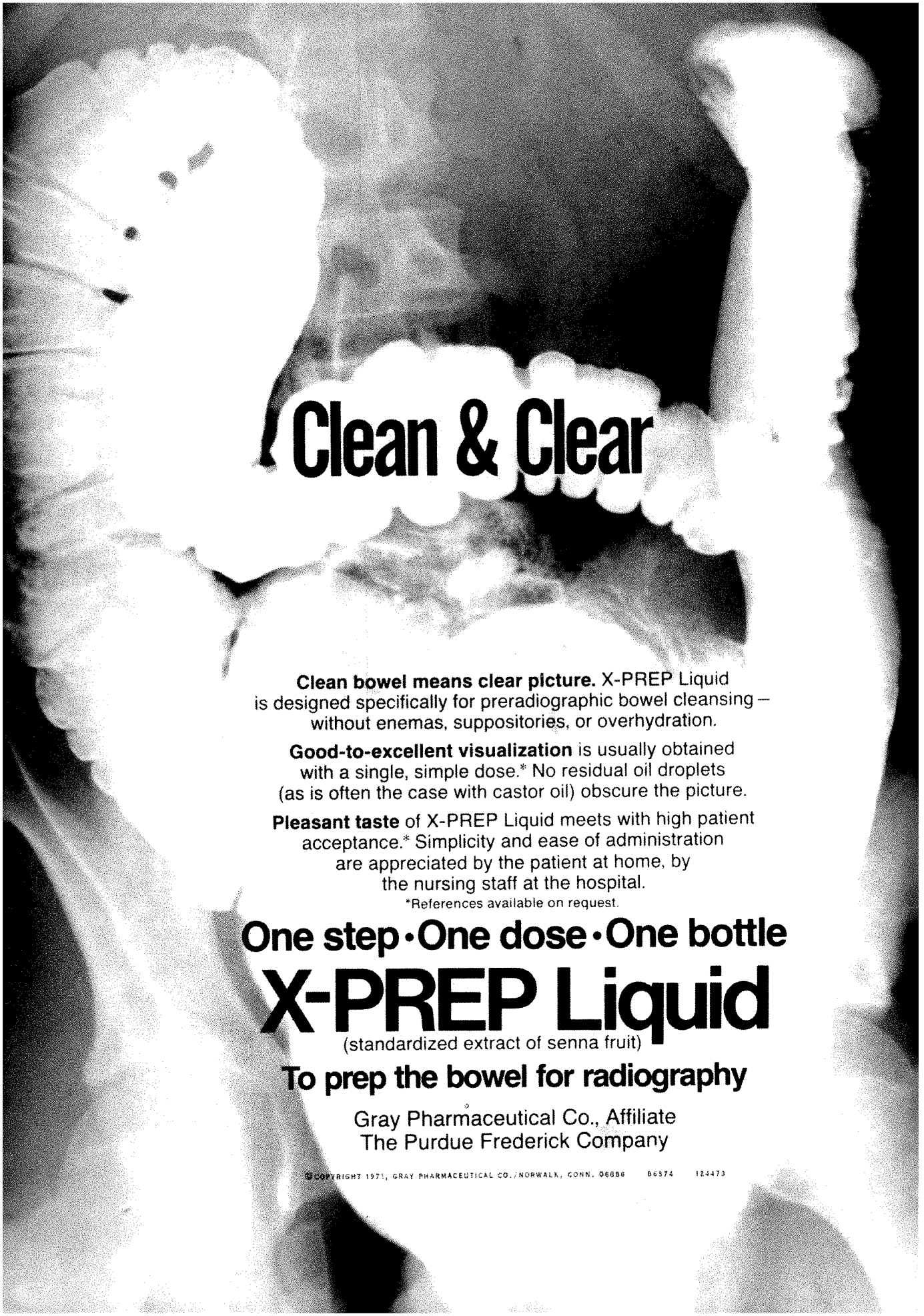
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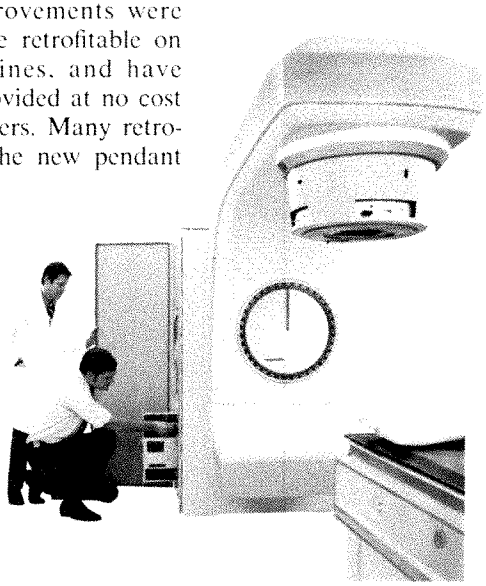
Thanks to Varian's Update Program, it still does.

The Update Program is Varian's on-going effort to maintain optimum product performance. In 1969, the Clinac 4 was both an advanced and a reliable machine. Today — 57 innovations later — it still is.

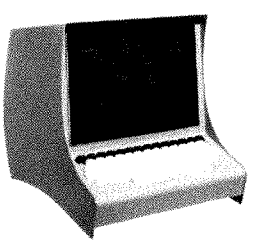
Varian's update program has provided many performance, reliability and safety features into previously installed units. Tomorrow's technical advances will similarly benefit today's users.

Improvement program is continuous. Varian engineers monitor performance data from well over a hundred operating units. When we find a way to improve reliability, safety or operational convenience of the Clinac, we follow up on it. The majority of these improvements were designed to be retrofitable on earlier machines, and have often been provided at no cost to our customers. Many retrofits, such as the new pendant assembly, enhance reliability and improve on human engineering. Others, such as the small field flattening filter and new range finder light assembly, improve the Clinac's operating characteristics and ease of setup.





Compatible new accessories, for addition to existing Clinacs are continually under development. Some recent examples include CART-D, our Computer Assisted Radiation Therapy record and verify system, and a precision treatment chair which simply attaches to the side rails of the existing couch.



Varian's update philosophy, one more reason why Varian is the world leader in medical linear accelerators. Varian Radiation Division, 611 Hansen Way, Palo Alto, CA (415) 493-4000; Springfield, New Jersey, (201) 376-6632; Detroit (Plymouth), Michigan, (313) 455-1570; Chicago (Park Ridge), Illinois, (312) 825-6232; Atlanta, Georgia, (404) 256-9363. In Europe: Steinhauserstrasse, 6300 Zug, Switzerland; Russell House, Molesey Road, Walton-on-Thames, Surrey, England. In South America: Alameda Lorena, 1834 ZC 5 Cerqueira Cesar, Sao Paulo — SP — Brazil.

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Clinical Beta Sources.
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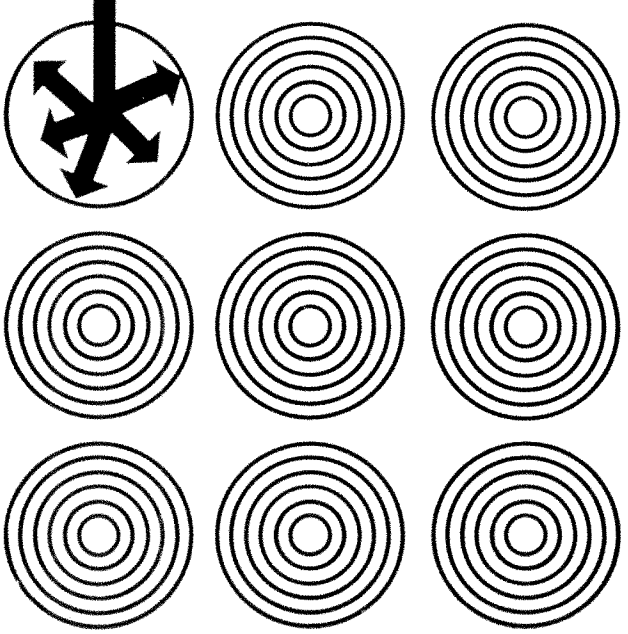
Please write or call our home office—details of specifications and prices will be sent on request.

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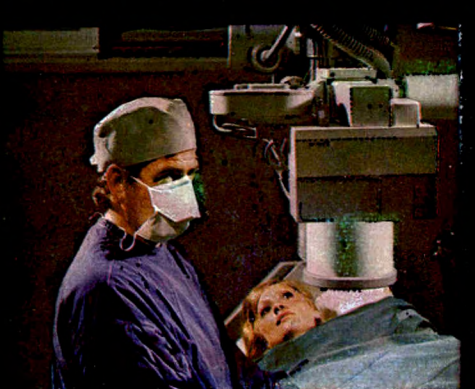




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Philips have been associated with X-ray apparatus since the earliest days of radiology and much of the equipment on which modern techniques depend was first introduced by us. The Philips tradition of innovation is continued at the 13th International Congress of Radiology to be held in Madrid this October. You are cordially invited to visit our stand at the technical exhibition, where experts from our organisations all over the world will be pleased to demonstrate the latest additions to our programme.

Many people will, of course, not be able to join us in Madrid. If you are one of these, but would nevertheless like to have information on our new equipment, please ask your local Philips organisation or Philips Medical Systems Division, Eindhoven, The Netherlands, for our souvenir brochure on the exhibition.

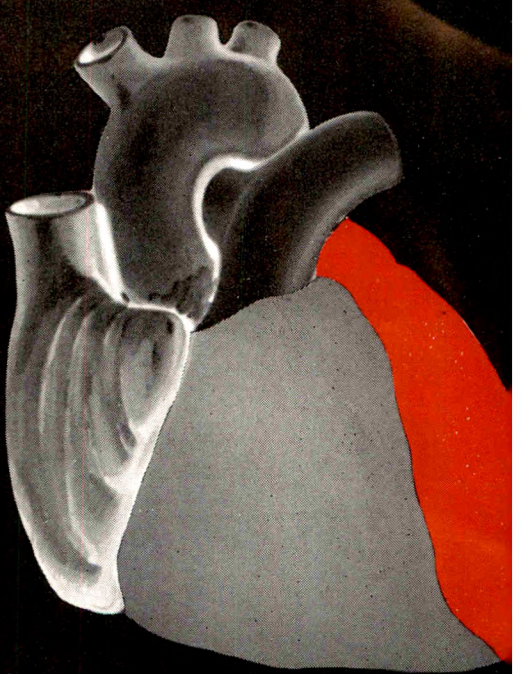


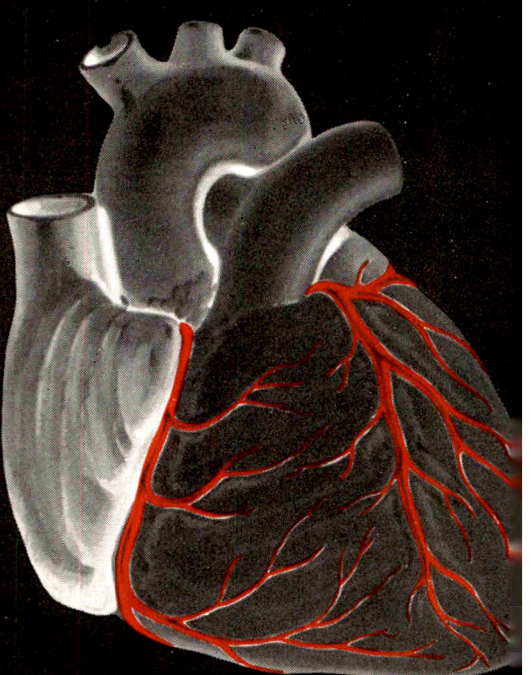
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in a single bottle...

enough contrast agent for selective coronary arteriography combined with left ventriculography







ntroducing
the new 200cc.
single-dose bottle

RENOGRAFIN®-
76 (MEGLUMINE DIATRIZOATE
66% AND SODIUM DIATRIZOATE
0% INJECTION)

quantity sufficient for combined procedure—selective coronary
teriography with left ventriculography
extra convenience—a single bottle to open
cap may be removed or spiked
lso available: 20 cc. and 50 cc. single-dose vials for other procedures
enografin-76 is indicated for excretion urography, aortography,
ediatric angiocardiography, peripheral arteriography, selective
oronary arteriography, and selective coronary arteriography
ombined with left ventriculography.

QUIBB HOSPITAL DIVISION

For brief summary, see next page.

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ıe new 200cc.
ngle-dose bottle

RENOGRAFIN®-76

Meglumine Diatrizoate 66% and
odium Diatrizoate 10% Injection)

enografin-76 is supplied as a sterile, aqueous lution providing 66% meglumine diatrizoate d 10% sodium diatrizoate with 0.32% sodium trate as a buffer and 0.04% disodium edetate a sequestering agent. The solution contains proximately 37% (370 mg./cc.) bound iodine d approximately 4.48 mg. (0.19 mEq.) sodium r cc.

ontraindications: In patients with a hypersenivity to salts of diatrizoic acid. Urography ntraindicated in patients with anuria.

arnings: A definite risk exists with the use of ntrast agents in excretion urography in paents with multiple myeloma. There has been uria with progressive uremia, renal failure d death. This risk of the procedure in these tients is not a contraindication; however, rtial dehydration in preparation for study is t recommended since it may predispose for ecipitation of myeloma protein in renal bules. No therapy, including dialysis, has en successful in reversing this effect. yeloma should be considered in persons over before undertaking urographic procedures.

In cases of known or suspected pheochrocytoma, if the physician feels that the posle benefits outweigh the considered risks, diopaque materials should be administered th extreme caution; however, an absolute nimum of material should be injected, the ood pressure should be assessed throughout e procedure, and measures for treating a pertensive crisis should be available.

Contrast media may promote sickling in mozygous individuals when injected I.V. or ra-arterially. Although a history of sensitivity iodine *per se* or to other contrast media is t an absolute contraindication, administran of diatrizoate requires extreme caution in ch cases. Renografin-76 should be used in egnant patients only when the physician ems its use essential to the welfare of the tient since safe use during pregnancy has t been established. Perform thyroid function sts prior to administration of Renografin-76 ıce iodine-containing contrast agents may er the test results.

Weigh the inherent risks against necessity performing *angiocardiography* in cyanotic ants and patients with chronic pulmonary ıphysema. In *pediatric angiocardiography*, a se of 10 to 20 cc. may be particularly hazdous in infants weighing less than 7 kg.; this k is probably significantly increased if these ants have preexisting right heart "strain," ht heart failure, and effectively decreased obliterated pulmonary vascular beds. Perm *urography* with extreme caution in perns with severe concomitant hepatic and ıal disease. Perform *selective coronary arıiography* only in selected patients and those whom expected benefits outweigh the produral risk.

ecautions: Diagnostic procedures involving e of contrast agents should be performed der the direction of personnel with prereqsite training and a thorough knowledge of the rticular procedure. Appropriate facilities ould be available for coping with situations ich may arise as a result of the procedure d for emergency treatment of severe reıtions to the contrast agent itself; competent rsonnel and emergency facilities should be ailable for at least 30 to 60 minutes after I.V. ministration since delayed reactions have en known to occur. These severe lifeeatening reactions suggest hypersensitivity the contrast agent. A personal or family hisy of asthma or allergy or a history of a preıus reaction to a contrast agent warrants special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 cc. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents; therefore, if known or suspected hepatic or biliary disorder exists, administration of Renografin-76 should be postponed following the ingestion of cholecystographic agents. Consider the functional ability of the kidneys before injecting Renografin-76. Use cautiously in severely debilitated patients and in those with marked hypertension. Bear in mind the possibility of thrombosis when using percutaneous techniques.

In *excretion urography*, adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be hazardous; this also applies to *pediatric angiocardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography*, hypotension or moderate decreases in blood pressure seem to occur frequently with intra-arterial (brachial) injections; this is transient and usually requires no treatment. Monitor blood pressure during the immediate 10 minutes after injection. It is recommended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and adequate facilities for immediate resuscitation and cardioversion.

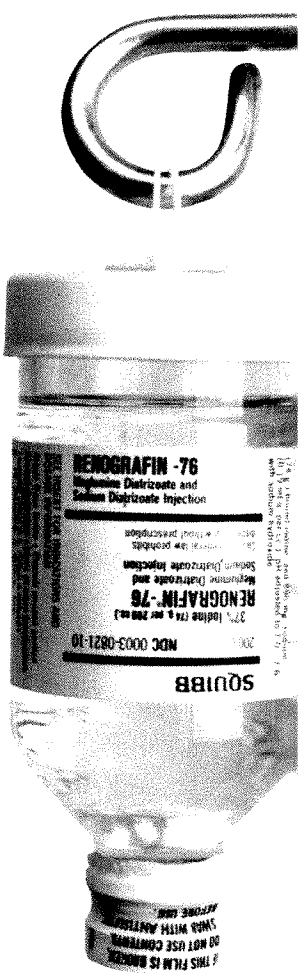
Adverse Reactions: Nausea, vomiting, flushing, or a generalized feeling of warmth are the reactions seen most frequently with intravascular injection. Symptoms which may occur are chills, fever, sweating, headache, dizziness, pallor, weakness, severe retching and choking, wheezing, a rise or fall in blood pressure, facial or conjunctival petechiae, urticaria, pruritus, rash, and other eruptions, edema, cramps, tremors, itching, sneezing, lacrimation, etc. Antihistaminic agents may be of benefit; rarely, such reactions may be severe enough to require discontinuation of dosage. There have been a few reports of a burning or stinging sensation or numbness, of venospasm or venous pain, and of partial collapse of the injected vein. Neutropenia or thrombophlebitis may occur. Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovascular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, agitation, confusion, and cyanosis progressing to unconsciousness. An allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoid shock may occur. Temporary renal shutdown or other nephropathy may occur.

Adverse reactions as a consequence of *excretion urography* include cardiac arrest, ventricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to generalized vasodilation. Risks of *aortography* procedures include injury to aorta and neighboring organs, pleural puncture, renal damage (including infarction and acute tubular necrosis with oliguria and anuria), accidental selective filling of right renal artery during translumbar procedure in presence of preexistent renal ease, retroperitoneal hemorrhage from tr lumbar approach, spinal cord injury pathology associated with syndrome of tr verse myelitis, generalized petechiae, death following hypotension, arrhythmia, anaphylactoid reactions. In *pediatric an cardiography*, arrhythmia and death have curred. During *peripheral arteriography*, her rhage from puncture site, thrombosis of vessel, and brachial plexus palsy (follov axillary artery injection) have occurred. Du *selective coronary arteriography* and *selec coronary arteriography with left ventricu raphy*, transient ECG changes (most patier transient arrhythmias (infrequent); ventrici fibrillation (from manipulation of catheter administration of medium); hypotension; ch pain; myocardial infarction; transient elevat of creatinine phosphokinase (occurred in ab 30% of patients tested); fatalities have b reported; hemorrhage, thrombosis, pseu aneurysms at puncture site, dislogment arteriosclerotic plaques, dissection of coron vessels, and transient sinus arrest have curred due to the procedure.

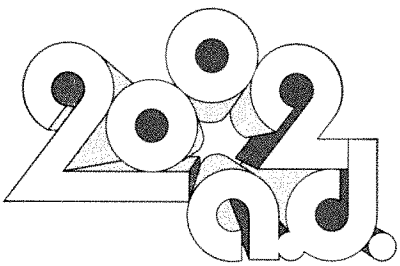
For full prescribing information, cons package insert.

How Supplied: Available in 20 cc. and 50 single-dose vials and in 200 cc. single-d bottles.

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Gone is the guesswork when you photoscan with Pho/Dot. Because Pho/Dot incorporates a number of significant advances in electromechanical design and engineering, to bring you the highest order of fidelity and convenience in clinical isotope scanning. To name a few advances . . .

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Maximum Tap Rate—Tapper is capable of operating at 70 pulses per second, continuously! (Occasional higher repetition rates will not jam the tapper.)

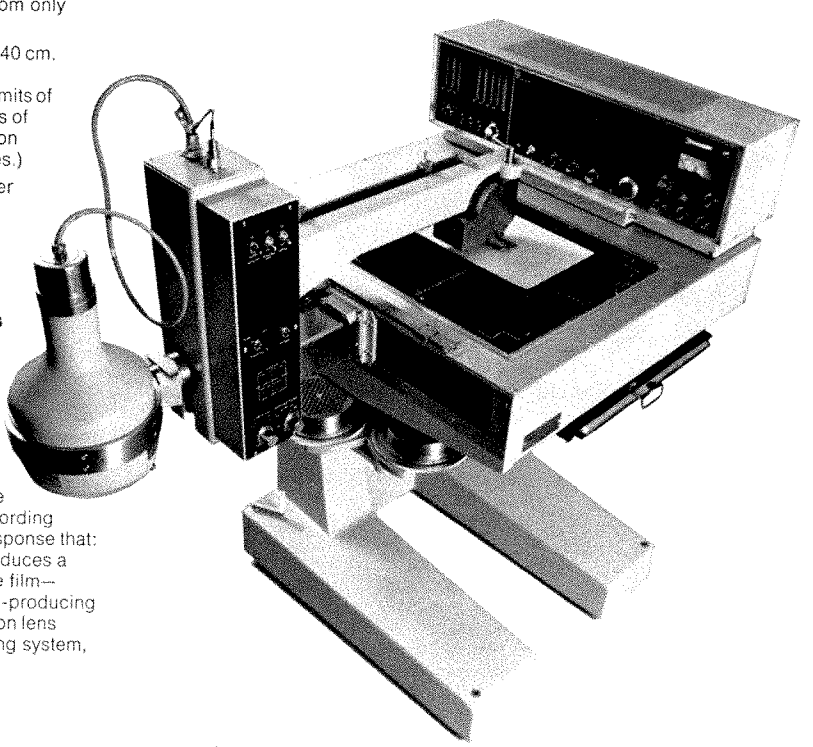
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Enough to whet your interest? If you'd like to learn more about all the features of this truly unusual instrument that's 'way ahead of its time ...more like 2002 A.D. than 1973 ...contact your Searle Radiographics (formerly Nuclear-Chicago) sales engineer or write to us for our free Pho/Dot brochure.

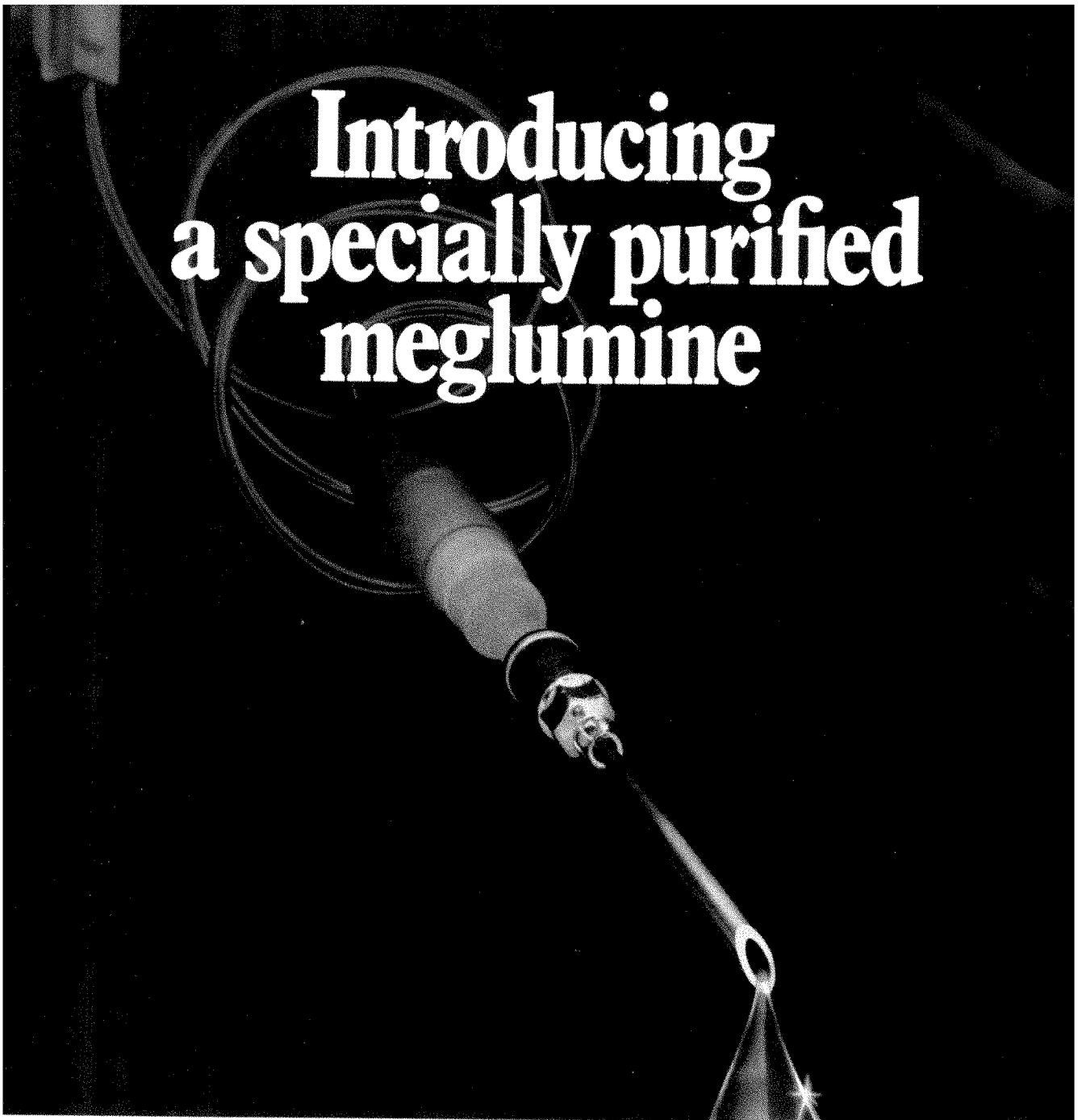
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CM-278



specific for Drip Infusion Urography

New Hypaque-DIU 30%

brand of **meglumine diatrizoate, USP**

- Extremely low sodium content (less than 0.02 mg./ml. in stabilizing agent)—of special advantage in patients with congestive heart failure.
- Highly diagnostic urographic contrast—reduces the need for retrograde pyelography.
- Good systemic tolerance.*

*Consult important product information on next page for patient selection, prescribing, and precautionary recommendations.



A logical addition to the expanding Hypaque® product line

New Hypaque-DIU® 30%

brand of

meglumine diatrizoate, USP

Sterile Aqueous Solution
For Infusion Urography

Description: Hypaque-DIU 30 per cent is a solution of meglumine 3, 5-diacetamido-2, 4, 6-triiodobenzoate ($C_{18}H_{26}I_3N_3O_9$). The 30 per cent solution (weight/volume) contains approximately 141 mg. of organically-bound iodine per ml. It has a viscosity of 1.94 cps at 25°C. and 1.42 cps at 37°C. The pH is adjusted with hydrochloric acid or meglumine solution. The sterile, aqueous solution is clear and colorless to pale yellow. Hypaque-DIU 30 per cent is relatively thermostable and may be autoclaved. It should be protected from strong light. The solution contains calcium disodium edetate 1:10,000 as a sequestering stabilizing agent.

Action: Hypaque-DIU 30 per cent is a diagnostic radiopaque medium which provides visualization of internal structures.

Indication: Hypaque-DIU 30 per cent is indicated for infusion urography.

Warnings: *Usage in Pregnancy.* No teratogenic effects attributable to a 60 per cent solution of Hypaque meglumine have been observed in reproduction studies in animals. However, before administration of Hypaque-DIU 30 per cent to women of child-bearing potential, the benefit to the patient should be carefully weighed against the possible risk to the fetus. In addition, most authorities consider elective contrast radiography of the abdomen contraindicated during pregnancy.

Serious or fatal reactions have been associated with the administration of radiopaque media. It is of utmost importance that a course of action be carefully planned in advance for the immediate treatment of serious reactions, and that adequate and appropriate facilities be readily available in case a severe reaction occurs.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravascularly.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake and direct thyroxine assays, are not affected.

Precautions: Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered. **Diphenhydramine hydrochloride (i.e., Benadryl®), however, should not be mixed with Hypaque-DIU 30 per cent since it may cause precipitation.** The intravenous injection of a test dose of 0.5 to 1 ml. of the contrast agent before infusion of the full dose has been employed in an attempt to predict severe or fatal adverse reactions. The preponderance of recent scientific literature, however, now demonstrates that this provocative test procedure is not reliably predictive of serious or fatal reactions. Severe reactions and fatalities have occurred with the test dose alone, with the full dose after a nonreactive test dose, and with or without a history of allergy. No conclusive relationship between severe or fatal reactions and antigen-antibody reactions or other manifestations of allergy has been established. A history of allergy may be useful in predicting reactions of a mild or intermediate nature.

Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations, or of sensitivity to iodine.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure, it is essential to omit preparatory dehydration, to administer fluids, and attempt to alkalinize the urine.

Immediate adverse reactions to infusion urography have not been reported to occur at a higher frequency or with greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Infusion urography imposes a sudden osmotic load. This should be considered in patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, there is a possibility of the development of congestive heart failure hours after the procedure.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating infusion urography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion urography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

Adverse Reactions: Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

The following have been reported after administration of diatrizoates and other iodinated contrast media. *Cardiovascular reactions:* vein cramp and thrombophlebitis following intravenous injection, rare cases of cardiac arrhythmias, hypertension, hypotension and shock, and cardiac arrest. *Allergic reactions:* asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria, and rarely, anaphylactic reactions. Signs and symptoms related to the *respiratory system:* pulmonary or laryngeal edema, bronchospasm; or to the *nervous system:* restlessness, convulsions. *Other reactions:* flushing, pain, warmth, nausea, vomiting, anxiety, headache, dizziness, hematoma, and ecchymosis. Occasionally, transient proteinuria, and rarely, oliguria or anuria. Rarely, immediate or delayed rigors can occur, sometimes accompanied by hyperpyrexia. Infrequently, "iodism" (salivary gland swelling) from organic iodinated compounds appears two days after exposure and subsides by the sixth day.

Dosage and Administration: The recommended dose for infusion urography is calculated on the basis of 2 ml. of Hypaque-DIU 30 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimal results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

The solution is infused over a period of three to ten minutes. Radiographs are usually taken at 10, 20, and 30 minutes from the beginning of the infusion. Earlier films may be obtained for the evaluation of hypertension. Nephrograms are usually obtained at the end of the infusion, and voiding cystourethrograms are usually obtained 30 minutes after the beginning of the infusion.

How Supplied: Calibrated bottles of 300 ml., rubber stoppered, packed with and without sterile disposable intravenous infusion sets.

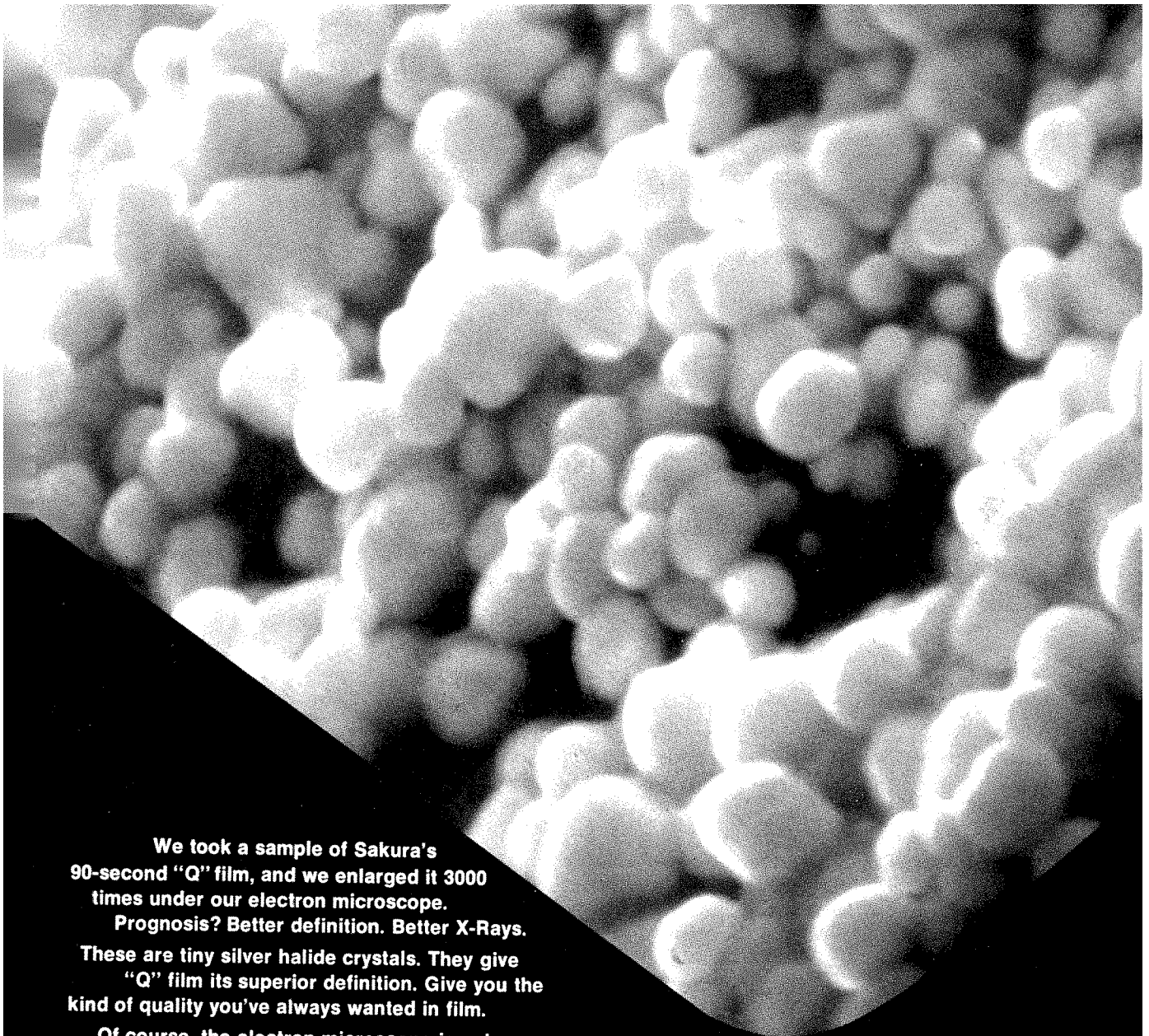
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(1621M)



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Clysodrast works and works safely.

Studies have shown that Clysodrast, administered as recommended, rapidly and completely cleanses the colon while producing no more adverse effects than enemas using water alone.[1,2]

Clysodrast is the only colonic evacuant that can perform all three of these essential actions:

1. effectively cleanses the colon
2. prepares the mucosa for deposition of Barium
3. aids in the complete and rapid elimination of contrast media when added to the Barium enema.

Since the release of Clysodrast over a million procedures utilizing Clysodrast have been performed. Clysodrast is packaged in a convenient unit-dose, pre-measured and prepacked for optimum convenience and safety.

Indications: CLYSODRAST (bisacodyl tannex) may be indicated for the preparation of patients for radiologic examinations of the colon, sigmoidoscopy and proctologic examinations.

Contraindications: CLYSODRAST is contraindicated in patients under the age of 10 because the possibility of absorption of tannic acid has not been adequately studied in this age group to warrant a conclusion of safety. CLYSODRAST is also contraindicated in cases with known or suspected extensive ulcerative lesions of the colon.

Warning: *Usage in Pregnancy.* Safe use of CLYSODRAST has not been established with respect to the adverse effects upon fetal development. Therefore, it should not be used in women of child-bearing potential, particularly during early pregnancy, except where, in the judgment of the physician, the potential benefits outweigh the possible hazards.

Precautions: Tannic acid, one of the active ingredients of CLYSODRAST, is hepatotoxic if absorbed in sufficient quantity. Deaths have been reported from hepatic damage due to tannic acid used in barium enema examination. Thus CLYSODRAST should be used with caution in a regimen where multiple enemas are administered. Certain patients, because of age, debility, or underl disease, require more gentle preparation than the routine castor oil and CLYSODR preparation. It is important that the instructions for preparation and administratio followed in detail, and that the recommended dosages not be exceeded.

Adverse Reactions: The following adverse reactions have been reported: Cramp weakness, nausea and fainting.

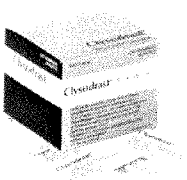
Dosage and Administration: It is important that the entire medical history and co tion of the patient be considered in deciding the dosage regimen. Traumatizing cedures, such as repetition of enemas (with or without CLYSODRAST) should be l at the minimum necessary to achieve the desired result.

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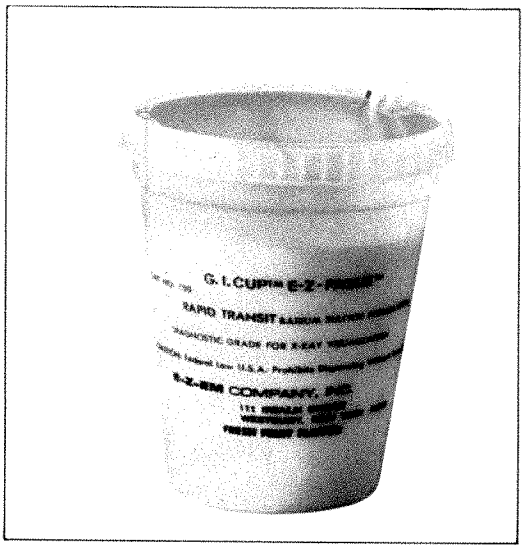
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1. Steinbach, H. L., Am. J. Roentgenology 87: 644, 1962.
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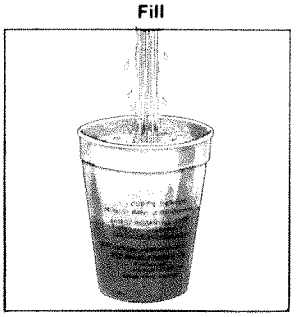
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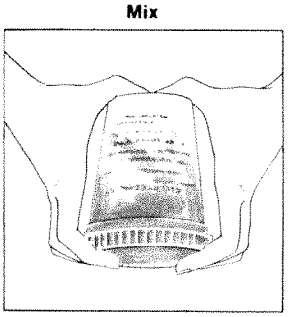
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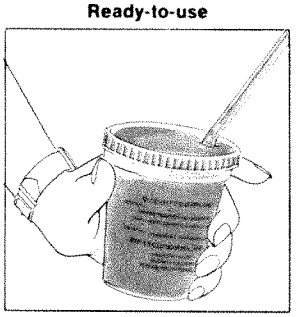
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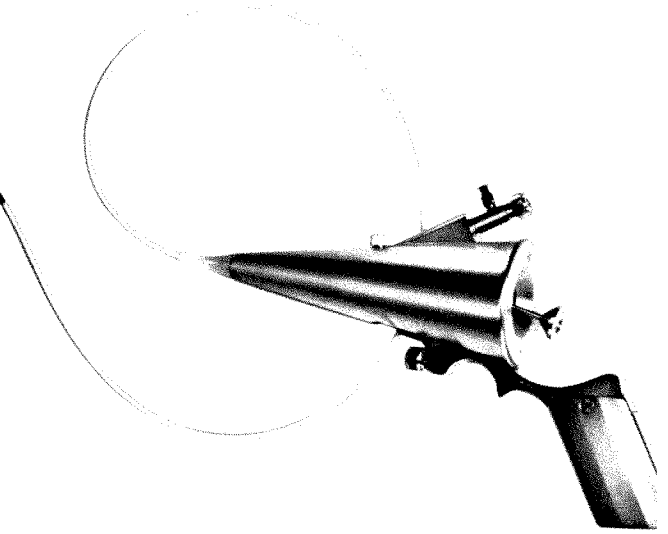
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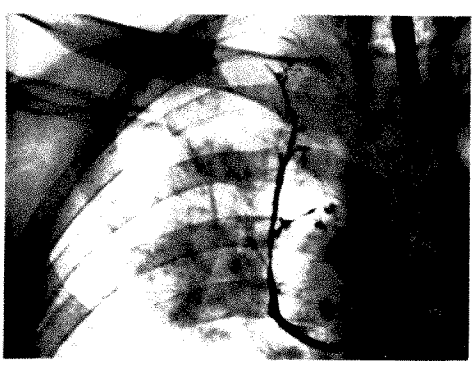
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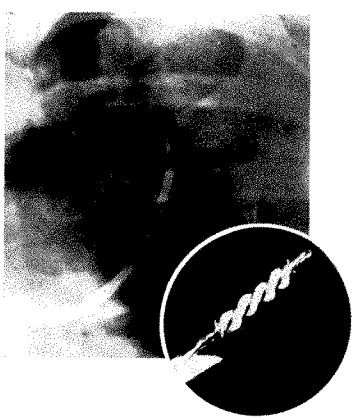
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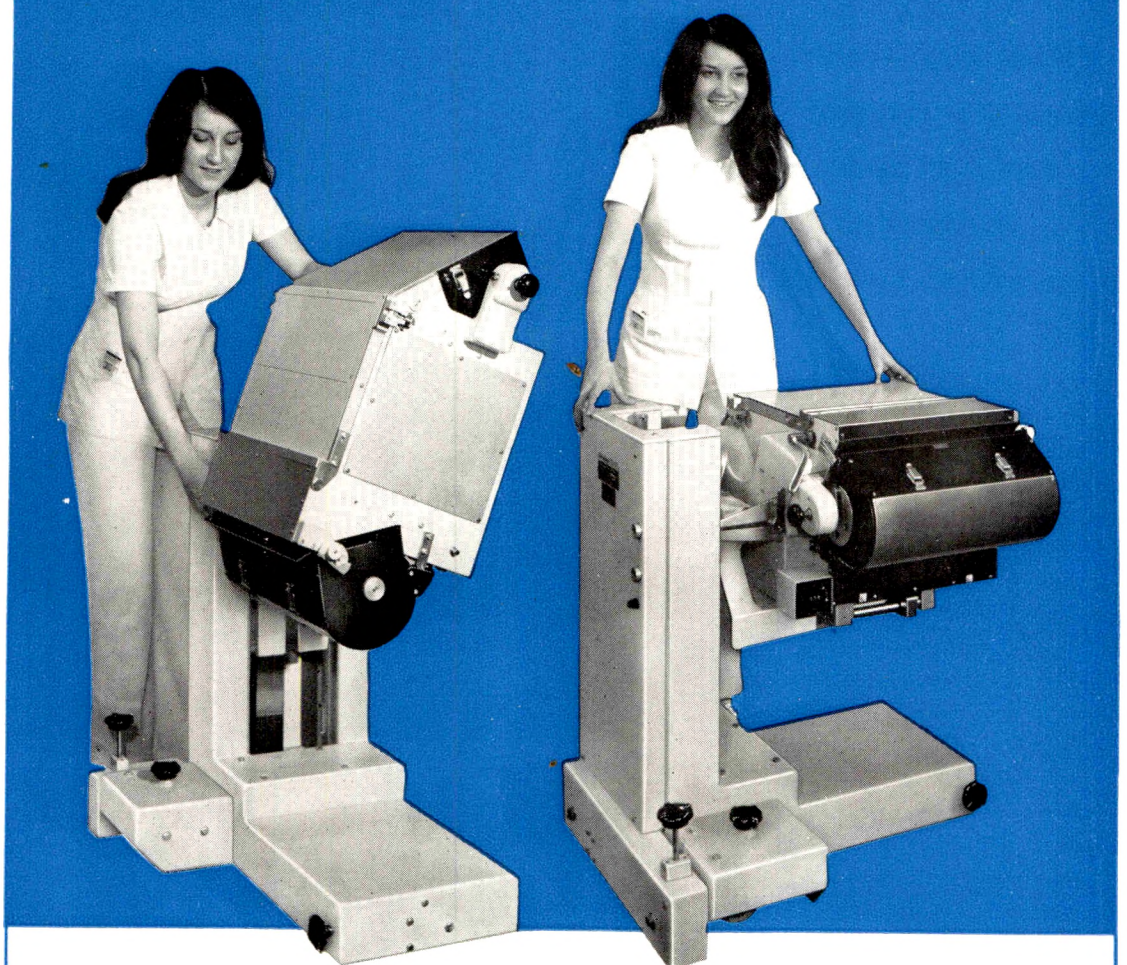
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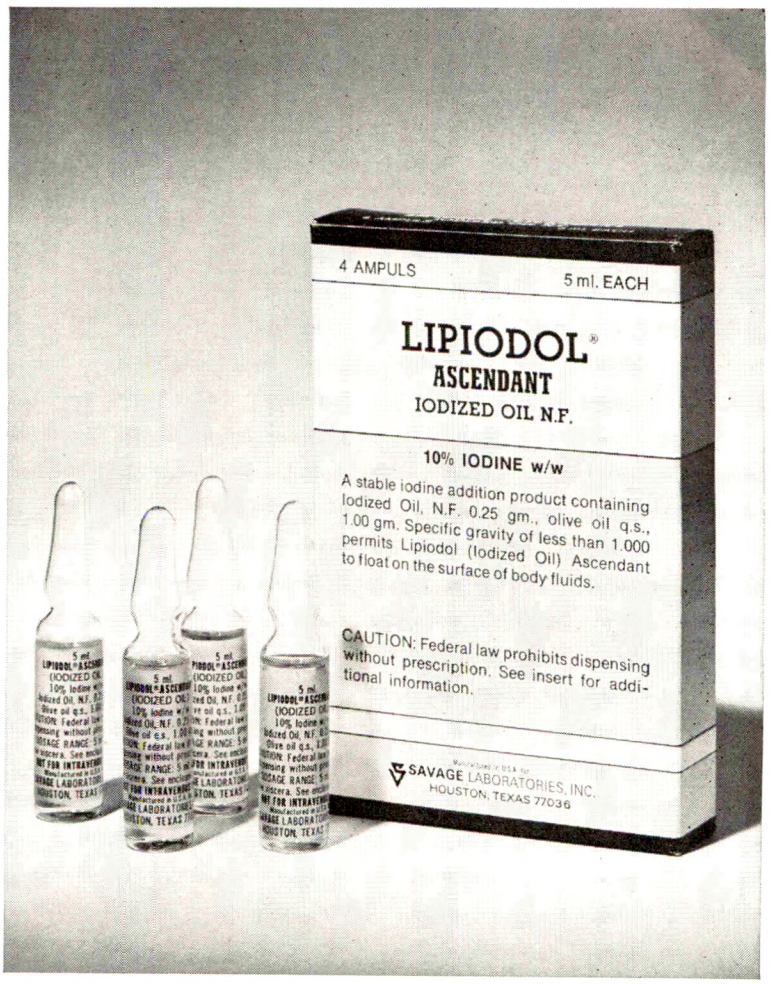
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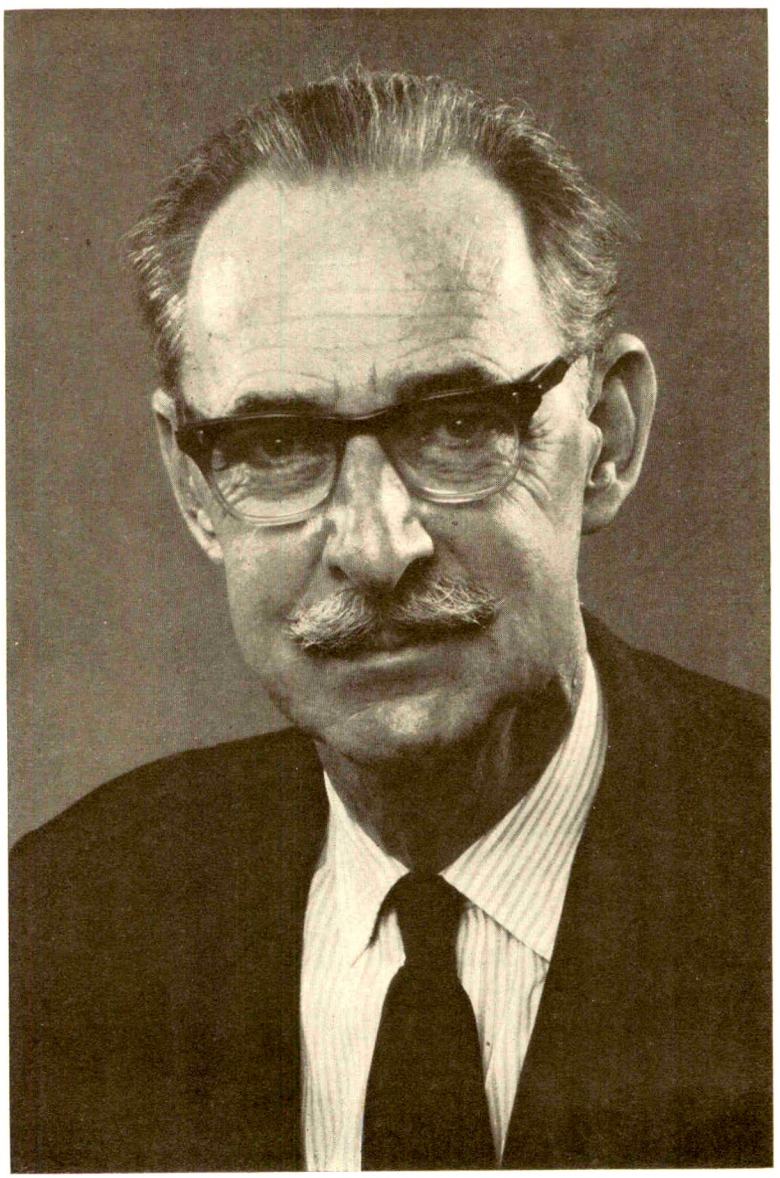
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THE FIRST WALTER BRADFORD CANNON LECTURE BEFORE THE SOCIETY OF GASTRO-INTESTINAL RADIOLOGISTS*

By HORACE W. DAVENPORT, D.Sc.
ANN ARBOR, MICHIGAN

I am pleased and proud to give this first Walter Bradford Cannon Lecture before your Society. I am pleased, for although I do not have an M.D. or any clinical experience, I have worked hard and long in many ways to contribute to Medicine. Your invitation shows that my work has been in some ways effective. I am proud to be associated through this lecture with Cannon who was America's pre-eminent physiologist. I went to Harvard after he had retired, and I met him briefly only once. However, I did have the task of organizing the teaching of gastrointestinal physiology by means of fluoroscopy in Cannon's old department. More recently, in helping to celebrate his centenary, I have become steeped in his work on the digestive tract. I have read articles of his that even his family didn't know about, and I have developed a just appreciation of his work and his greatness.[8] *I am happy that in choosing me to give this first Cannon Lecture you have reaffirmed the association between the basic science of physiology and the science and practice of radiology.*

IT was on Wednesday, December 9, 1896, that Walter Cannon and Albert Moser (Fig. 1) began the work which was to become the application of roentgen rays to the study of the mechanical factors of digestion. The work was described in a paper (Fig. 2) in Volume 1 of the *American Journal of Physiology* in 1898—a paper which still serves as a model of how to use new methods for the solution of physiologic problems.

Cannon was 25 years old and a first-year medical student at Harvard. Moser was 26 and a second-year student. We all know about Cannon, but I am probably the only one here who knows about Moser. I am, in fact, in possession of all that probably can be known about him, and I think some clinical society might now have an Albert Moser Lecture had he not been cut down by tuberculosis in 1903.[9]

The apparatus they used had been provided by Professor Bowditch, and it was housed in a darkroom on the second floor rear of the Harvard Medical School on

* From the Department of Physiology, The University of Michigan, Ann Arbor, Michigan.



Fig. 1. Walter B. Cannon, left, and Albert Moser, right, as medical students at Harvard Medical School.

Boylston Street. It was powered by a Wimshurst machine generating a current of 5 microamperes at about 40,000 volts. In the first experiments the animal was held in front of the tube (Fig. 3). In later experiments the tube was beneath a table top on which were placed the animal and the screen; Cannon crouched over the tube, receiving the burns from which he was to suffer for the rest of his life.

THE MOVEMENTS OF THE FOOD IN THE ŒSOPHAGUS.

By W. B. CANNON and A. MOSER.

[*From the Laboratory of Physiology in the Harvard Medical School.*]

At Dr. Bowditch's suggestion and with his valuable assistance — which we gratefully acknowledge — we made the following series of experiments.

To render the swallowed mass opaque subnitrate of bismuth was used. The salt is tasteless, practically inert, and can be fed in large quantities without harm. In order that observations could be made by more than one person, all experiments were conducted in a dark room. On the side of the animal opposite the Crookes tube was placed an open fluorescent screen on which the different tissues of the animal were outlined with varying degrees of light and shade. Among these shadows the swallowed mass appeared as a darker object, and thus its motion could be studied.

Fig. 2. The title and some important sentences from Cannon and Moser's paper in the *American Journal of Physiology*, 1898, *1*, 435–444.

Cannon kept a notebook in which the progress of the work can be followed. On December 9, 1896, Cannon and Moser fluoroscoped a dog lengthwise while the dog swallowed a pearl button. The movement was described as "regular" which means that the button was not squirted down the esophagus as Kronecker and Meltzer[13] had said liquids are. Realizing that they needed a better contrast medium, they used, on Wednesday, December 16, 1896, bismuth subnitrate which was to be the contrast medium for 15 years. Then on December 29, 1896, they gave before the Ninth Annual Meeting of the American Pysihological Society the first public demonstration of fluoroscopy.[14]

The penetrating power of the rays was so feeble that photography was impossible. Cannon pulled a roll of tissue paper over the screen, and using colored pencils, red for the stomach, green for the small intestine and brown for the colon, he traced the shadows. A few samples are treasured here and there: I have a set showing digestion of starch which was given to me by Professor E. M. Landis in commemoration of my stay at Harvard.

I propose to tell you some of the major results of the research I have been doing in the last 10 years which have to some extent changed our concepts of gastric ulceration.

It seems obvious that peptic ulceration is the result of the attack of acid and pepsin upon the mucosa, but the really remarkable fact is that in most instances acid and pepsin do not attack the mucosa. This property of the mucosa to contain acid within the lumen of the stomach I call, following Charles Code, the gastric mucosal barrier. In the normal stomach, acid diffuses so slowly into the mucosa through the barrier that it does the mucosa no harm.

The barrier must be a special property of the plasma membrane of the cells of the mucosa. It is common knowledge in general physiology that membranes behave as though they were formed by a layer of lipid and protein penetrated by relatively few water-filled channels. Small water-soluble substances can diffuse through these pores, and the ability of the gastric mucosa to contain acid can be accounted for by the small number of pores through its surface membrane.

On the other hand, fat-soluble substances can diffuse through the lipid layer itself, and, not being restricted to passage through pores, can penetrate the mucosa rapidly.

To study passage of acid through the mucosa I have used the preparation of a vagally denervated (Heidenhain) pouch of the oxyntic glandular area of the dog's stomach, irrigated by means of a simple arrangement of tubes and syringes. I know

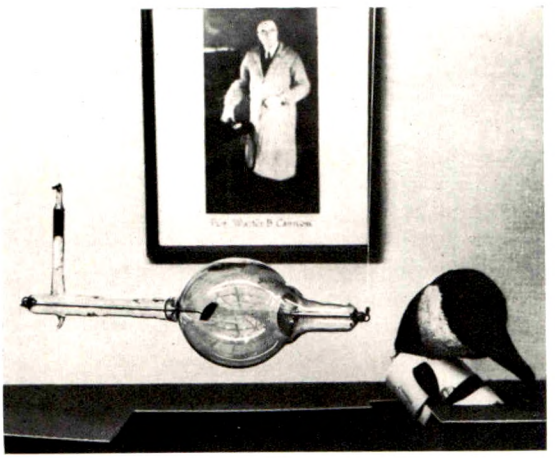


Fig. 3. One of the original tubes used in the first work by Cannon as it was on display in the Francis A. Countway Library of Medicine in Boston. The stuffed goose with its "fashionable cardboard collar" substitutes for the live goose fluoroscoped in the demonstration before the American Physiological Society on December 29, 1898. The live goose was probably domestic rather than Canadian. In the background, out of focus, is a photograph of the Cannon presentation portrait. (From a photograph taken by Mr. H. Goslyn.)

the volume and composition of the fluid I put into the pouch at the beginning of an experimental period and the volume and composition of the fluid I remove at the end. Consequently, I can calculate the rate at which substances have moved through the mucosa in either direction. By use of isotopes I can measure individual fluxes, and if I put a tracer in the blood I can measure its movement into the lumen. Use of T-1824 or chromated albumin allows me to measure the rate plasma is shed.[3]

I have studied the effect of aspirin upon the stomach, partly because of its great medical importance and partly because the effect of aspirin depends in an interesting way upon the acidity of gastric contents. Aspirin has a pK_a of 3.5. In neutral or alkaline solution aspirin—or its near relative, salicylic acid—is ionized and water soluble. It diffuses slowly into the mucosa. In acid solution it is un-ionized and fat-soluble, so that it diffuses rapidly into the mucosa.

Salicylic acid behaves the same way and

is easier to use. Table 1 shows the rates of gastric absorption of salicylic acid at 3 different acidities. In neutral solution it enters the mucosa slowly. In 1 millimolar acid, pH 3, and in 100 millimolar acid it enters the mucosa rapidly at approximately equal rates.

For some reason I do not understand, aspirin and salicylic acid, once they have entered the mucosa, destroy the gastric mucosal barrier, making it much more permeable to water-soluble substances such as hydrogen ions. Once the barrier has been broken, acid can enter the mucosa about five times faster than normal.

Figure 4 shows the results of 5 successive 30 minute periods of irrigation of a gastric pouch. Each cylinder contains the fluid removed at the end of 30 minutes. The first is a control solution of 100 mN HCl. The fluid is clear, and the volume is essentially equal to that placed in the pouch 30 minutes earlier. In the next 2 periods the pouch was irrigated with 100 mN HCl to which salicylic acid in a concentration equivalent to two aspirin tablets in 180 ml. of gastric juice was added to the acid. Observe that the volume recovered increased. In the last 2 periods the pouch was again irrigated with 100 mN HCl without salicylate. There was continued volume output and gross bleeding.

Figure 5 shows the results of a similar experiment in which, after an initial control period of irrigation with 100 mN HCl, the pouch was irrigated for 2 periods with salicylic acid in 1 mN HCl, pH 3. It has already been shown that salicylic acid is



Fig. 4. Fluids recovered from a pouch of the oxyntic glandular area of a dog's stomach irrigated for 30 min. with: (1) 100 mN HCl; (2 and 3) 20 mM salicylic acid in 100 mN HCl; and (4 and 5) 100 mN HCl.

absorbed rapidly from this solution. Chemical evidence which I will not detail showed that in these 2 periods salicylic acid broke the mucosal barrier. Nevertheless, there was no increase in volume and no bleeding. The reason is that at pH 3 the concentration of acid in the lumen is so low that there is no significant back diffusion of acid into the mucosa. This illustrates a major point: the gastric mucosa may be severely damaged, but it usually does not bleed unless acid is present to diffuse back into it.

As one might expect, neutralizing the contents of the stomach stops bleeding. I have frequently produced hemorrhagic gastritis by various means, and I have stopped the bleeding by irrigating the mucosa with neutral solutions. In fact, bleeding can be stopped by irrigating the mucosa with a solution of acetylsalicylate, pH 6.[5] Of course, bleeding is not stopped

Table I

Degree of ionization and rate of absorption of salicylic acid in pouches of the oxyntic glandular area of the dog's stomach

	Percentage Un-ionized	Rate of Absorption (μEq./30 min.)
100 mN HCl	99.9	329
1 mN HCl	75	365
Buffered, pH > 5	< 0.1	69

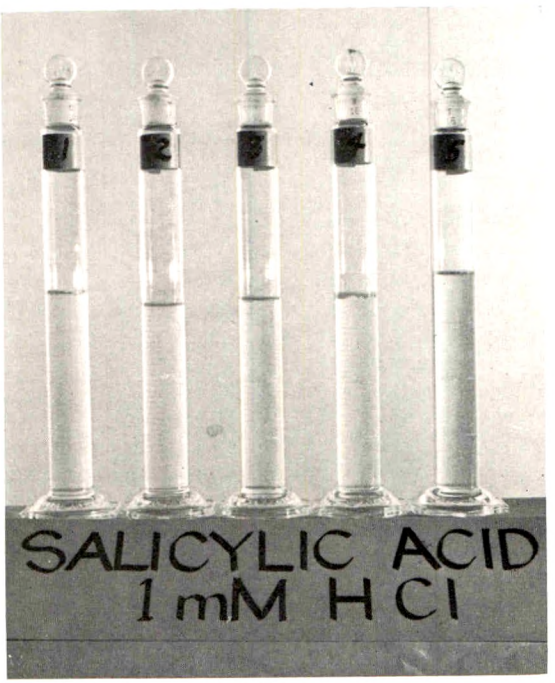


Fig. 5. Fluids recovered from a pouch of the oxyntic glandular area of a dog's stomach irrigated for 30 min. with: (1) 100 mN HCl; (2 and 3) 20 mM salicylic acid in 1 mN HCl; and (4 and 5) 100 mN HCl.

by the acetylsalicylate, but by the neutral solution in which it is dissolved. This point is an important one in the management of stress ulceration.

Cholinergic stimulation promotes bleeding from a mucosa which is both damaged and exposed to acid.[2,5] An engorged mucosa bleeds easily.[16] Cholinergic stimulation enhances contraction of gastric muscle, and veins draining the mucosa must pass through the muscle. For this reason I think that cholinergic stimulation probably promotes bleeding more by its effect on gastric blood vessels than by its stimulation of acid secretion.

Detergents destroy lipid membranes, and common commercial detergents such as one uses to wash dishes break the gastric mucosal barrier. By far the most important detergents are the natural ones: bile acids and lysolecithin of intestinal contents.[4] When these regurgitate into the stomach they impair the stomach's ability to protect itself against attack by acid, and this fact appears to account for some forms of gastric ulceration. Patients with gastric ulcers have bile in their stomachs more frequently and at a higher concentration than do normal subjects,[15] and regurgitation of bile in patients confined to intensive care units may be a major factor in causing "stress ulceration." Lysolecithin is present in intestinal contents during digestion of fat, and it occurs in barrier-breaking concentrations in patients with gastric ulcers.[10] Both bile acids and lysolecithin in contact with the gastric mucosa open the way to damage by acid, and hemorrhagic gastritis follows.

Acid diffusing into the mucosa releases histamine from stores within the mucosa,[12] and it greatly increases histamine-forming capacity.[11] This intramucosal release of histamine stimulates acid secretion, and the result is that a mucosa damaged by acid secretes more acid. Histamine causes vasodilatation, and blood flow through the damaged mucosa increases.[1] Histamine also increases capillary permeability, and this together with elevation of capillary pressure promotes filtration of large amounts of protein-containing fluid into the interstitial spaces; hence, mucosal edema. Edema fluid flows between cells of the surface epithelium into the gastric lumen, and this process accounts for much of the shedding of fluid by the mucosa. Since edema fluid dilutes and neutralizes acid, we have an explanation for the fact that patients with gastric ulcers appear to secrete a large volume of dilute acid.

Under some experimental circumstances the amount of plasma protein shed by the mucosa can be spectacularly large. I think my record is something like 48 ml. of fluid containing 31 ml. of plasma shed by 60 $cm.^2$ mucosa in 30 minutes. So far I have found 3 ways to produce the effect: topical application of histamine to a damaged mucosa;[3] irrigation with the sulfhydryl reagent dithiothreitol;[7] and by the production of a local allergic reaction.[6] I have, in effect, experimentally produced a state very like

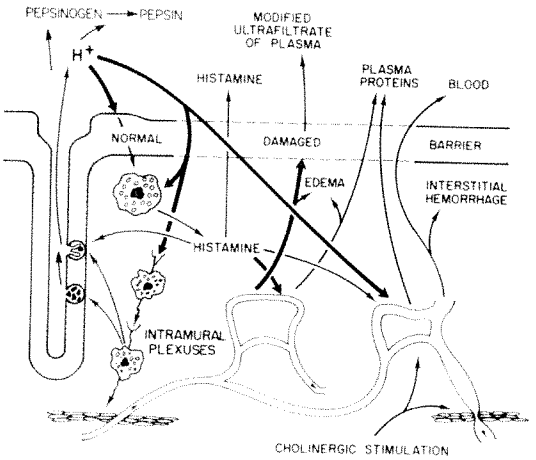


FIG. 6. A summary of the responses of the gastric mucosa to injury by back diffusion of acid.

Menetrier's disease, although production of mucosal hypertrophy still eludes me.

The processes I have been discussing are summarized in Figure 6.

One of Cannon's most notable characteristics was that, given an observation, he followed it logically and experimentally as far as he was able, and the result was that he laid the foundations of our knowledge of the behavior of the digestive tract. I hope that by following a similar path I have been able to add a little to our understanding of the pathophysiology of the digestive tract, and I am proud that you think enough of my work to associate me with Cannon in this lecture.

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VOL. 119, NO. 2

MECHOLYL ESOPHAGOGRAPHY*

By JOSEPH T. ENNIS, M.B., D.M.R.D., F.F.R., *and* ANN M. LEWICKI, M.D.
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MANOMETRIC studies of esophageal motility have considerable value in the differential diagnosis of diffuse motor disorders of the esophagus,[6] but also are time-consuming and exacting, dependent to a large extent upon highly trained personnel. Because such expertise does not always fall within the sphere of the attending radiologist, these investigations usually must be supervised by a specially trained gastroenterologist.

The subcutaneous administration of Mecholyl (acetyl-β-methylcholine chloride) combined with cinefluorography of the barium-filled esophagus provides an accurate assessment of motility throughout the entire esophagus and obviates the necessity for premedication or esophageal intubation. To the radiologist, ever searching for greater accuracy in diagnosis, this simple pharmacologic procedure offers a reasonable approach to the differentiation of benign and malignant esophageal strictures.

We have diagnosed achalasia in 5 patients examined with this technique.

TECHNIQUE

The patient is placed in the supine oblique position. The esophagus is filled with barium and kept so during the entire examination. Mecholyl is supplied in 25 mg. components. This amount can be diluted with sufficient sterile water (5 ml.) so that each milliliter contains 5 mg. of Mecholyl. As a precaution to arrest any unwanted muscarinic effects, it is advisable to have at hand a syringe containing 1 mg. of atropine.

Initially 2.5 mg. of Mecholyl is injected subcutaneously (never intramuscularly or intravenously) and the effect upon the barium-filled esophagus is observed fluoroscopically. A positive response may occur within 2 to 3 minutes and is seen as a lumen-obliterating contraction of the esophagus with reflux of barium into the patient's mouth (Fig. 2, *A–C*). A negative response is failure of the esophagus to contract and should be followed by a further subcutaneous injecton of Mecholyl after an interval of 4 to 5 minutes. The dosage may be adjusted upward in 2.5 mg. increments to as much as 10 mg./single dose in order to achieve a response. Because of the rapid degeneration of Mecholyl, there is no cumulative effect from doses made 5 to 10 minutes apart. No single dose should ever exceed 10 mg., however.

DISCUSSION

The response of the denervated esophagus to the parenteral administration of Mecholyl is determined on the basis of Cannon's law,[2] which emphasizes the supersensitive response in a denervated structure. Cannon's law states: "When in a series of efferent neurons a unit is destroyed, an increased irritability to chemical agents develops in the isolated structure or structures, the effect being maximal in the part directly denervated."

The dramatic esophageal response of patients with achalasia to the subcutaneous administration of this parasympathicomimetic agent offers further evidence that this neuromuscular disorder affects not only the cardia but the distal two-thirds of the esophagus. The part of the swallowing mechanism under voluntary control (supplied by the fifth, seventh, ninth, and eleventh cranial nerves) can be seen to be normal in these patients. Thus the ineffective peristalsis occurs only in the distal two-thirds of the esophagus where the smooth muscle is supplied by the vagal

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myenteric plexuses. Adams *et al.*[1] have demonstrated histochemically that there is complete disappearance of all of the nerve cells and fibers from the dilated esophageal segment and that the absence of enzyme activity in this region is reflected in the considerable reduction of true cholinesterase in the muscle as compared with normal esophageal muscles at similar levels.

Mecholyl, a synthetic choline derivative, is related chemically to acetylcholine but is less readily hydrolyzed by cholinesterase and so has a longer duration of action. The unpleasant nicotinic effects of Mecholyl are also less significant than those of acetylcholine. The effects of Mecholyl are the exact opposite of those produced by epinephrine. In therapeutic doses, therefore, Mecholyl (which may slow the heart rate and cause a transient fall in blood pressure) increases gastrointestinal tone and peristalsis.

The muscarinic side effects which complicate the use of Mecholyl, especially substernal pain,[6] have occurred in only one of our patients and were not serious enough to terminate the examination. Of more importance is the case reported by Troutman and Taylor[9] in which hematemesis and melena developed 12 hours after a positive Mecholyl test. Such complications are fortunately rare and do not justify exclusion of this valuable diagnostic test in the appraisal of achalasia. Flushing may occur prior to the dose level at which an esophageal response occurs but has no clinical significance or sequelae.

Although the dosage of Mecholyl is considered by the manufacturer to be weight and age related, no correlation between the dosage, the response, and the weight of the patient was apparent in our patients. This perhaps could be explained by the small numbers involved.

The degree of esophageal dilatation did appear to have an effect upon the response: the 3 patients whose esophagi measured 4 cm. or less responded to 5 mg. of Mecholyl (Fig. 1, *A–C*) while both patients whose esophageal diameters were 5 cm. or more necessitated an increased dosage (Fig. 2, *A–C*).

Cinefluorography is indispensable in the investigation of patients considered to have achalasia of the esophagus. Manometric studies alone without the aid of cinefluorography depend upon a small sensitive transducer to record changes in the intraesophageal pressure and may produce false-negative results in a grossly dilated, denervated esophagus. False-negative Mecholyl esophagograms also have been reported, however.[5,10] Hightower *et al.*[5] have advanced two theories for these failures in patients with known achalasia: the inability of the grossly dilated decompensated esophagus to respond to Mecholyl, and the failure of the small pressure transducer to record contractions occurring in isolated segments of the dilated esophagus. This point is illustrated in one of our patients in whom a negative manometric study was followed by positive Mecholyl esophagography which recorded slow but definite esophageal contractions cinefluorographically over a long segment of the distal esophagus. We have had no experience with a grossly dilated esophagus yielding a false-negative result.

Recent reports have also indicated that caution must be exercised in the interpretation of a positive Mecholyl esophagogram. Initially considered highly specific for the diagnosis of achalasia by Kramer and Inglefinger,[7] positive Mecholyl tests have now also been demonstrated in patients with esophageal spasm[8] or Chagas disease[3] and in a few patients with infiltrating carcinoma of the esophagus.[4] The positive Mecholyl response in the latter patients is caused by complete destruction of the myenteric plexuses to the level of the aortic arch by an infiltrating gastric fundal adenocarcinoma.

Despite this, however, a positive esophageal response to Mecholyl stimulation remains the convincing diagnostic test for achalasia and serves in particular to distinguish this entity from other causes of esophageal dilatation such as scleroderma,

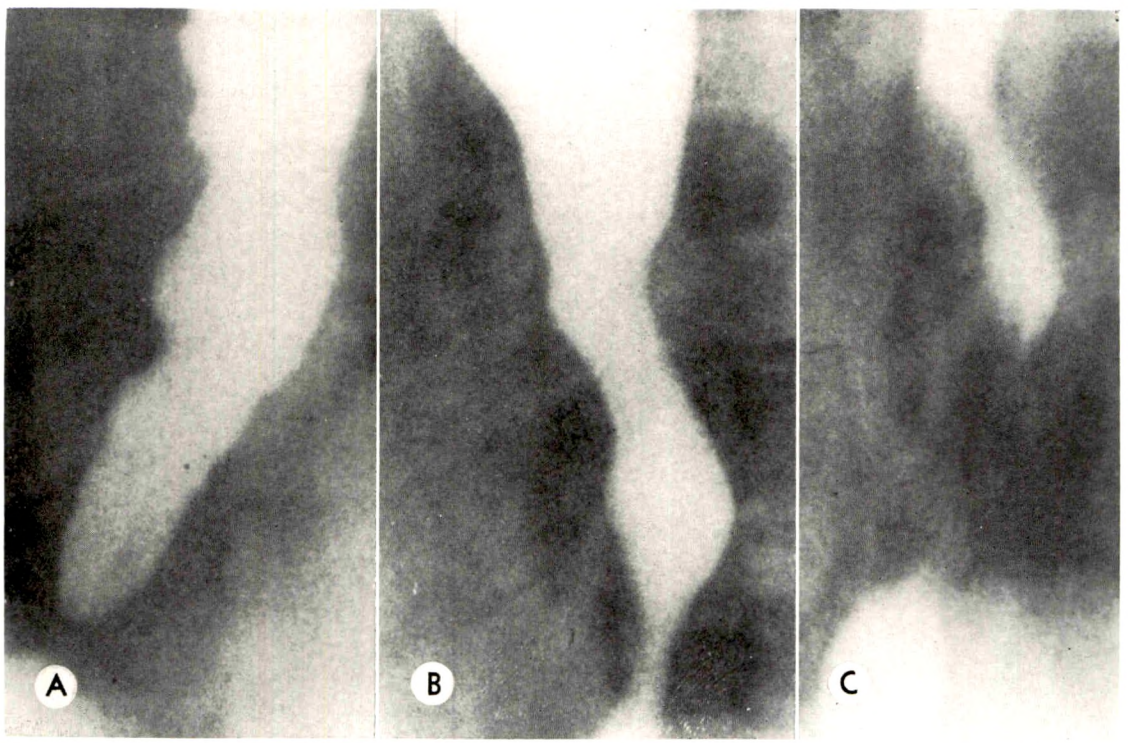


Fig. 1. Selected frames from a cine esophagography in a patient with moderate esophageal dilatation. (*A*) Baseline study. (*B* and *C*) Following the subcutaneous injection of 2.5 mg. of Mecholyl. Some motor activity occurred in the distal esophagus with a decrease in esophageal dilatation. The full response, however, only resulted after a 5 mg. dose (*C*). The esophagus has emptied by adoral regurgitation of barium after long segmental esophageal contractions.

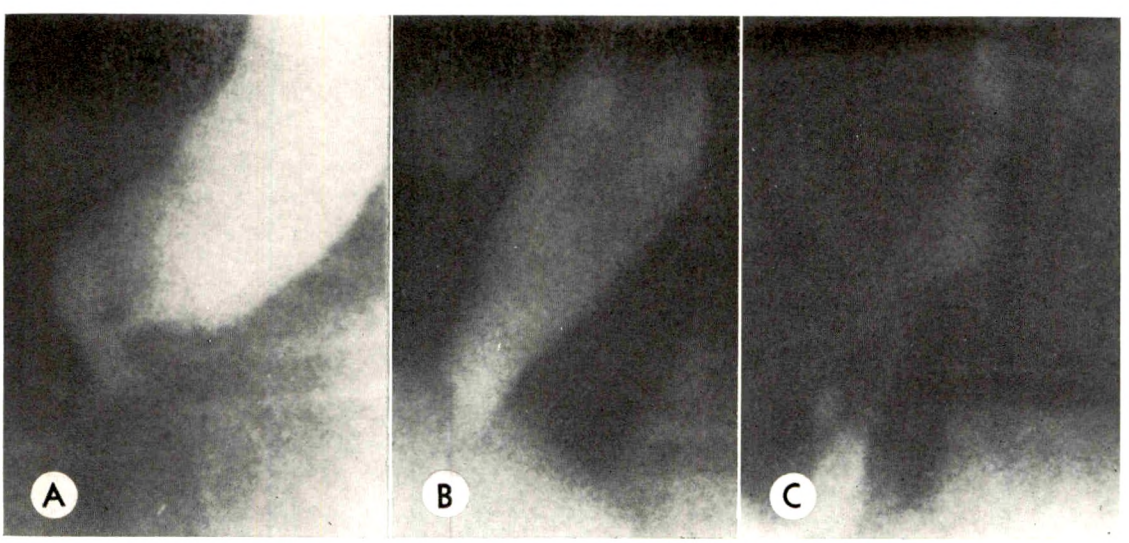


Fig. 2. Selective frames from a cine esophagography in a patient with more marked esophageal dilatation. (*A*) Baseline examination. (*B*) After subcutaneous injection of 5 mg. of Mecholyl. Here the first observable motor response of the distal esophagus occurred. (*C*) After 7.5 mg. of Mecholyl. A diagnostic response with a lumen-obliterating contraction followed the injection.

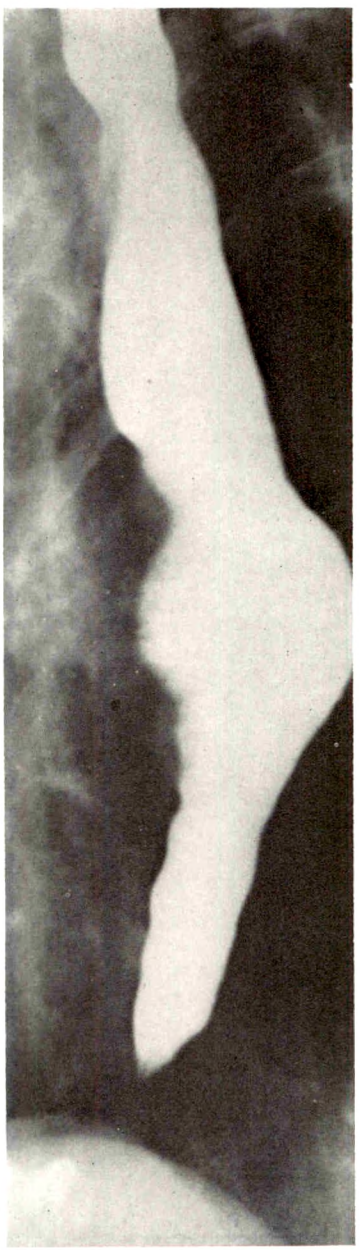


Fig. 3. Barium esophagogram. The moderate esophageal dilatation and achalasia are representative of the patients studied in this series by Mecholyl esophagography.

benign peptic stricture, and diffuse esophageal spasm (Fig. 3). The response to Mecholyl stimulation in all of this latter group has been demonstrated by Kramer and Inglefinger[7] and confirmed by Hightower and his colleagues[5] to be the same as in normal people. The ease and simplicity with which the test can be performed supply the diagnostic radiologist with additional evidence on which to base his diagnosis.

SUMMARY

The subcutaneous administration of Mecholyl (acetyl-β-methylcholine chloride) combined with cinefluorography offers an easy and accurate assessment of achalasia.

The technique and differential diagnosis are described.

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ROENTGENOGRAPHIC APPEARANCE OF THE DISTAL ESOPHAGUS AND THE STOMACH AFTER HIATAL HERNIA REPAIR*

By HIND S. TEIXIDOR, M.D., *and* JOHN A. EVANS, M.D.
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IT is generally accepted that the symptoms of a hiatal hernia are mostly due to the reflux of gastric secretion into the lower esophagus leading to esophagitis. Therefore competence of the cardia is of utmost importance. Factors that contribute to such competence include: (1) an intrinsic sphincter which is believed to be physiologic rather than anatomic; (2) the pinchcock action of the right crus of the diaphragm which enhances the effect of an intrinsic sphincter; (3) the acute angle of entry of the esophagus into the stomach; and (4) an intra-abdominal segment of esophagus.[9] In the treatment of hiatal hernia, various surgical procedures have been devised for the restoration of these anatomic features. Some have proven to be more effective than others.

It is not our purpose here to evaluate the effectiveness of such treatment, but to show the roentgenologic appearances of the distal esophagus and stomach following such procedures. Familiarity with the resulting filling defects and anatomic alterations will not only help the radiologist identify the

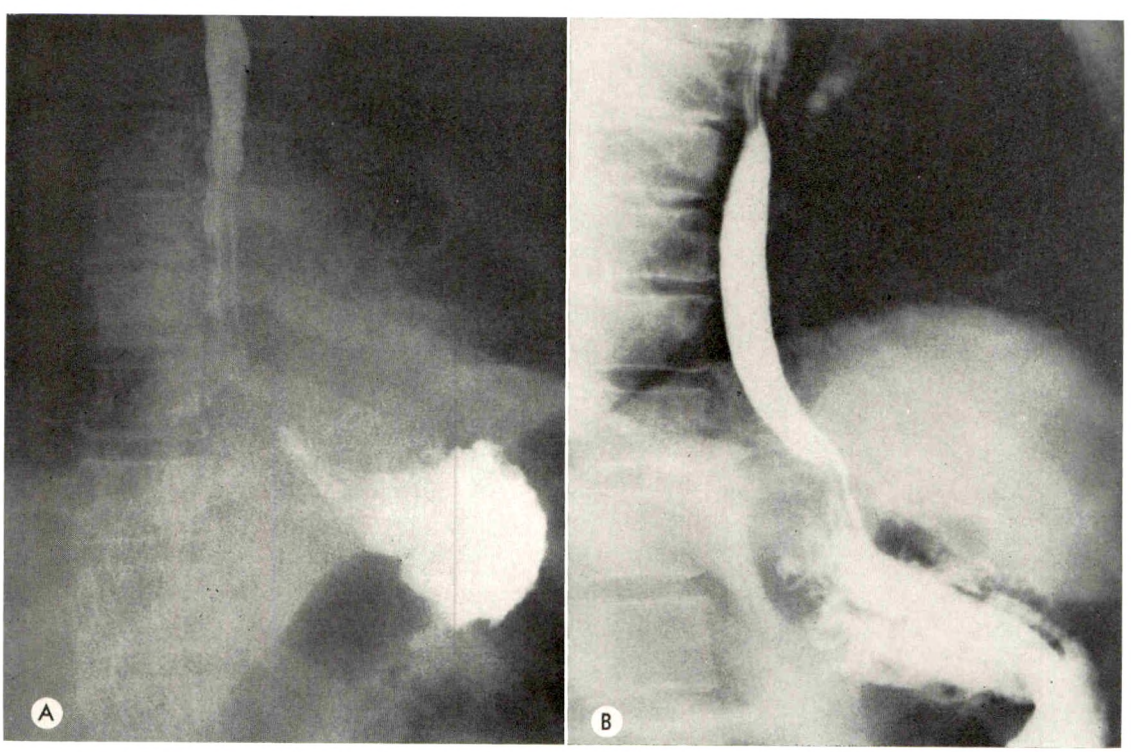


FIG. 1. Allison repair, 1 week postoperatively. (*A*) Anteroposterior and (*B*) lateral views. Note anterior angulation of the distal esophagus.

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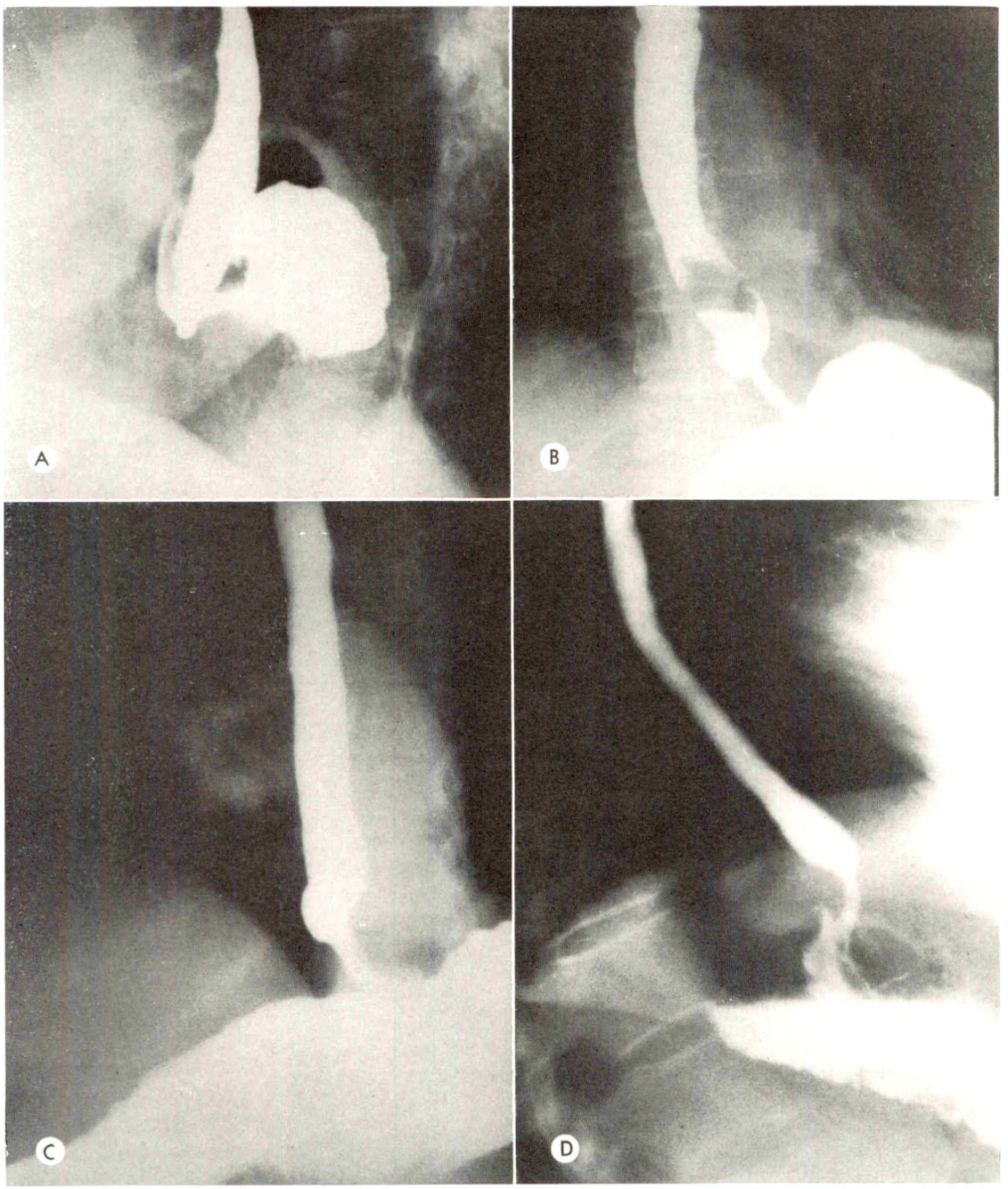


FIG. 2. (*A*) Combined paraesophageal and sliding hiatal hernia, preoperative. (*B* and *C*) Ten days after an Allison repair. A 2.5 cm. narrowed segment with asymmetric edges has resulted. (*D*) Lateral view. Note similarity to appearance in Figure 1*B*.

type of surgical repair roentgenographically, but will also make him avert diagnostic pitfalls.

MATERIAL

Fifty-five cases that had a hiatal hernia repair in The New York Hospital between

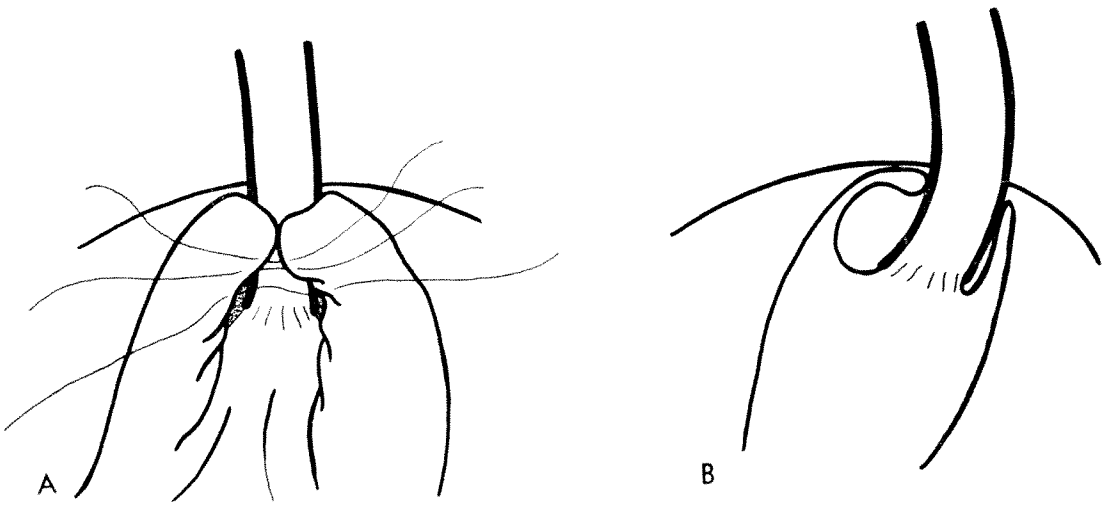


FIG. 3. Nissen procedure. (*A*) Folds of fundus are wrapped around the esophagus then sutured to each other. (*B*) Sagittal section of final stage showing relationship of fundus to esophagus.

1969 and 1971 were reviewed. They all had hiatal hernia and esophagitis diagnosed roentgenologically and by esophagoscopy. The operations were performed by several surgeons and various surgical procedures were used. Fifteen cases did not have postoperative roentgenologic examinations and therefore were dropped from this study. The remaining 40 cases had their pre- and postoperative roentgenograms studied and the findings correlated with the operative procedure performed. Examples of typical and some unusual appearances are described and discussed.

SURGICAL PROCEDURES AFFECTING THE DISTAL ESOPHAGUS AND/OR FUNDUS OF THE STOMACH

I. ALLISON REPAIR

This consists of reducing the hernia below the diaphragm, excising the hernia sac, suturing the phrenoesophageal ligament to

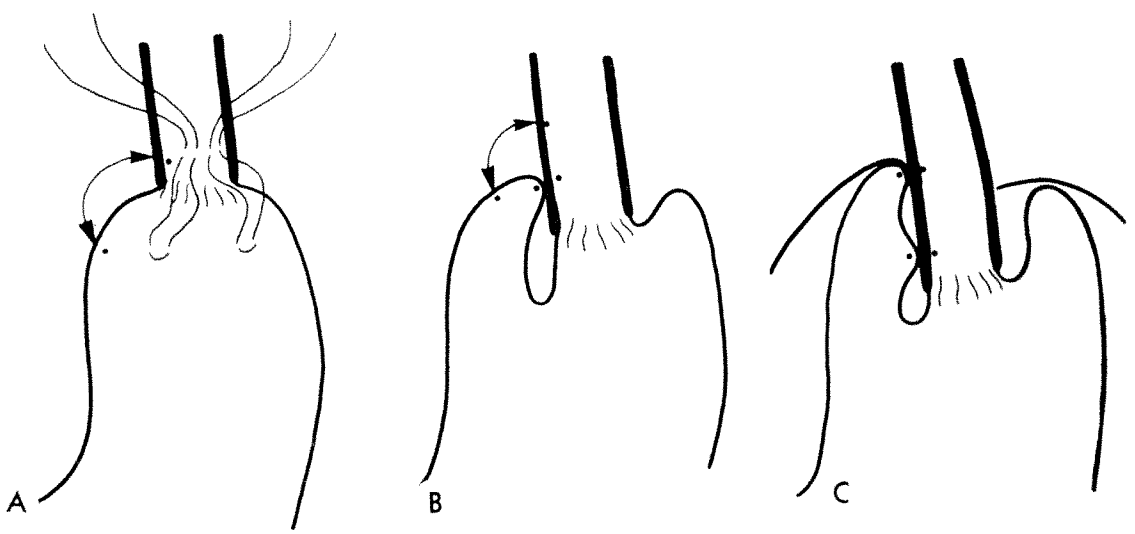


FIG. 4. Belsey Mark IV procedure. (*A*) Gastroesophageal sutures are placed at site of dots. (*B*) Sagittal section of stomach after having invaginated distal esophagus into fundus. (*C*) Sagittal section of final stage. Note similarity to appearance in Figure 3*B*.

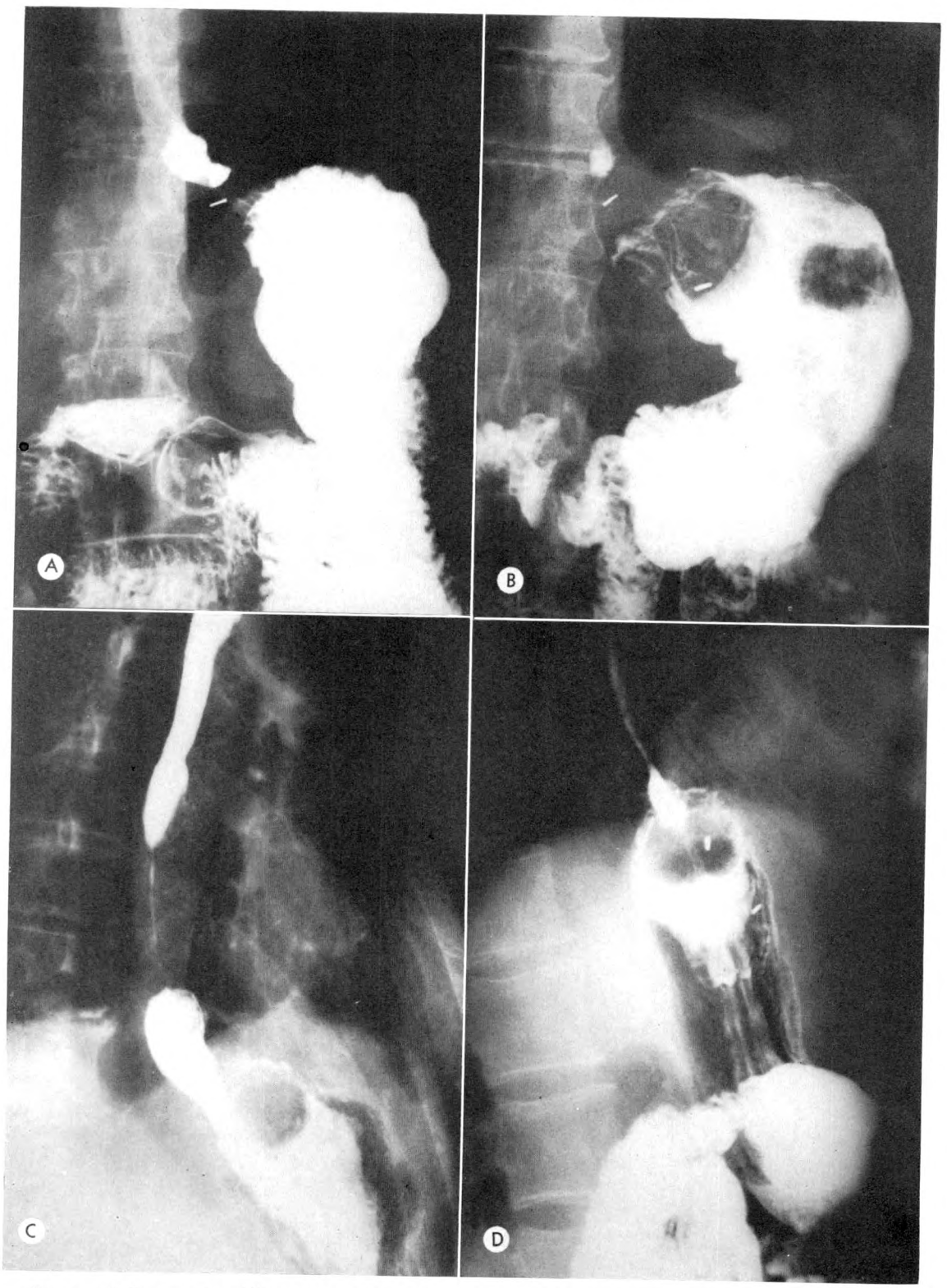


FIG. 5. (*A*) Status after Allison procedure and vagotomy. (*B*) Status 13 days after Nissen procedure. (*C* and *D*) Status 3 months later. Note only slight decrease in size of pseudotumor.

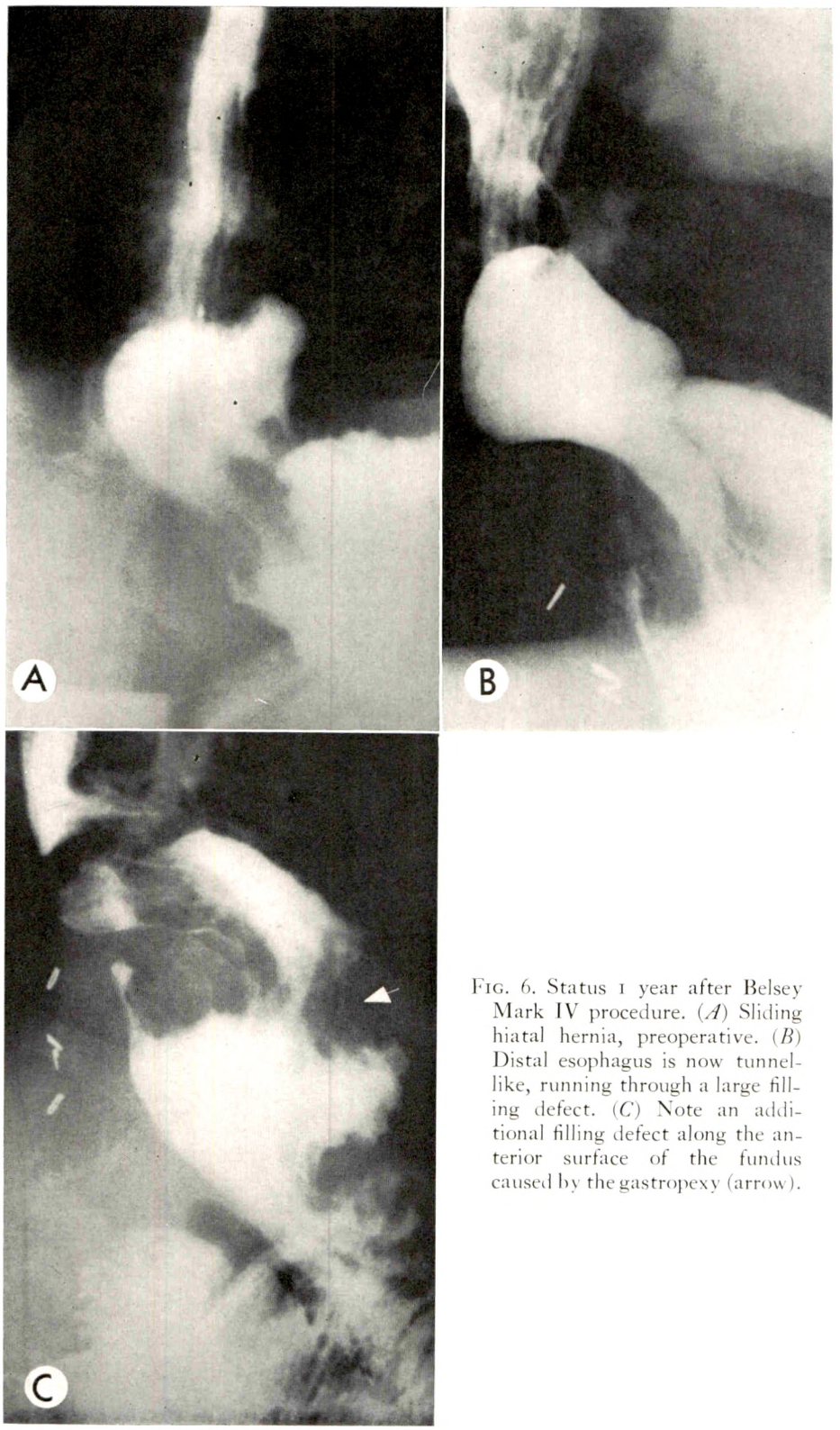


Fig. 6. Status 1 year after Belsey Mark IV procedure. (*A*) Sliding hiatal hernia, preoperative. (*B*) Distal esophagus is now tunnel-like, running through a large filling defect. (*C*) Note an additional filling defect along the anterior surface of the fundus caused by the gastropexy (arrow).

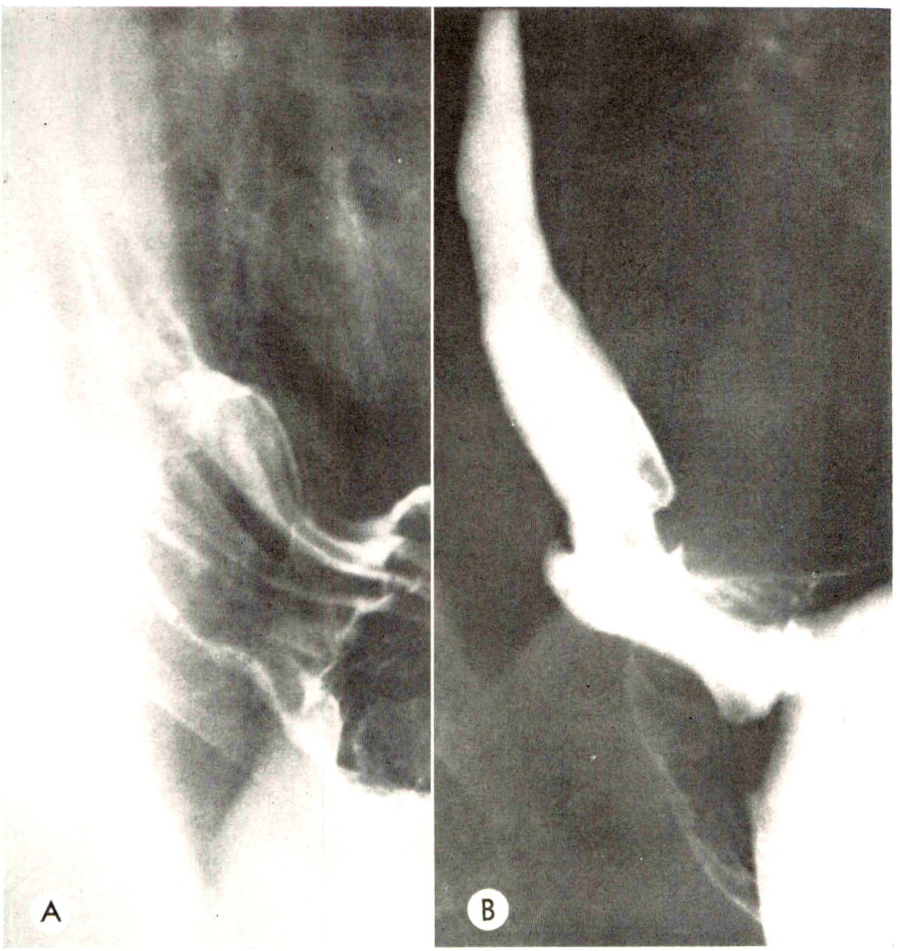


Fig. 7. (*A*) Sliding hiatal hernia, preoperative. (*B*) Thirteen days after Belsey Mark IV procedure. Note appearance of the subdiaphragmatic esophagus and absence of a "pseudotumor."

the undersurface of the diaphragm in front and to the left of the cardia and narrowing the hiatus to the desired size by suturing the right crus of the diaphragm behind the esophagus.[1,8] Several modifications of this procedure are also used. Figure 1, *A* and *B*, shows a successful anatomic result of a sliding hiatal hernia after an Allison repair. In the majority of cases the Allison and modified Allison operations produce no roentgenographic changes in the distal esophagus and the diagnosis of surgical interference may not be made roentgenographically. Occasionally, an unusual appearance of the distal esophagus is seen. Figure 2, *B–D*, shows a postoperative status of a repaired combined sliding and paraesophageal hiatal hernia. While the lateral view (Fig. 2*D*) is very similar to that in Figure 1*B*, the anteroposterior and the right posterior oblique views (Fig. 2, *B* and *C*) show a 2.5 cm. narrowed segment at the distal esophagus which may well be mistaken for a constricting esophageal lesion. Excessive narrowing of the hiatus may have been responsible for this appearance. However, the patient did not complain of any dysphagia up to 7 months postoperatively. She was subsequently lost to follow-up.

II. Fundoplication Procedures
Nissen and Belsey Mark IV

Although the Allison repair is considered by some surgeons as the method of choice

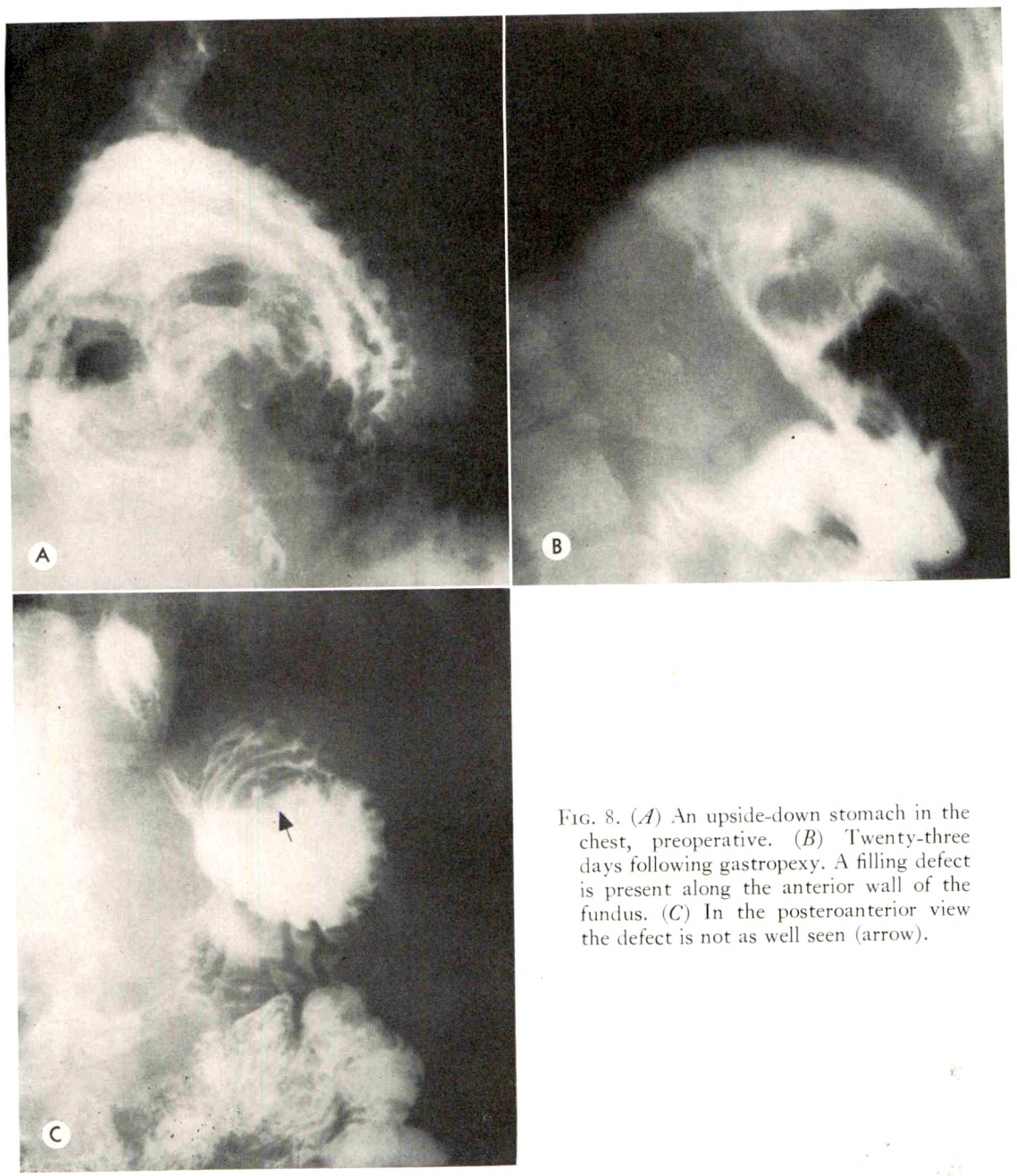


FIG. 8. (*A*) An upside-down stomach in the chest, preoperative. (*B*) Twenty-three days following gastropexy. A filling defect is present along the anterior wall of the fundus. (*C*) In the posteroanterior view the defect is not as well seen (arrow).

in adults,[8] it was found to give unsatisfactory results by others.[9,11] A recurrence rate and unrelieved symptoms of 28 to 50 per cent of cases is reported after such a repair.[6,7,9] Fundoplication was then devised and this tends to "overcorrect" the hernia in order to prevent recurrences.

The Nissen procedure consists of mobilizing and pulling the distal esophagus down into the abdominal cavity. The fundus of the stomach is then wrapped around this esophageal segment and the folds fixed with sutures[6] (Fig. 3, *A* and *B*).

In the Belsey Mark IV fundoplication, the distal esophagus is invaginated into the fundus of the stomach which is then wrapped around it and both are secured by sutures passing from the seroso-muscular

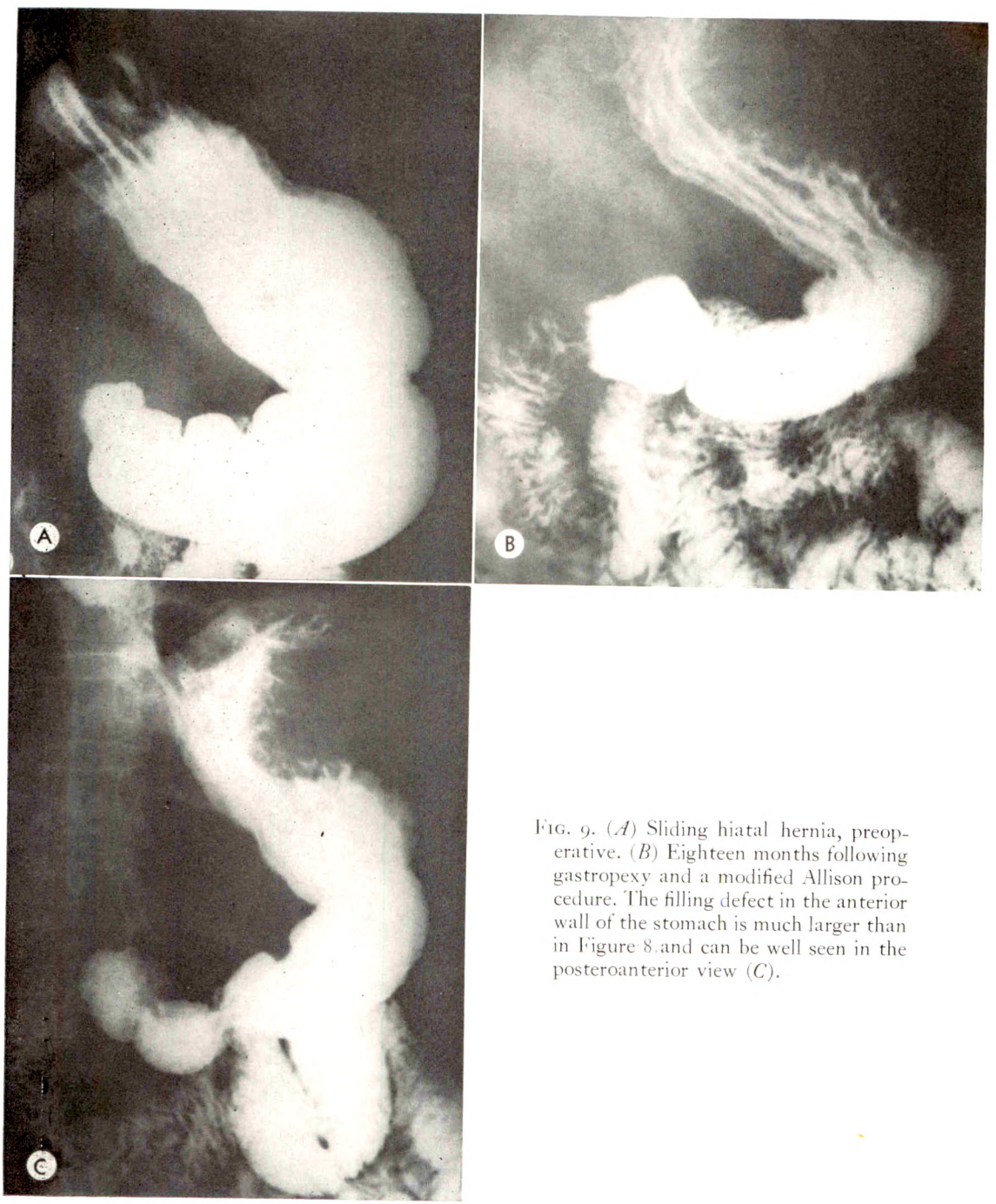


FIG. 9. (*A*) Sliding hiatal hernia, preoperative. (*B*) Eighteen months following gastropexy and a modified Allison procedure. The filling defect in the anterior wall of the stomach is much larger than in Figure 8 and can be well seen in the posteroanterior view (*C*).

layer of the stomach to the muscular layer of the esophagus. A second row of sutures is put in the same manner but these pass through the diaphragm for fixation (Fig. 4, *A–C*).

The Nissen procedure is more often performed transabdominally and the Belsey Mark IV transthoracically, but these approaches can be used interchangeably in both procedures depending on the patient's particular medical problem. However, the major technical difference between the 2 is

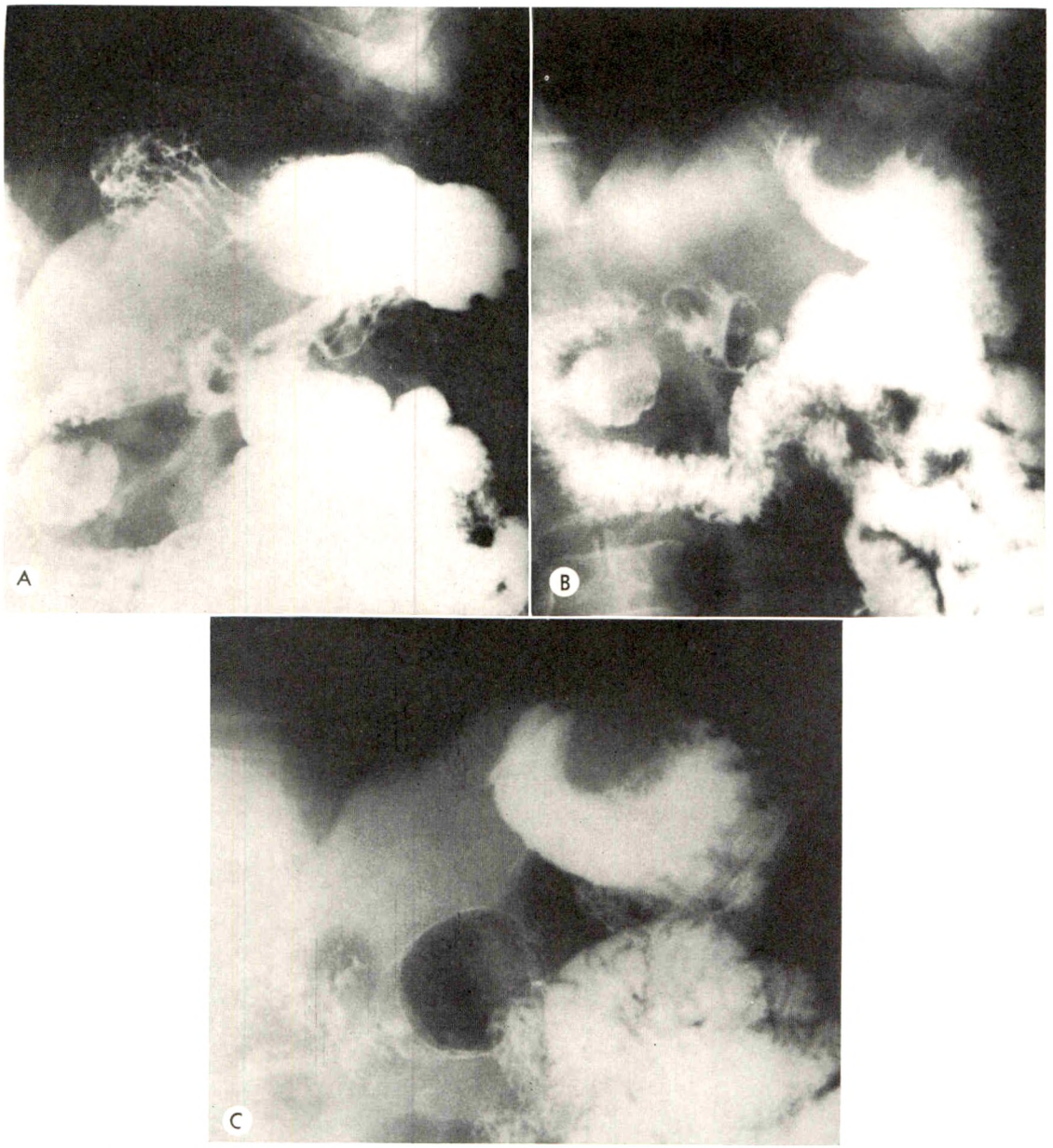


Fig. 10. (*A*) Sliding hiatal hernia, preoperative. (*B*) Postoperative anteroposterior view, after fixation of the fundus to the undersurface of the diaphragm. Filling defect in the upper part of the fundus. (*C*) Right posterior oblique view. Same filling defect seen again.

that in the former, suturing is gastrogastric, while in the latter it is gastroesophageal. The end result of both is very similar, an intra-abdominal segment of esophagus is produced and is surrounded by a mound of tissue consisting of the plicated fundus. In the Belsey Mark IV method this mound surrounds the distal esophagus more concentrically, while it is located more anteriorly in the Nissen repair. Roentgenographically a filling defect is produced which may be quite large in the immediate postoperative period because of the accompanying tissue edema. After the postoperative swelling subsides, the defect will decrease in size and will presumably re-

main unchanged thereafter.[5] A "pseudotumor" of the fundus has been reported following both procedures.[4,5] That such a filling defect may be mistaken for a tumor of the stomach is evident. Figure 5, *B–D*, shows the postoperative status following a Nissen repair. The large filling defect in the fundus of the stomach is located mostly anterior to the subdiaphragmatic esophagus. It is smoothly demarcated and well circumscribed. The surrounding mucosa is intact. This patient had had an Allison repair and vagotomy previously (Fig. 5*A*). The Nissen procedure was done for recurrence of gastroesophageal reflux and esophagitis. Two postoperative studies about 3 months apart are available and a slight decrease in the size of the "pseudotumor" is seen on the latter examination (Fig. 5, *C* and *D*). Figure 6, *A–C*, shows a patient who underwent a Belsey Mark IV repair and gastropexy. The large fundal "pseudotumor" is slightly irregular in contour and the esophageal "tunnel" runs through it rather than along its side. Such a "pseudotumor" does not occur in every patient undergoing fundoplication, although it was reportedly present in 33 of 34 operated patients in the series of Ettinger *et al.*[5] Figure 7, *A* and *B*, shows another patient who underwent a Belsey Mark IV procedure. A tunnel-like subdiaphragmatic segment of esophagus is maintained, but no filling defect is seen around it. This is possibly explained by the lack of filling of the potential fundal space surrounding the invaginated esophagus.

III. GASTROPEXY

Fixing the stomach to the anterior abdominal wall under traction has been advocated as a surgical treatment for hiatal hernia.[3,6,13] Usually the lesser curvature, the fundus, or both, are tacked to the left

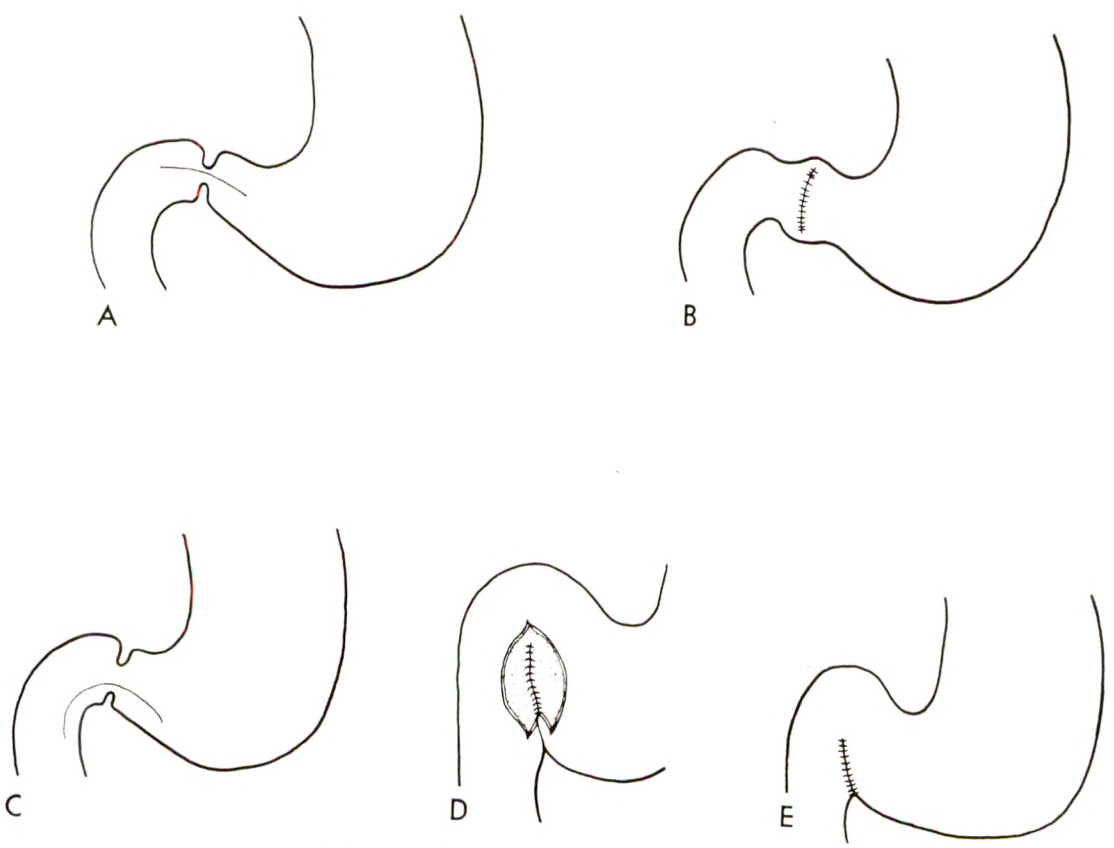


Fig. 11. (*A* and *B*) Heineke-Mikulicz pyloroplasty. (*C–E*) Finney pyloroplasty.

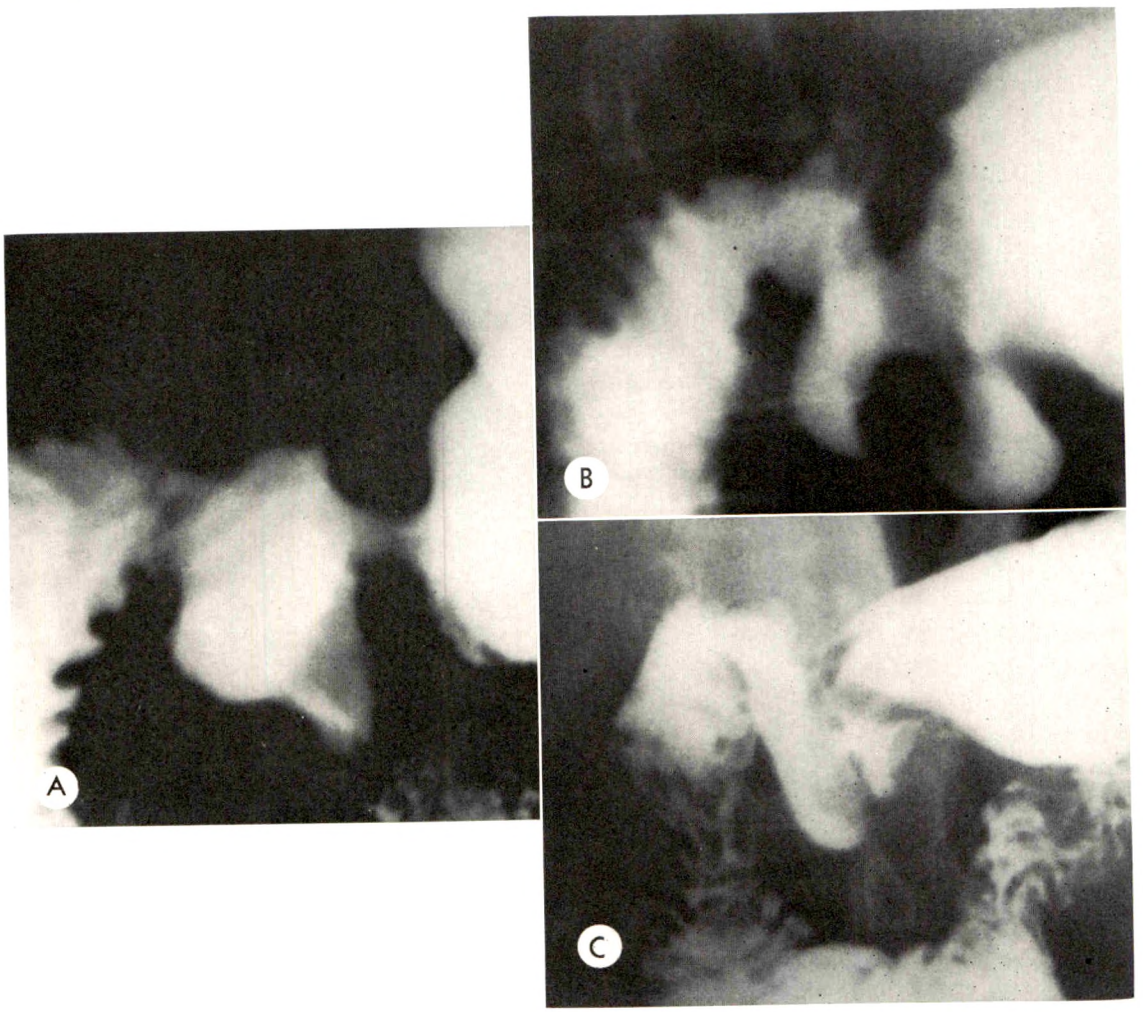


Fig. 12. Three different cases after Finney pyloroplasty. Note acute angle produced by the greater curvature side of the antrum and the first part of the duodenum. (*A* and *B*) Typical postpyloroplasty appearance. (*C*) Single large filling defect along greater curvature side of antrum.

rectus sheath with several sutures. The site of the gastrostomy tube has also been used for the same purpose.[3] Gastropexy is done with or without other corrective procedures (Fig. 6*C*) and has proven to be an effective mode of therapy even when performed alone. It has been used mostly for the correction of paraesophageal hernias and is advised for the very elderly and the poor-risk patient.[6] Roentgenologically a constant filling defect is seen along the anterior wall of the fundus, the mucosa in its vicinity being intact. It may give the impression of being caused by an extrinsic mass except that it is not sharply marginated. Its exact size and location depend on the part of the stomach selected by the surgeon for the gastropexy and on the number of sutures used (Fig. 8, *A–C*; and 9, *A–C*). Occasionally, the fundus of the stomach may be fixed to the undersurface of the diaphragm rather than to the abdominal wall. This produces a similar filling defect which is located higher on the fundus and more laterally (Fig. 10, *A–C*).

Indirect Surgical Procedures in the Treatment of Hiatal Hernia

1. Vagotomy and Pyloroplasty

Gastric hyperacidity has been found to have a relationship to hiatal hernia. There-

fore, reducing the peptic activity of gastric secretions and improving gastric drainage by vagotomy and pyloroplasty, respectively, have been advocated by some surgeons for the treatment of hiatal hernia.[9] However, severe reflux esophagitis has been found to occur in patients with low acidity or with achlorhydria,[11] so that these procedures, when performed as the only mode of therapy, have shown unpredictable results. Since such an approach has actually been used as a surgical treatment of hiatal hernia, we are including the resulting postpyloroplasty appearance in this discussion.

In our series, vagotomy and pyloroplasty have been performed in conjunction with other corrective procedures on the esophagus, most often an Allison or a modified Allison repair. All of these cases had had a normal antrum and duodenal bulb preoperatively; therefore, presenting an opportunity of studying the roentgenologic appearance of the postpyloroplasty status without any preceding deformity of peptic ulcer disease.

The 2 commonly used types of pyloroplasty are the Heineke-Mikulicz and the Finney procedures. In the former a longitudinal incision 3–4 cm. on either side of the pylorus is made and then is closed transversely (Fig. 11, *A* and *B*). The latter consists of a U-shaped incision in the gastro-duodenum about 10 cm. in length, the pyloric canal being its center. The pyloric end of the stomach is then anastomosed to the first part of the duodenum and the anterior walls of the incision are approximated and sutured (Fig. 11, *C–E*). The object of both types is to enlarge the outlet of the stomach.

Roentgenographically the only distinguishing feature between these 2 procedures is that in the Finney type the proximal part of the duodenum bears a constant and close relationship to the antrum of the stomach because these 2 parts are actually anastomosed to each other, thus forming an acute angle (Fig. 12, *A–C*). No such relationship is present in the Heineke-Mikulicz pyloroplasty (Fig. 13, *A–C*). The deformity of the antrum itself does not have a characteristic appearance in the 2 procedures and may actually be very variable. Bloch and Wolf[2] have described in detail the typical configuration of the postpyloroplasty antrum. This consists of a patulous gastroduodenal channel with 2 broad ridges on either side followed by 2 pseudodiverticula (Fig. 12, *A* and *B*). The ridges, marked by surgical clips, have been found to represent the suture line of the pyloroplasty incision. We have found a more atypical appearance in our group of cases where a predominant single filling defect is present on the lesser or on the greater curvature side of the antrum (Fig. 12*C*; and 13, *A–C*). This prob-

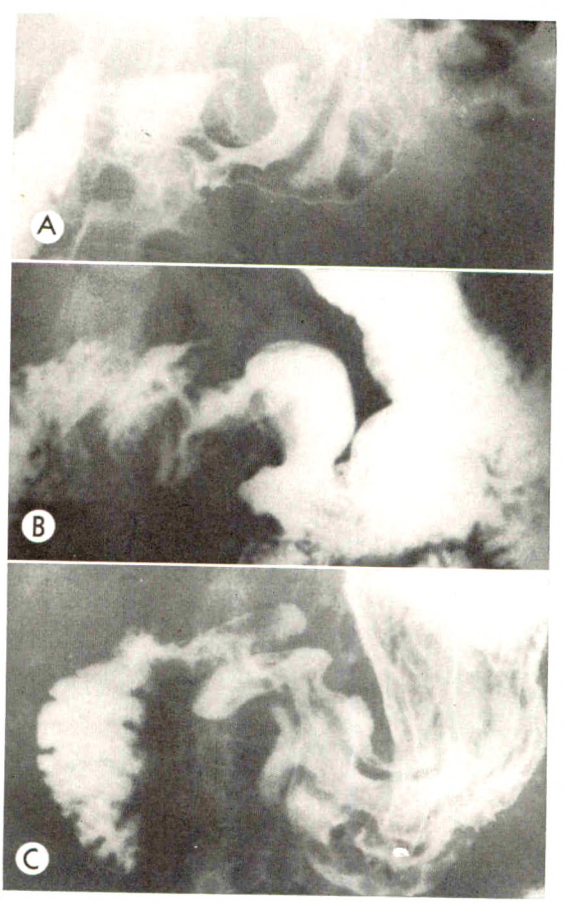


FIG. 13. Three different cases after Heineke-Mikulicz pyloroplasty. (*A*) Constant filling defect along lesser curvature side of the pyloroduodenum. (*B* and *C*) Predominant filling defect along greater curvature side of antrum.

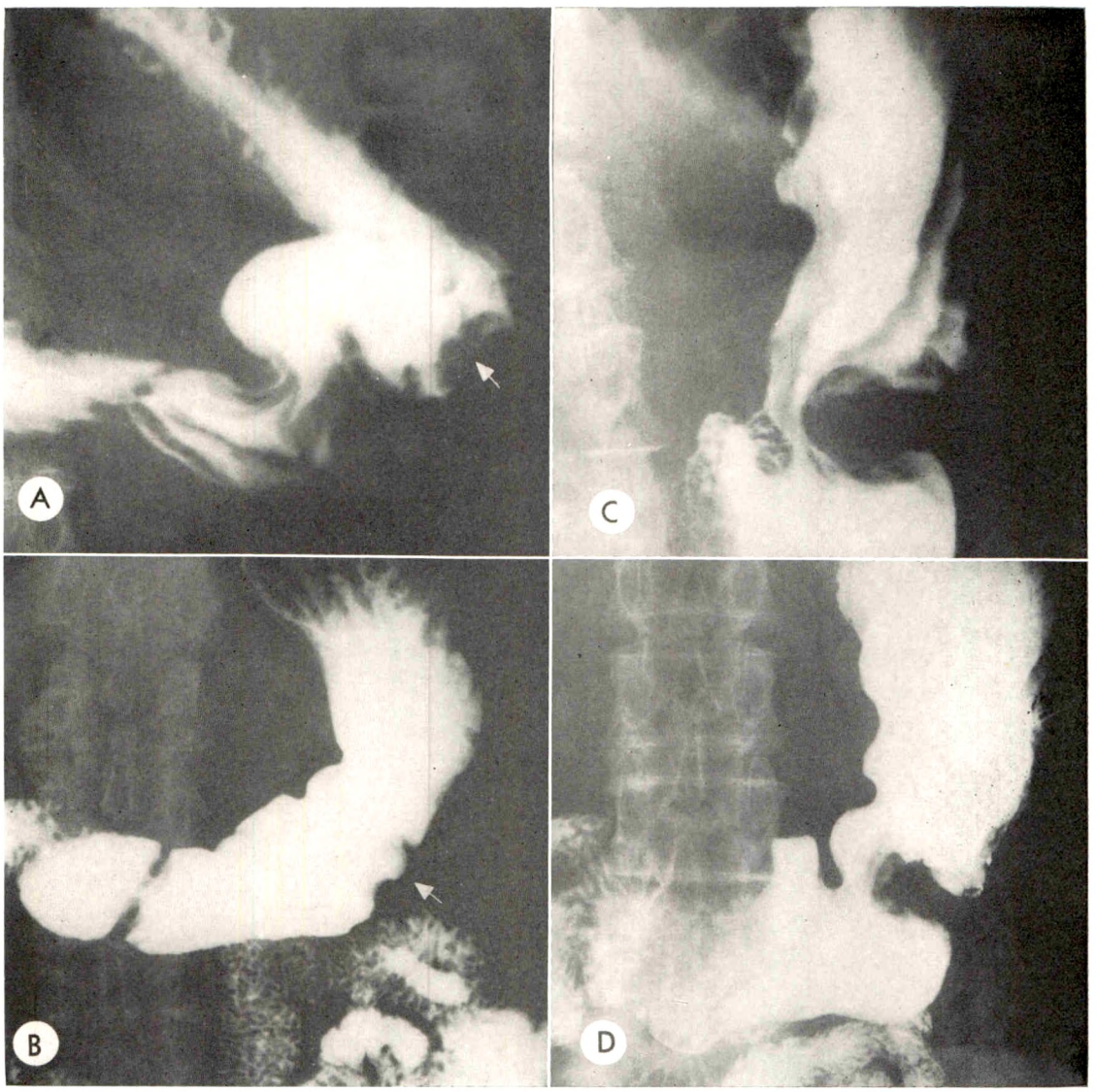


Fig. 14. Greater curvature defect after gastrostomy. (*A*) Three weeks after removal of the gastrostomy tube. The defect is fairly smooth (arrow). (*B*) Same case 6 months later. The defect simulates a gastric ulcer (arrow). (*C*) Another case, 2 weeks after removal of gastrostomy tube. (*D*) Same case 2 years later. The defect became smaller but more irregular.

ably represents a single "ridge" which has resulted from a more eccentric pyloroplasty incision. The length of the incision may also account for such variability.[10]

If a history of previous surgery is not available, such atypical filling defects may present a diagnostic problem to the radiologist. A benign tumor can hardly be excluded in some cases (Fig. 13, *A* and *C*). Ulcers have also been found difficult to rule out from such sites,[2,12] as is illustrated in a case (Fig. 12*C*). (In fact, the latter patient has never had an ulcer.)

II. GASTROSTOMY

Occasionally a gastrostomy is performed in conjunction with vagotomy and pyloroplasty in order to improve gastric drainage and prevent gastroesophageal reflux in the immediate postoperative period. A perma-

nent filling defect along the greater curvature side of the stomach has been found in some of our cases. This defect is usually smooth and the surrounding mucosa is intact (Fig. 14, *A* and *C*). At times this defect is not as smooth as expected and may simulate an ulcer, pancreatic rest or neoplasm (Fig. 14, *B* and *D*).

CONCLUSION

Several surgical procedures have been devised for the treatment of the symptomatic hiatal hernia. These have been found roentgenologically to produce a variety of anatomic alterations, some of which have a typical appearance and are easily diagnosed. However, others may have unusual appearances and may simulate tumors or ulcers.

Postoperative roentgenologic examination should, therefore, be obtained routinely in order to provide a baseline for future reference.

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TUMORS OF THE DIAPHRAGM*

By LARRY S. ANDERSON, M.D., *and* JOHN V. FORREST, M.D.
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TUMORS of the diaphragm are rare lesions which are often difficult to assess and classify either clinically or roentgenographically. Using the word tumor in its broadest sense to mean a swelling, this paper will classify these lesions of the diaphragm and present some typical roentgenographic examples.

CLASSIFICATION

Diaphragmatic tumors may be divided into the following groups: (1) primary benign neoplasms; (2) primary malignant neoplasms; (3) secondary malignant neoplasms; (4) cysts; (5) inflammatory lesions; and (6) endometriosis.

1. PRIMARY BENIGN NEOPLASMS

Any of the normal tissue components of the diaphragm may give rise to a benign neoplasm. Lipomas, fibromas, angiofibromas, neurofibromas and neurilemmomas are the most common types.[7,22,26,29] Tissue types not normally found in the diaphragm may also produce benign tumors in this structure. Adrenal cortical adenoma, liver cell adenoma, chondroma, hamartoma and mesothelioma have all been found in the diaphragm.[15,23]

The roentgenographic appearance is nonspecific and usually consists of a small irregularity of the diaphragm (Fig. 1). Calcification, although generally associated with an inflammatory lesion, has been demonstrated roentgenographically in an angiofibroma.[29] Benign neoplasms are often found only at postmortem examination.

2. PRIMARY MALIGNANT NEOPLASMS

The majority of primary malignant tumors found in the diaphragm are of fibrous tissue origin (fibrosarcoma, fibromyosarcoma, fibroangioendothelioma) or undifferentiated sarcomas. Other less common malignant tumors include mixed cell sarcoma, myosarcoma, rhabdomyosarcoma, neurofibrosarcoma, hemangioendothelioma, hemangiopericytoma, leiomyosarcoma, malignant synovioma and mesenchymoma.[1,8,22,23]

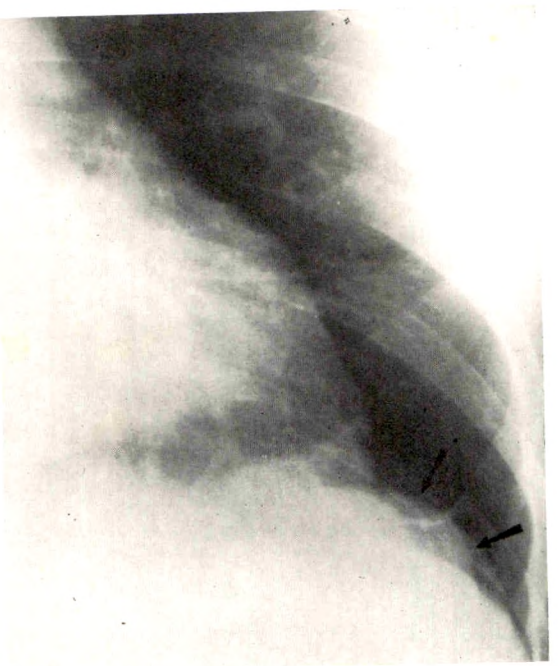


FIG. 1. Lobulation of the lateral aspect of the left hemidiaphragm (arrows) shown to be a lipoma at autopsy.

Of the reported primary tumors of the diaphragm, malignant neoplasms predominate in a ratio of about 60:40.[23] The majority of patients with primary malignant diaphragmatic tumors are symptomatic with pleuritic chest pain or pain referred to the epigastrium.[29]

Local roentgenographic manifestations are of an enlarging mass which abuts on

* Presented as a Scientific Exhibit at the Seventy-third Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 3–6, 1972.
From the Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

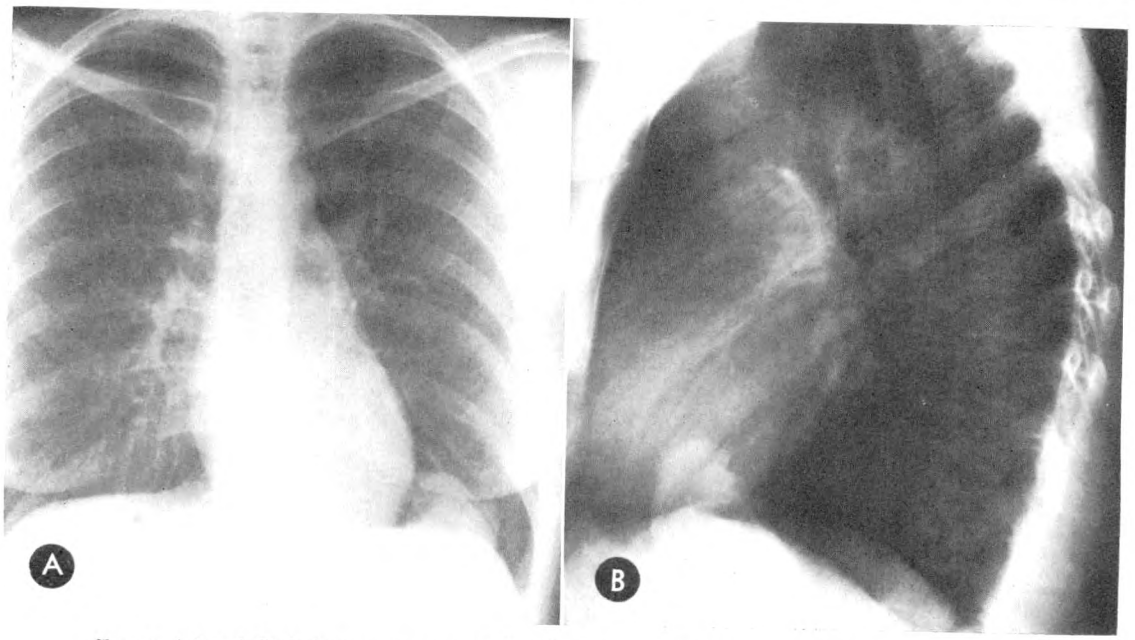


Fig. 2. (*A* and *B*) Leiomyosarcoma of the diaphragm. (Courtesy of Dr. Hyman Senturia, Jewish Hospital, St. Louis, Mo.)

the diaphragm. The mass usually maintains an extrapleural configuration (Fig. 2, *A* and *B*).

3. Secondary Malignant Neoplasms

Secondary malignant neoplasms of the diaphragm may be due to direct invasion from adjacent lesions or metastatic spread *via* vascular channels.[4,11,20,21,28] Metastatic disease will roentgenographically resemble primary malignant diaphragmatic tumors and the knowledge of the known primary tumor or a pathologic specimen will be needed to make the distinction.

Examples of direct invasion of the diaphragm recorded in the Departments of Pathology and Surgical Pathology, Washington University Medical Center, include: carcinoma of the lung, stomach, pancreas, kidney, adrenal, and colon; primary and secondary malignancies of the liver (Fig. 3); chondrosarcoma; disseminated reticulum cell sarcoma; and Hodgkin's disease.

Bloodborne metastatic implants are rare. In the few reported cases it is not clear whether the implants were visualized

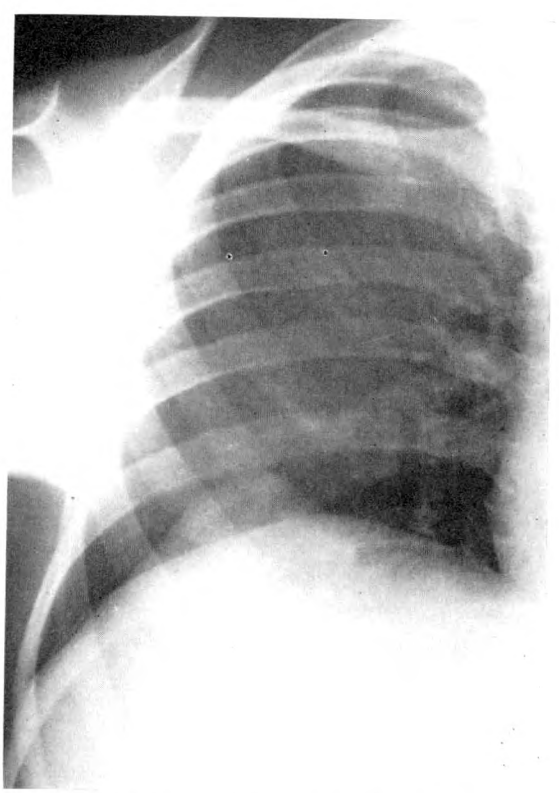


Fig. 3. Diaphragmatic invasion by a primary malignant liver tumor in a 6 year old boy.

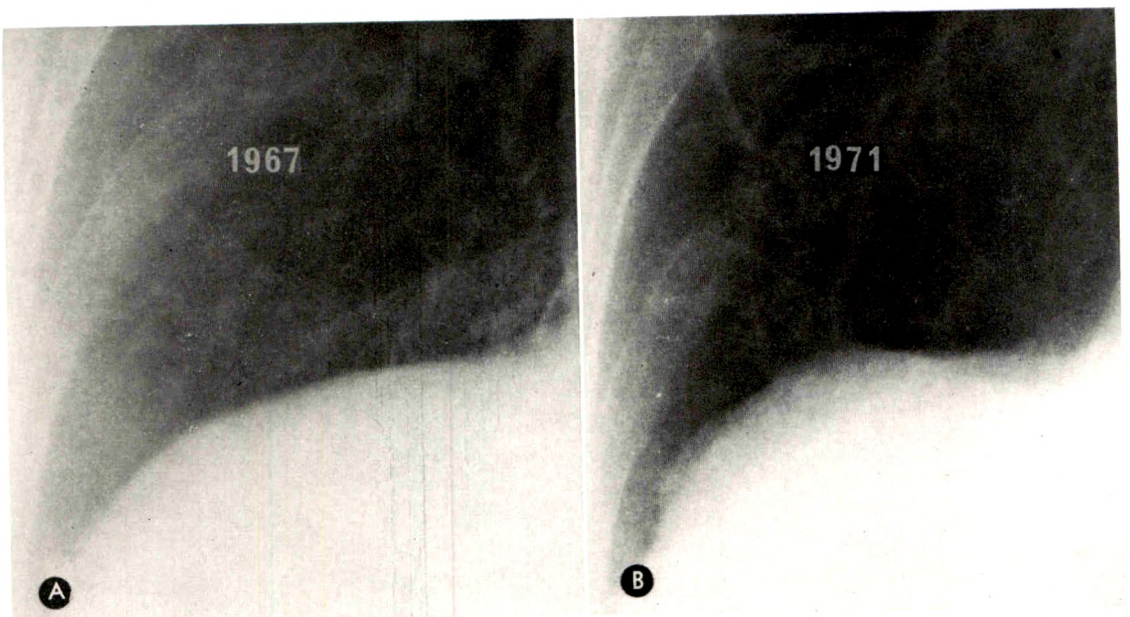


Fig. 4. Hematogeneous metastasis from an ovarian carcinoma. (*A*) Roentgenogram from 1967 showing a normal diaphragmatic contour. (*B*) Roentgenogram from 1971 showing interval appearance of a lobulated tumor—metastatic ovarian carcinoma confined to the intramuscular portion of the diaphragm.

roentgenographically as well as microscopically. They are usually found in cases of widely disseminated disease (Fig. 4, *A* and *B*).[4,21]

Abdominal malignancies invading the diaphragm are usually limited by the pleura. However, if the pulmonary parenchyma is invaded, the roentgenographic appearance is characteristic, since the margin of the lesion is irregular and ill-defined,

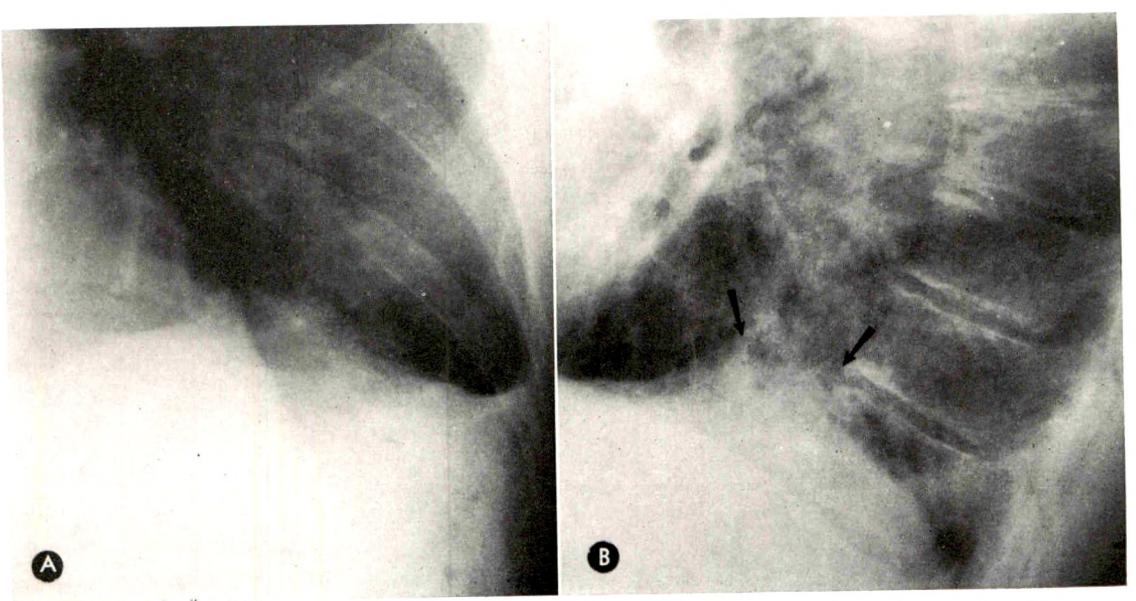


Fig. 5. (*A* and *B*) Splenic flexure colon carcinoma invading the lung through the diaphragm. The ill-defined margin of the advancing edge (arrows) suggests the lung involvement.

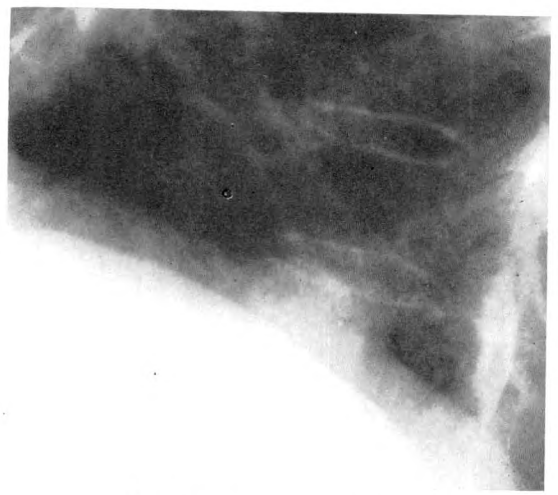


Fig. 6. Diaphragmatic cyst.

similar to the border of a primary lung cancer (Fig. 5, *A* and *B*).

4. CYSTS

Cystic lesions of the diaphragm may be acquired or congenital. Trauma with resulting hematoma and degeneration has been reported as a cause of diaphragmatic cysts.[18] Simple cysts and fibrous lined cysts make up another group, the origin of which is uncertain (Fig. 6).[7]

Congenital cysts include those of pulmonary origin which histologically resemble bronchogenic cysts or sequestrations; teratoid cysts; and mesothelial lined cysts. The latter are thought to arise from embryonic rests of septum transversum and the primitive pleuropericardial and pleuroperitoneal coelomic membranes.[5,12,16]

5. INFLAMMATORY LESIONS

It is not uncommon for the diaphragm to be involved by an inflammatory process in the abdomen or adjacent organs. However, it is rare for an inflammatory process to present as a primary intradiaphragmatic abscess. Hydatid disease and tuberculosis rarely may cause inflammatory tumors of the diaphragm.[2,25,27,30]

Diaphragmatic involvement by echinococcosis is generally secondary to liver or lung involvement. It is only at surgery or necropsy that primary diaphragmatic origin is established.[30] Hydatid cysts may demonstrate irregular central, scattered

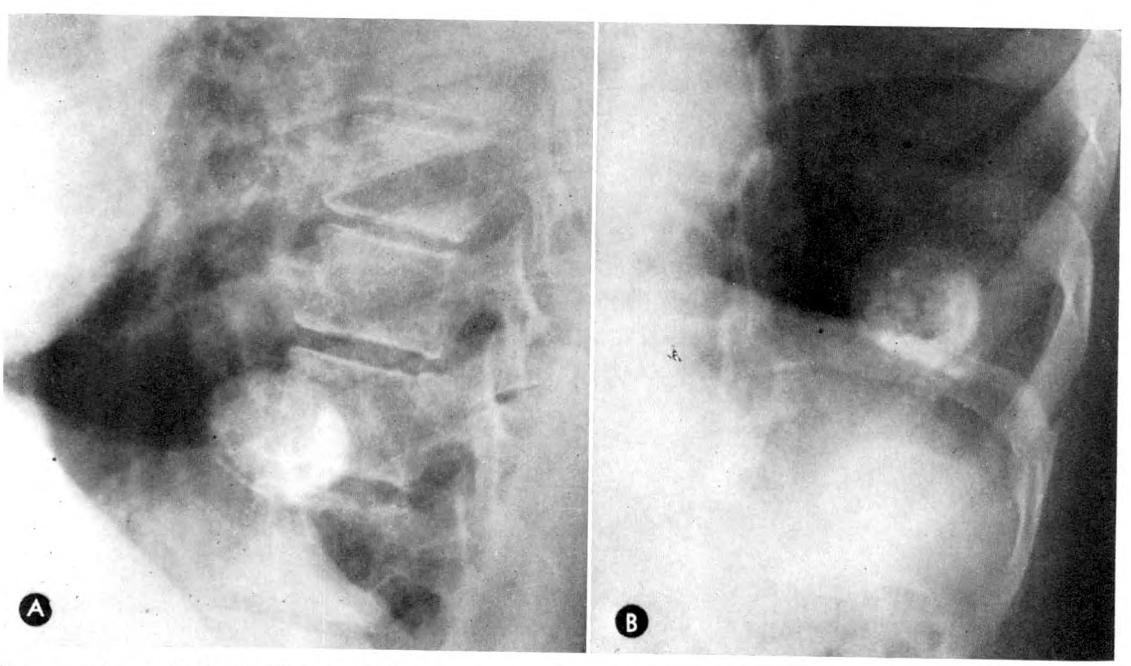


Fig. 7. (*A* and *B*) Hydatid cyst of the diaphragm with extensive calcification. (Courtesy of Dr. Federico Reiter, Universidad Catolica de Chile, Santiago, Chile.)

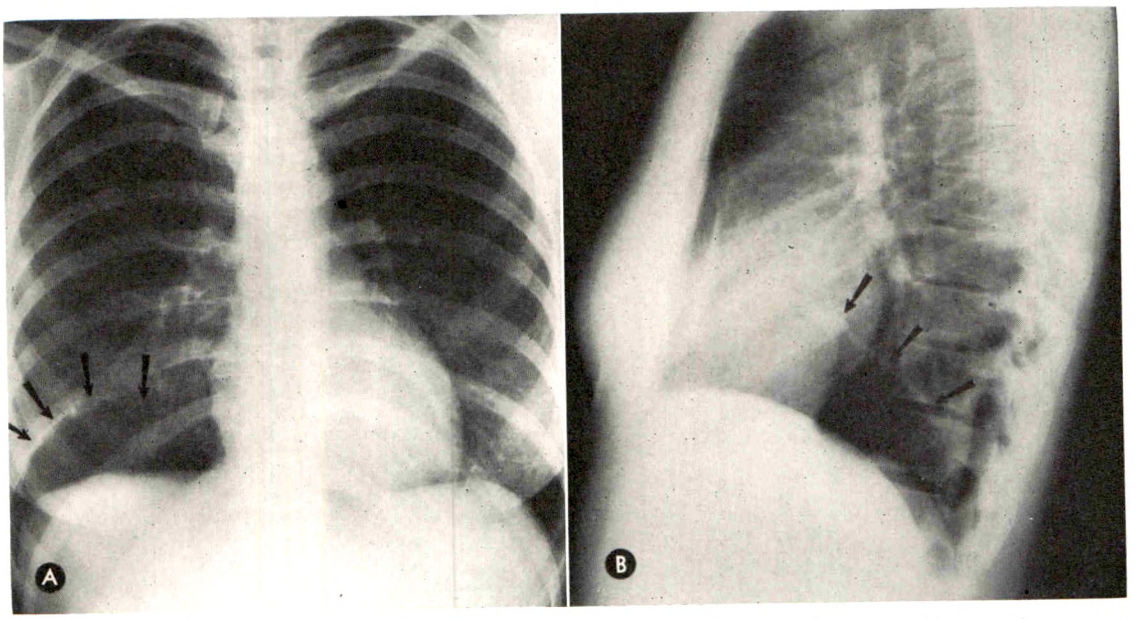


Fig. 8. (*A* and *B*) Diaphragmatic endometriosis in a 32 year old woman with 1½ year history of recurrent pneumothorax on the right side associated with menses. A right superior partial pleurectomy did not prevent pneumothoraces which then became loculated in a subpulmonic position (arrows). Subsequent resection of a portion of tendinous diaphragm demonstrated endometriosis associated with three small diaphragmatic perforations.

crescentic or rim-like calcifications and thus give a clue to the etiology on the roentgenogram (Fig. 7, *A* and *B*).[6]

6. ENDOMETRIOSIS

Although endometriosis of the diaphragm cannot be seen roentgenographically, the clinical history of recurrent episodes of spontaneous pneumothorax occurring on the right side in a woman at the time of her menses should suggest this diagnosis (Fig. 8, *A* and *B*).[9,10,17,19]

The pathophysiology of recurrent pneumothorax associated with endometriosis of the diaphragm is still unknown. Defects in the diaphragm have been associated with endometrial deposits; however, at surgery the pathology is not always found in these patients with catamenial pneumothorax.[17]

DISCUSSION

The roentgenographic appearance of a diaphragmatic tumor is generally nonspecific, and in most cases irregularity of the diaphragm is due to a normal variant of diaphragmatic lobulation or localized eventration. Intrathoracic lesions adjacent to the diaphragm and subdiaphragmatic conditions elevating or deforming the diaphragm are also seen commonly. The frequency with which these normal variants and juxtadiaphragmatic abnormalities occur and the rarity of diaphragmatic tumors make the discovery of true tumors even more difficult.

Old roentgenograms and sequential studies are most important in the evaluation of a diaphragmatic irregularity. The metastatic ovarian carcinoma pictured in Figure 4, *A* and *B*, was missed roentgenographically until careful comparison with old roentgenograms was made.

Fluoroscopy is the initial approach once a diaphragmatic abnormality has been noted on the standard chest roentgenogram. Position in relation to adjacent structures, movement of the diaphragm and alterations of contour with respiration and position are evaluated.

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We have had no experience with the use of diagnostic pneumothorax and pneumoperitoneum which, in the absence of adjacent pleural or peritoneal adhesions, would allow one to distinguish an intrinsic diaphragmatic lesion from one in adjacent structures.[1]

Laminagraphy, barium studies, intravenous urography, bronchography, angiography[13,14] and liver-spleen isotope scannings,[3,24] may be helpful in distinguishing diaphragmatic tumors from the lesions which mimic them.

Our general policy is to assume that a diaphragmatic irregularity is of no significance unless: (a) clinical symptomatology indicates a diaphragmatic lesion; (b) serial roentgenograms show a change; or (c) there is a definite tumor roentgenographically.

SUMMARY

Tumors of the diaphragm are classified into benign, primary malignant, secondary malignant, cystic, inflammatory and endometriosis.

Clinical correlation and sequential roentgenographic changes are emphasized.

Roentgenographic examples are presented including unusual cases such as direct extension into the lung by an abdominal malignancy, hydatid cyst of the diaphragm and catamenial pneumothorax associated with diaphragmatic endometriosis.

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EPIPHRENIC DIVERTICULA*

AN ANALYSIS OF 80 CASES

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DIVERTICULA of the distal esophagus, epiphrenic diverticula, are relatively rare, but are more frequently of clinical importance than the common traction diverticula of the mid-esophagus. Epiphrenic diverticula have a tendency to enlarge and are frequently associated with neuromuscular dysfunction of the esophagus.

We are presenting our experience with 80 patients with epiphrenic diverticula who were seen at the Columbia-Presbyterian Medical Center between the years of 1954 and 1972. Fifty-one of these were studied by cine-roentgenographic techniques. The pathologic findings on 11 excised diverticula will also be discussed.

DEFINITION

The epiphrenic segment of the thoracic esophagus has been defined as the distal 4 cm.,[4] the distal 10 cm.,[3,11,14] or the distal one-third. Habein *et al.*[11] were even less specific by referring to them as lower esophageal diverticula.

Barrett[2] in 1933 spoke of 3 types of esophageal diverticula; traction, pulsion and traction-pulsion. He stated that traction diverticula occurred in the mid-esophagus, and were usually secondary to an inflammatory process in the carinal lymph nodes. These are called true diverticula because their walls consist of all 4 layers of the normal gastrointestinal tract.

Pulsion diverticula are thought to be the result of increased intraluminal pressure secondary to neuromuscular dysfunction. At least this seems to be true for the pharyngo-esophageal or Zenker's diverticula and the evidence is also accumulating to support this concept for the pathogenesis of colonic diverticula. Pulsion diverticula are also called false diverticula since they consist only of mucosa and a variable amount of submucosa which has herniated through a hiatus in the muscularis propria.

There have been very few descriptions of the pathology of epiphrenic diverticula. Janes[12] described 4 surgically excised diverticula as pulsion or false diverticula, while Goodman and Parnes[9] stated that epiphrenic diverticula were pulsion in type, but then claimed that the wall contained all layers of esophagus. Putney and Clerf[17] made a similar comment and said that the muscle layer was present, although thin and sparse. Quoting Putney and Clerf's paper, Habein *et al.*[11] also stated that pulsion diverticula were true diverticula.

Some authors have confused the issue even further by speaking of a traction-pulsion diverticulum[2,18] and have claimed that it originally begins as a traction diverticulum, but is subsequently enlarged by pulsion forces. To our knowledge, this sequence of events has never been documented.

Congenital epiphrenic diverticula do exist, but are exceedingly rare. Up to 1962 there were only 2 well documented, histologically proven examples reported. According to Mendl,[16] these represent duplications of the esophagus which have developed a connection with the esophageal lumen and may contain gastric, pancreatic or small intestinal epithelium. There is a normally developed muscularis propria.

CLINICAL MATERIAL

The age of the 80 patients in this series ranged from 18 to 88, with 75 per cent be-

* Presented at the Seventy-third Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 3–6, 1972.
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tween 41 and 70 years. Eight patients had multiple diverticula. The male/female ratio was 2 to 3. Small diverticula measuring 3 cm. or less made up 59 per cent of the group, while 25 per cent were larger than 5 cm. (Table I). This distribution was about the same as for the 149 cases reviewed by Habein *et al.*[11]

Epiphrenic diverticula were more common on the right (Table II); however, it was the large diverticula which accounted for this right-sided predominance since the small diverticula were evenly divided between right and left, while 15 of the 19 large diverticula were on the right. Perhaps the aorta and the heart inhibited enlargement on the left side.

The shape of epiphrenic diverticula was dependent on their size, position and stage of esophageal dynamics. For small diverticula, the shape varied from broad and flat with a poorly defined neck to a rounded sac and well-defined neck, as the sequential cine frames in Figure 1, *A–D* show. The larger diverticula tended to have better defined necks and a portion of the sac was dependent and acted as a passive reservoir. These large diverticula tended to fill either concomitantly or preferentially to the more distal esophagus (Fig. 2, *A* and *B*).

The defect in the esophageal wall at the orifice of the diverticulum was sometimes quite large and ranged in size from ½ to 7 cm. in the superoinferior direction. It tended to be larger with the larger diverticula. Occasionally, one encountered a rather large sac with a very narrow neck; these tended to retain the barium and food for long periods. The portion of the diverticulum below the orifice did not empty until the patient assumed a recumbent position.

The incidence of hiatal hernia and neuromuscular dysfunction is listed in Table III. The majority of patients with small diverticula had either a hiatal hernia or neuromuscular dysfunction. The latter was characterized by diminished peristaltic activity and numerous tertiary waves—a common phenomenon in this age group.

TABLE I

DISTRIBUTION ACCORDING TO SIZE OF DIVERTICULUM OF 88 LOWER ESOPHAGEAL DIVERTICULA OCCURRING IN 80 PATIENTS

Size (cm.)	No. of Diverticula	Per Cent
Less than 1	5	6
1–3	47	53
3–5	14	16
5–7	15	17
Greater than 7	7	8
	88	100

Severe neuromuscular dysfunction occurred in 10 of the 20 patients with large (over 5 cm.) diverticula. Four of these could be classified as typical achalasia. The others were characterized by aperistalsis and significant amounts of residual barium in the esophagus after the completion of deglutition.

All 4 patients with lower esophageal spasm had small diverticula.

Tables IV and V correlate the symptoms with the size of the diverticulum and the presence or absence of associated esophageal disease. The small diverticulum without other demonstrable esophageal abnormalities was usually asymptomatic. However, if associated esophageal disease was present, symptoms were common. It is probable that the latter is responsible for the symptoms and not the diverticulum.

All of the patients with large (over 5 cm.)

TABLE II

DISTRIBUTION OF DIVERTICULA ACCORDING TO DIRECTION OF PROTRUSION FROM ESOPHAGUS

	All	Small*	Large*
Right	45	30	15
Left	35	31	4
Anterior	3	2	1
Posterior	2	2	0
Unknown	3	2	1
	88	67	21

* Small: less than 5 cm. Large: greater than 5 cm.

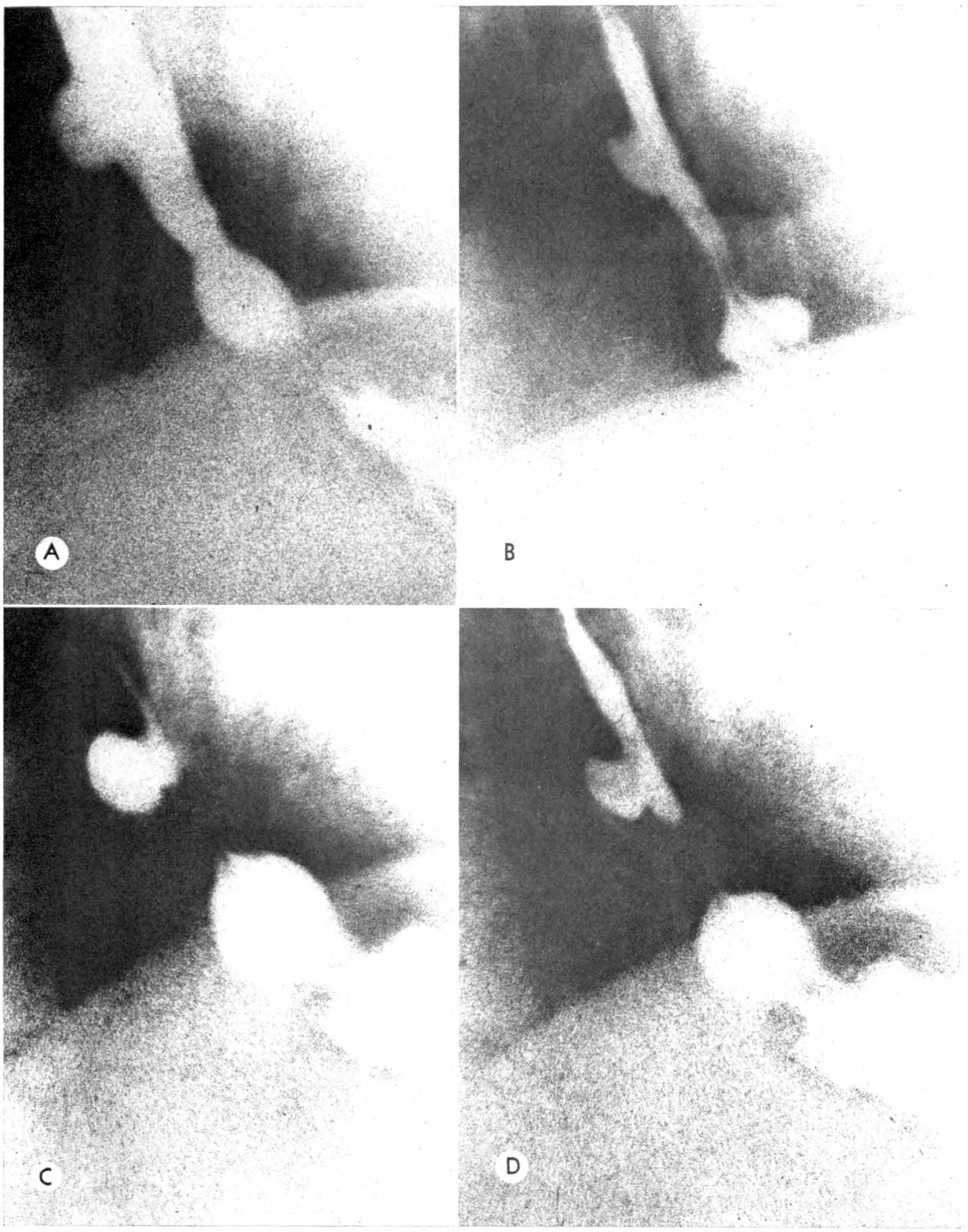


FIG. 1. (*A–D*) The variable shape of an epiphrenic diverticulum. Four cine frames showing different stages of filling and emptying of esophagus and diverticulum. The shape can vary from a broad and flat sac with poorly defined neck (*A* and *B*) to a rounded sac with a well-defined neck (*C*). Note small hiatal hernia and retrograde filling of esophagus during phase of diverticular emptying (*D*).

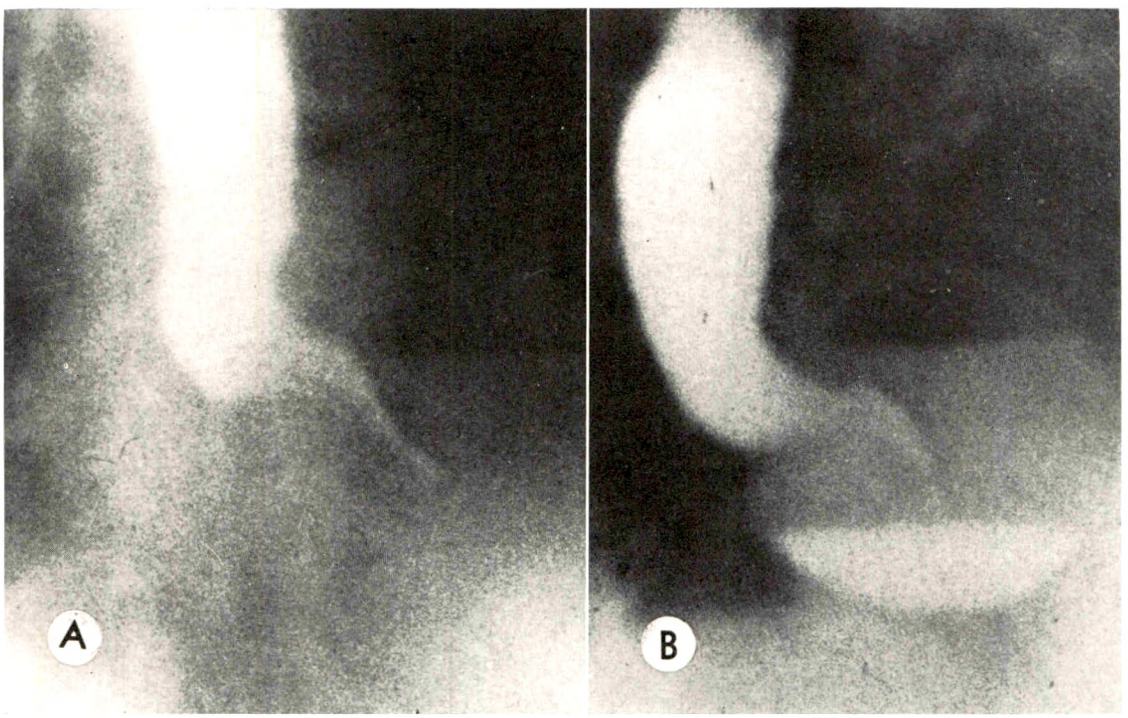


Fig. 2. (*A* and *B*) Preferential filling of diverticulum before filling of distal esophagus. Air-fluid level is in diverticulum (*B*).

diverticula had associated esophageal abnormalities (Table v).

Some patients who had minimal esophageal motor dysfunction or small hiatal hernias without reflux complained of nocturnal regurgitation or regurgitation of undigested food 3 or 4 hours after a meal if they assumed a recumbent position. This was the only symptom which seemed secondary to the diverticulum and not due to the associated disease. Substernal fullness and discomfort on eating might also be related to the distention of the sac. The majority of the complaints, especially those of dysphagia and pain seemed related to the associated disease and not the diverticulum.

Table III

INCIDENCE OF ASSOCIATED ESOPHAGEAL DISEASE IN PATIENTS WITH DIVERTICULA

	Small* Diverticula	Large* Diverticula
Normal examination	19	2
Minor neuromuscular dysfunction	22	7
Hiatal hernia	24	7
Achalasia	3	4
Lower esophageal spasm	4	0
Unclassified severe neuromuscular dysfunction	2	6

* Small: less than 5 cm. (60 patients). Large: greater than 5 cm. (20 patients).

Table IV

INCIDENCE OF SYMPTOMS AND ASSOCIATED DISEASES WITH SMALL DIVERTICULA IN 60 PATIENTS

Symptoms	Associated Disease Absent	Associated Disease Present
Dysphagia	0	9
Regurgitation	2	6
Substernal pain	1	9
Belching, burping, etc.	0	4
Asymptomatic	17	24

TABLE V

INCIDENCE OF SYMPTOMS IN 20 PATIENTS WITH LARGE DIVERTICULA*

Symptoms	No. of Patients
Dysphagia	7
Regurgitation	11
Substernal pain	1
Belching, burping, etc.	3
Asymptomatic	3

* All patients with large diverticula had associated disease.

The vast majority of diverticula when first seen had reached a stable size. Twenty-five cases were re-examined 1 to 12 years after the initial study. In only 4 cases did they increase in size. Two of these occurred in patients with achalasia. During the growth of the diverticula, the achalasia worsened, as manifested by increasing dysphagia, greater dilatation of the esophagus and more esophageal retention of barium (Fig. 3, *A–C*). A third case had severe neuromuscular dysfunction with 2 diverticula, both of which enlarged. The fourth case is perhaps the first recorded example where a normal eophagus was documented prior to the development of a diverticulum (Fig. 4, *A* and *B*). Initially, the esophagus showed borderline distal narrowing with diminished peristalsis and delayed emptying. Dysphagia and regurgitation were present at this time. Four years later, a large diverticulum with a broad neck was found. Again diminished peristalsis with tertiary contractions were noted. The mecholyl test and esophagoscopy were both negative. No symptomatic improvement resulted from bouginage, and a repeat barium swallow study showed no change. These findings support the concept of the acquired nature of epiphrenic diverticula and that their growth and development are related to esophageal motor dysfunction.

PATHOLOGY

Eleven diverticula were resected and the microscopic sections were reviewed. In every case, the wall of the diverticulum lacked a muscularis propria (Fig. 5*B*). A well defined mucosal layer with squamous

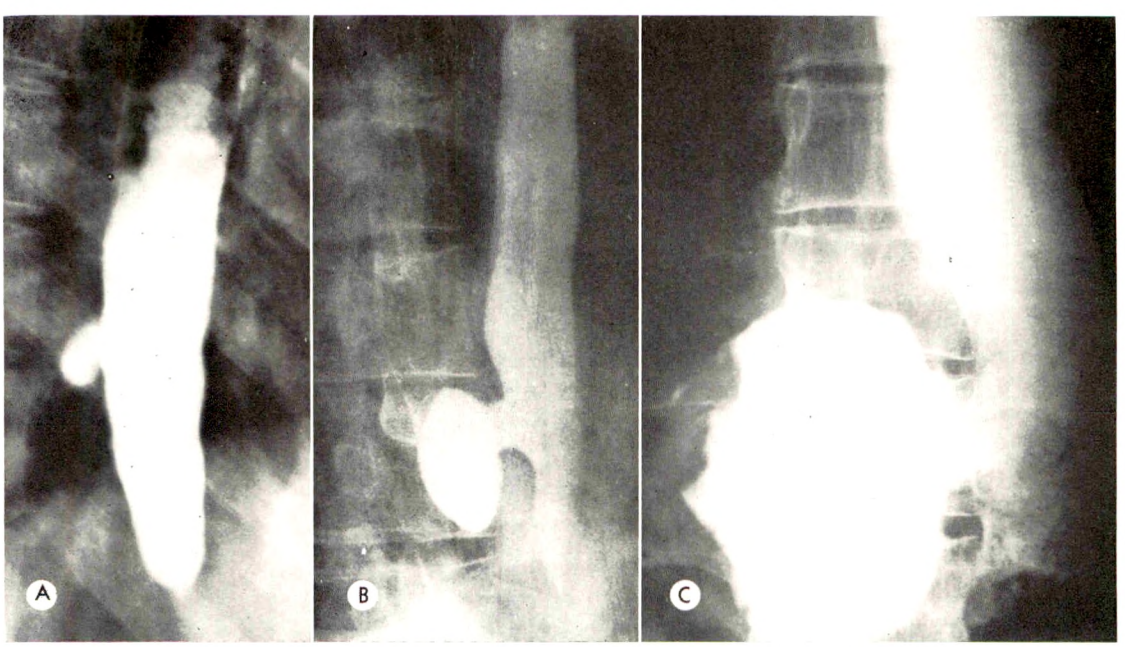


FIG. 3. (*A–C*) An enlarging diverticulum associated with advancing achalasia. (*A*) Small diverticulum and normal caliber esophagus. (*B*) Four years later, diverticulum has enlarged. (*C*) Six years later, markedly enlarged diverticulum containing food and a dilated esophagus are seen.

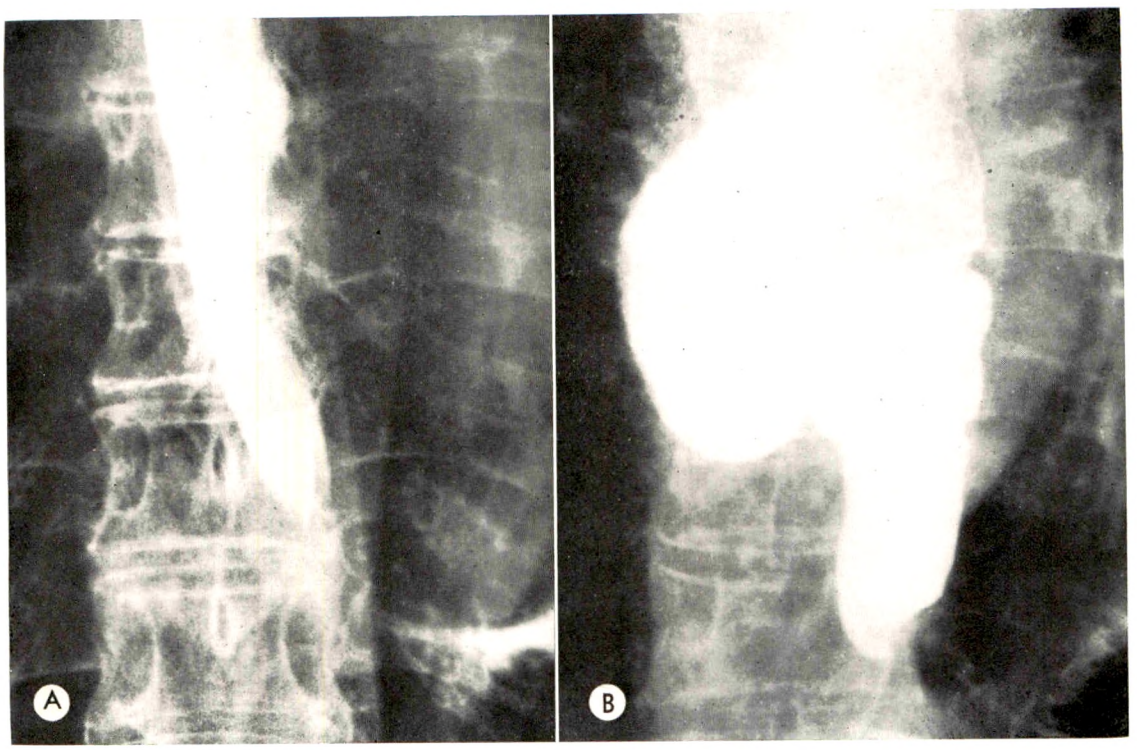


Fig. 4. (*A*) Esophagus without diverticulum. (*B*) Four years later, a large diverticulum has appeared in it.

epithelium, lamina propria and muscularis mucosae was seen. The squamous epithelium showed hyperplasia of mild to moderate degree and some inflammation. No areas of ulceration were seen microscopically; however, 1 case showed ulceration on the gross specimen. Three specimens showed some hyperkeratosis and parakeratosis. One specimen showed some cellular atypia, but no malignant changes were identified. The muscularis mucosae was focally hyperplastic ranging from 2 to 8 times its normal thickness and showed varying degrees of fibrosis. Beneath the muscularis mucosae there was a band of connective tissue representing the submucosal and adventitial layers. There were scattered areas of mild to moderate inflammation in the connective tissue layer. The total wall thickness measured from 0.2 to 0.8 cm.

Thus, all of the resected diverticula in this series resulted from herniation of the mucosal layer through a defect in the muscularis propria. They are, therefore, "false" diverticula and their mechanism of formation is felt to be secondary to pulsion forces.

DISCUSSION

Once a diverticulum is identified, the roentgenographic examination should be directed towards two primary goals. The first is to clearly show the diverticular sac, neck and esophageal lumen simultaneously and maximally distended. This is usually best done with the patient recumbent, not erect, and with the proper degree of obliquity. The length of the defect in the esophageal wall and the direction of protrusion of the neck should be determined because these are important landmarks for surgical excision. The second goal is to carefully examine the esophagus for associated neuromuscular dysfunction and hiatus hernia. Peristaltic stripping waves are important to identify, for these are absent in achalasia and absent in the lower esophagus in lower

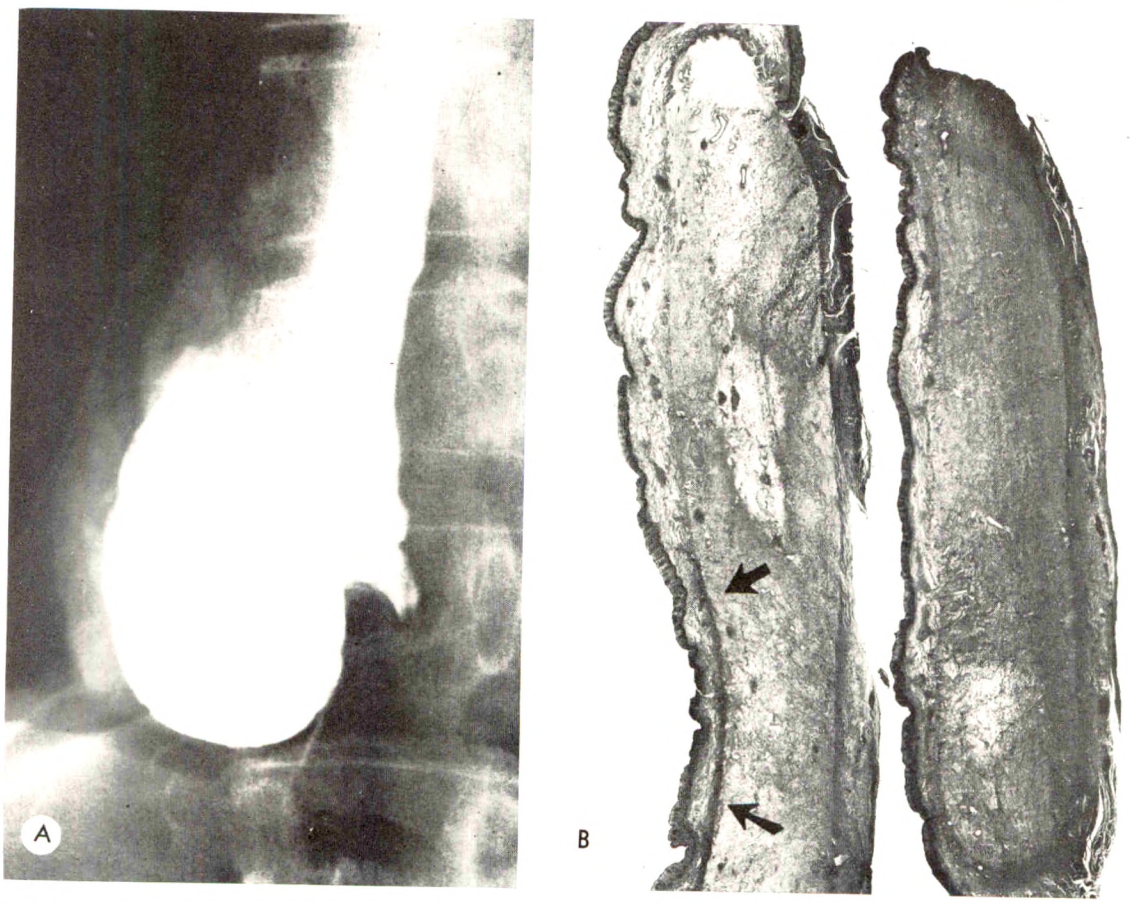


FIG. 5. (*A*) Roentgenogram of diverticulum with thick wall. (*B*) Sections through wall of same diverticulum. Arrows point to prominent layer of focally hyperplastic muscularis mucosae. The epithelium is intact. Below the muscularis mucosae is a thick band of connective tissue without any muscularis propria.

esophageal spasm. These are best seen by following the progression of the bolus following a single swallow of barium. Repeated swallows will inhibit primary peristalsis. The esophagus should be studied in all stages of filling and emptying. Because of the association of neuromuscular dysfunction, the importance of performing an esophagomyotomy on all patients at the time of diverticulectomy has been emphasized.[1,7] The need for this in the presence of an otherwise normal esophagus is open to question. However, it is extremely important for the radiologist to determine whether neuromuscular dysfunction is present.

All 11 resected specimens were felt to represent pulsion diverticula which varied in size from small to large. The shape of small diverticula changed considerably, depending on the stage of esophageal dynamics. Habein *et al.*[11] described a typical traction diverticulum of the lower esophagus as having a broad neck and sac which was not dependent. They grouped their 149 cases into 97 patients with "pulsion" diverticula and 52 patients with "traction" diverticula based purely on morphologic roentgenographic appearance. None of their cases of traction diverticula was studied histologically nor were we able to find any histologically proven cases of traction diverticula of the lower esophagus in the English language literature.

One case, of particular interest, had had a large pulsion diverticulum resected. The diverticulum recurred (Fig. 6) and by its morphology should have been a "traction" diverticulum. However, this recurrent diverticulum was resected and showed the typical microscopic findings of a pulsion diverticulum. An esophagomyotomy was performed at the second operation and no recurrence was noted on follow-up examination 8 months later.

Thus, morphology alone cannot be used to distinguish pulsion from traction diverticula. In addition, pulsion diverticula have been reported in the mid-esophagus,[15] indicating that position alone is not an adequate criterion for distinguishing traction from pulsion diverticula. In our experience, however, epiphrenic diverticula would appear to be pulsion diverticula in almost all instances except the very rare congenital diverticulum.

It seems probable that neuromuscular dysfunction is an important factor in enlarging the diverticula. This was personally observed in 2 cases of achalasia mentioned earlier. Under these conditions, with a functional obstruction distal to the diverticulum, a swallowed bolus entered the low pressure diverticular sac with rapidity as a result of gravity and failure of distal esophageal relaxation. This is felt to be a major factor in causing the growth of these diverticula. Another important factor in achalasia may be the hydrostatic pressure from the column of fluid and food sac which is present for long periods in the dilated esophagus of these patients.

These factors were not present in lower esophageal spasm and it is interesting to note that our 4 cases of this disease all had small diverticula (Table III). The distal esophagus in these patients was not dilated, but was in a state of spasm. As we can see in Figure 7, *A* and *B*, the diverticulum occurred within the area of spasm. Despite elevated pressures, the mechanical force of the swallowed bolus entering the diverticulum was not present, as the region of spasm served to partially obstruct flow above the level of the diverticulum. Thus, these diverticula were not prone to enlargement.



Fig. 6. Recurrent diverticulum. Cine frame showing flat, broad-based diverticulum which recurred after simple resection. Reoperation and removal showed a recurrent false pulsion diverticulum.

The factors responsible for association of small and large diverticula with hiatal hernia are obscure.

It has been stated in the literature that the distended dependent sac of a pulsion diverticulum obstructs the distal esophagus causing dysphagia.[10,11] Putney and Clerf[17] stated that the main reason for removing these diverticula was to relieve the symptoms of esophageal obstruction. Review of our material failed to confirm these statements. There may be slight displacement of the esophagus around the sac,

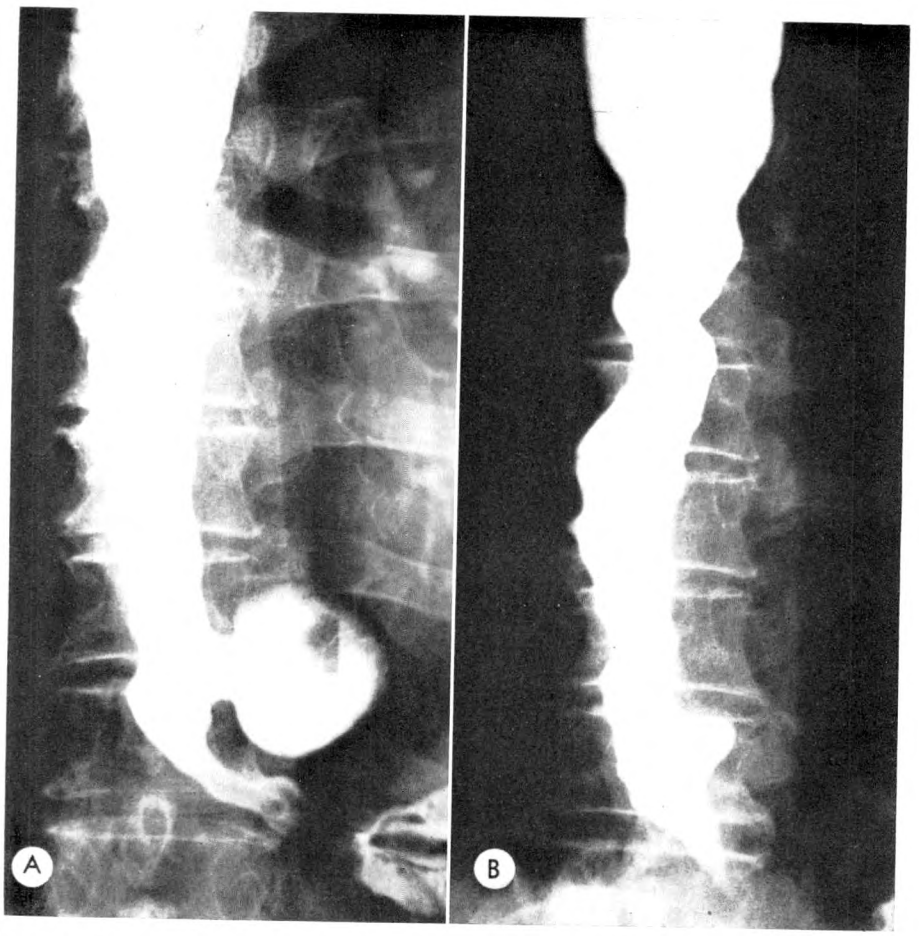


FIG. 7. (*A* and *B*) Diverticulum associated with lower esophageal spasm. (*A*) A small diverticulum is seen within the area of spasm in the distal esophagus. The proximal esophagus is dilated secondary to the functional obstruction in the distal esophagus. (*B*) No change in the associated disease after diverticulectomy. Symptoms also unchanged.

but its lumen was never significantly narrowed or obstructed by the sac. The swallowed bolus distended the esophagus at and below the level of the diverticulum. The lack of dysphagia in patients who had no associated esophageal disease (Tables IV and V) supports this concept.

Symptoms referable to the esophagus in patients with esophageal diverticula should arouse suspicion of associated esophageal disease. With diverticulectomy alone, there has been a significant rate of recurrence and failure to relieve symptoms.[1,10] Of the 4 patients in our series who had diverticulectomy alone, 1 died and 3 had persistent symptoms. This again suggests that the symptoms are usually not secondary to the diverticulum.

There have been 9 cases of carcinoma originating in an epiphrenic diverticulum reported in the world radiologic literature.[8] It is doubtful whether this indicates a predilection for neoplasia, although it is conceivable that the stasis and chronic infection that occur in the larger sacs might predispose to the development of a malignancy. Ulceration and perforation have also been described.

Differential diagnosis is not difficult. Extreme esophageal redundancy such a

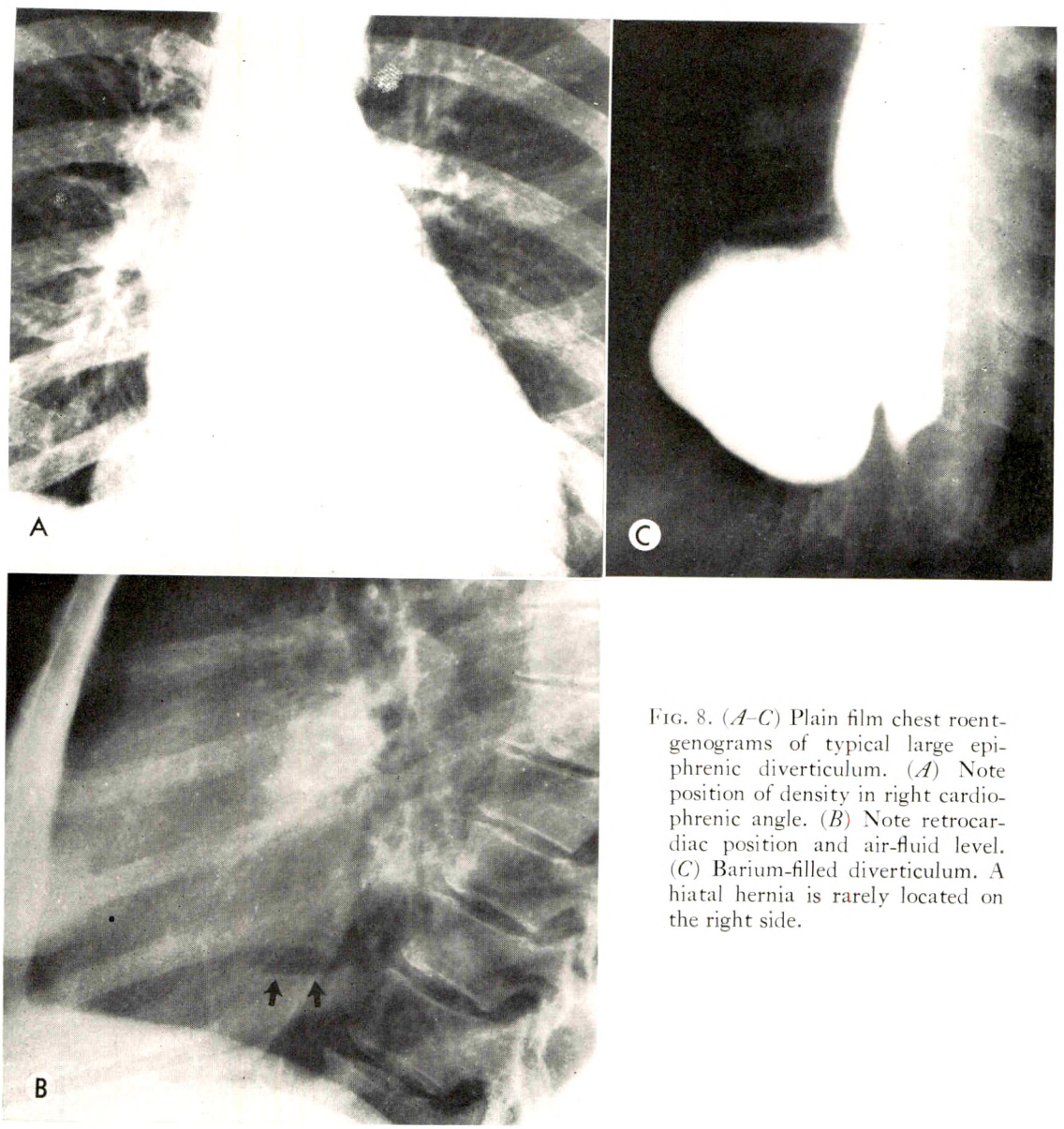


Fig. 8. (*A–C*) Plain film chest roentgenograms of typical large epiphrenic diverticulum. (*A*) Note position of density in right cardiophrenic angle. (*B*) Note retrocardiac position and air-fluid level. (*C*) Barium-filled diverticulum. A hiatal hernia is rarely located on the right side.

occurs in advanced achalasia or a hiatus hernia might conceivably simulate a large diverticulum. The pseudodiverticula associated with lower esophageal spasm are transient phenomena. The diagnosis can occasionally be inferred from a chest roentgenogram on the basis of a rounded mass in the right cardiophrenic angle containing an air-fluid level (Fig. 8, *A–C*). A hiatal hernia is only rarely found on the right side.

SUMMARY

The clinically important diverticula of the thoracic esophagus occur in the epiphrenic region. The vast majority are pulsion diverticula secondary to herniation of the mucosa through the muscularis propria.

Large diverticula may be symptomatic and are associated with hiatal hernia or neuromuscular dysfunction of the esophagus. Small diverticula are asymptomatic

except when associated esophageal disease is present.

Careful evaluation of the esophagus for the presence of associated disease is essential.

Surgery should be directed towards the associated disease as well as the removal of the diverticulum.

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ANGIOGRAPHIC DEMONSTRATION OF BLEEDING GASTRIC LEIOMYOMAS*

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LEIOMYOMAS are the most common benign gastric tumors other than adenomatous polyps. Approximately one-half of the patients with gastric leiomyomas present because of ulceration of the overlying mucosa. In the largest series of gastric leiomyomas[3] 42 per cent of the patients had manifest massive upper gastrointestinal bleeding.

We have recently identified the bleeding sites in two patients with ulcerated gastric leiomyomas. We believe that these are the first recorded cases in which active bleeding from these tumors has been demonstrated angiographically.

REPORT OF CASES

CASE I. A 30 year old man presented to the emergency room of Montefiore Hospital and Medical Center with massive vomiting of bright red blood following a weekend of moderate alcohol ingestion. There was no past history of abdominal pain or dyspepsia. His initial hematocrit was 37 per cent. Mallory-Weiss syndrome, gastric ulcer, and erosive gastritis were the diagnoses considered.

Endoscopy revealed extensive bleeding in the area of the gastric fundus, but no mass was visualized.

Endoscopy was followed by celiac angiography which showed extravasation of contrast material from branches of the left gastric artery in the gastric fundus. A tumor blush surrounded the extravasated contrast material (Fig. 1*A*). Massive hematemesis occurred several times during the angiographic procedure.

The patient's hematocrit fell to 31 per cent despite 8 units of packed cells and 2 units of fresh, frozen plasma.

At operation, 8 hours after admission, a leiomyoma was identified which arose from the anterior wall of the fundus of the stomach. A large ulceration was noted at the apex of the leiomyoma (Fig. 1*B*). A wedge resection of the tumor was performed.

The patient was discharged 10 days later in good condition.

CASE II. A 67 year old housewife presented with hematemesis. She had no prior history of other symptoms. Her blood pressure was 130/50 mm. Hg, with orthostatic hypotension. Her hematocrit was 25 per cent. Physical and laboratory data were otherwise negative.

Celiac angiography showed a well circumscribed tumor blush appearing in the arterial phase (Fig. 2*A*). The late arterial phase showed a collection of extravasated contrast material within the tumor blush (Fig. 2*B*).

The patient was transfused with 5 units of blood over 24 hours, but her hematocrit remained at 26 per cent.

Surgery revealed an intramural leiomyoma in the fundus of the stomach with central ulceration. A partial gastrectomy was performed.

The patient recovered uneventfully.

DISCUSSION

Because intramural gastric leiomyomas frequently present with massive upper gastrointestinal bleeding, they are often confused clinically with other causes of hematemesis such as Mallory-Weiss syndrome, erosive gastritis, and benign gastric ulcer. Angiographic evaluation is often requested despite the fact that most intramural gastric tumors can be identified in upper gastrointestinal series.

Most gastric leiomyomas will show a sharply circumscribed area of hypervascularity during the capillary phase of celiac angiography. Some caution must be exercised in interpretation, however, since a vascular blush is seen normally in the gastric fundus during angiography. Super-

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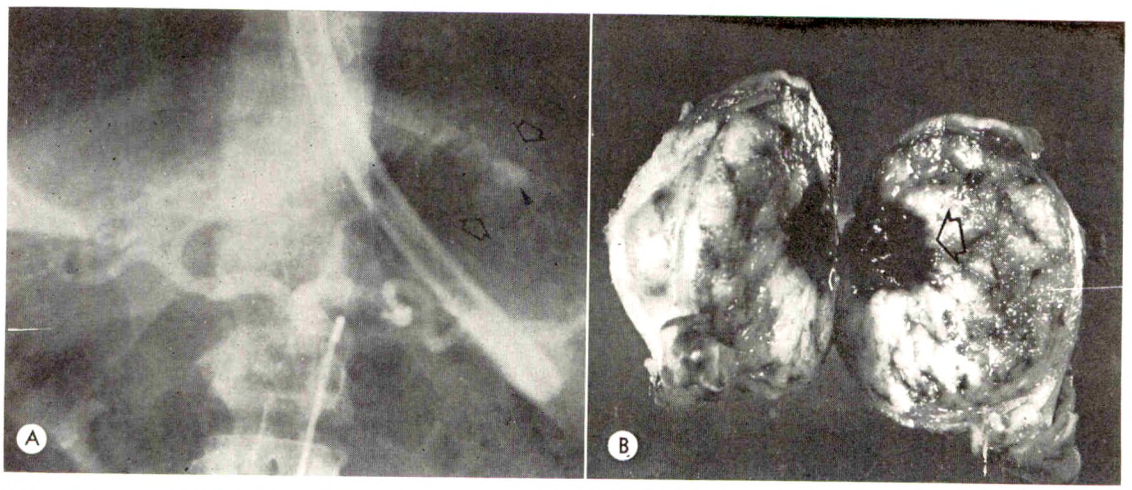


FIG. 1. Case I. (*A*) Celiac angiogram; late arterial phase. Tumor blush (open arrows) in the gastric fundus. Central extravasation of contrast material (arrowhead). (*B*) Surgical specimen. Submucosal leiomyoma with large central ulceration (open arrow).

imposition of vascular blushes in the tail of the pancreas, the spleen, or in accessory splenic tissue might also cause confusion. Two of 5 patients with gastric leiomyomas studied by Reuter *et al.*[5] had no angiographic abnormalities. To our knowledge, all other reports of angiographically demonstrated leiomyomas showed a well circumscribed area of hypervascularity.[1,5,6]

Although they are similar histologically, gastric leiomyomas seem in general to be less vascular angiographically than leiomyomas in the small intestine.[4] This might merely be a technical factor related to the small amount of contrast material delivered into the left gastric artery on celiac artery injection. The tumor blush might also be more difficult to detect on angiograms because of superimposition of normally hypervascular structures in the left upper quadrant of the abdomen.

The presence of large supplying arteries and draining veins with arterial-venous shunting is evidently not a constant finding in gastric leiomyomas as opposed to small bowel leiomyomas. These findings were not present in our cases.

The mechanism of ulceration in intra-

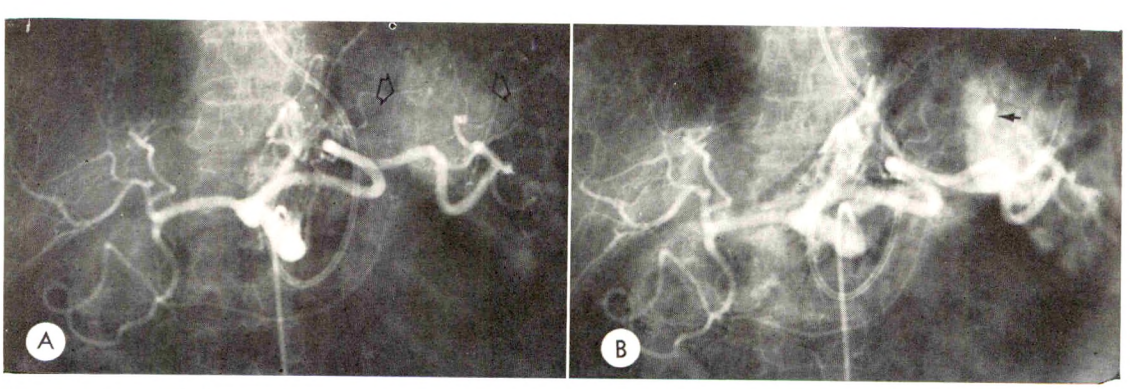


FIG. 2. Case II. Celiac angiogram. (*A*) Arterial phase. Well circumscribed tumor blush, region of gastric fundus (open arrows). (*B*) Late arterial phase. Extravasated contrast material (arrow) from bleeding ulcer within the tumor mass.

mural gastric tumors has been described by Siegelman *et al.*[7] The lesions by virtue of their submucosal growth stretch the overlying gastric mucosa with resultant ulceration.

The present report is the first demonstration, to our knowledge, of intraluminal extravasation of contrast material from a gastrointestinal leiomyoma. The association of intraluminal leakage of contrast material with a sharply demarcated tumor blush should suggest this diagnosis.

SUMMARY

Two patients with intramural ulcerated leiomyomas in the fundus of the stomach who presented with massive upper gastrointestinal bleeding were studied by celiac angiography.

The angiograms demonstrated both intraluminal extravasation of contrast material from the ulcer and a tumor blush surrounding the ulcer. This combination of ulcer and tumor stain suggests the diagnosis of bleeding intramural leiomyoma. However, one must exercise caution in view of the normal structures in the left upper quadrant which can produce a tumor blush.

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PHYTOBEZOAR FORMATION AND FOOD BOLI FOLLOWING GASTRIC SURGERY*

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"Once again, it may be said that food which enters as a friend may become an enemy in our midst."

R. J. Williams, 1957[32]

A BEZOAR is defined as a calculus or concretion found in the stomach and intestines. They are relatively frequent in ruminants, particularly goats and antelopes. The term "bezoar" is derived from the Arabic or Persian meaning "antidote or antipoison."[10] For centuries they were prized for their supposed medicinal and magical properties. Gastric bezoars are infrequently encountered in the general population. In an exhaustive review of the literature, DeBakey and Ochsner[10] compiled 266 cases in 1938. Surprisingly, there have been 28 cases[6,15,20,23,28,30] reported since 1949 in individuals who have undergone some form of gastric surgery, particularly partial gastrectomy. Likewise, obstruction of the small bowel by food substances must be considered unusual in the general population; Ward-McQuaid[31] reported 174 cases, involving 45 different foodstuffs, collected from the world literature in 1950. In individuals who have undergone gastric surgery, however, there are now reports of more than 130 cases of small bowel obstruction caused by phytobezoar occlusion of the intestinal lumen.

Most cases of postgastrectomy bezoar formation have been reported in the past 20 years. The increased incidence mirrors the increased use of partial gastrectomy in the treatment of intractable ulcer disease.

This report presents our experience with 10 cases of bezoar formation occurring in 10 patients following various forms of gastric resection, gastroenterostomy or pyloroplasty and vagotomy collected in a 6 year period from the greater Houston area.

Human bezoars are commonly divided into 3 types: trichobezoar; phytobezoar; and concretion. A trichobezoar or "hair ball" is comprised of ingested hair and found chiefly in women and children. In view of current hair styles, we await new reports of cases in adult males. Phytobezoars are composed of vegetable matter. The most common variety is formed following the ingestion of unripe persimmons and has been given the term "diospyrobezoar"[10] after the generic name of the wild persimmon tree, Diospyron. Concretions are masses of an inorganic substance. These are found most commonly in painters who drink their materials for their alcoholic content. The gums and resins contained in these materials precipitate within the stomach and form conglomerate masses. Paraffin, tar, shellac, asphalt and certain medicinal salts have been reported to form concretions.[3,24] Surprisingly, bezoars of meat have not been reported either within the stomach or small bowel.

The chief constituent of bezoars found in the postgastrectomy stomach or small bowel is the fibrous, pithy component of fruits and vegetables.[16] These congeal to form masses, entrapping a variable amount of stems, seeds, fruit skin, and fat globules. This conglomerated mass of fiber then becomes coated by gastric mucus secretions.

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These phytobezoars vary in size, color, consistency, and number. They may be large enough to fill and distend the gastric remnant or grape-sized and easily overlooked; they vary from rubbery and firm to mushy and soft in consistency; from greenish to yellow to brown in color; and although usually single, up to 4 or 5 separate masses may form.

Some authors have objected to the use of the term phytobezoar for all of these masses.[10,29,30] It is their belief that a true bezoar must, by definition, be compacted and hard in consistency and show some effects of digestion. Certainly this strict usage of the term would exclude many masses of fiber found in the postgastrectomy patient. They further state that the designation "food bolus" would be more exact. We consider both terms valid. It is quite likely that the difference between the 2 is only a matter of time; the longer they reside within the stomach, the more compacted they become. Their etiology and basic content are the same. We shall utilize the term phytobezoar for all.

In postgastrectomy patients over 80 per cent of phytobezoars[4–6,17] have consisted of the retained, fibrous pith of oranges. This contrasts with the predominance of diospyrobezoars, or persimmon bezoars, in the nongastrectomized individuals. Persimmon bezoar formation has geen reported following gastric surgery,[12,14] however. Sauerkraut, grapefruit, Brussel sprouts, coconut, apples, figs, dried fruits, potato skins, artichoke and dandelion greens have been implicated.[6] A concretion of antiacids has been reported.[6]

Walk[30] described the first case of phytobezoar within a gastric remnant in 1949. The largest series, 5 cases, was reported by Szemes and Amberg.[28] Twenty-eight cases have appeared in the literature prior to this report, 13 following Billroth I and 11 Billroth II procedures. Moseley[15] reported the only previous case following vagotomy and pyloroplasty. In three,[18,23] phytobezoar formation followed vagotomy in individuals who had previously undergone gastric resections. In the remaining three[4,7] the nature of the surgery could not be determined.

Once formed within the stomach a bezoar may pass into and obstruct the small bowel. The nondigested fibrous pith of recently ingested food, a food bolus, may do likewise. The usual site of obstruction[4] is in the terminal ileum 30 cm. to 100 cm. proximal to the ileocecal valve, the narrowest portion of the bowel. The point of obstruction in reported series has varied from the ligament of Treitz to the terminal ileum,[6] however. This complication was first reported by Seifert[21] of Germany in 1930. The offending foodstuff was, appropriately enough, sauerkraut. Smithwick and Mallory[26] reported the first case in the English literature in 1942. Norberg[17] reported the largest series, 26 cases. The total number reported to date would exceed 130 cases. This is more likely to occur following a Billroth II than a Billroth I type gastrectomy, presumably because of the larger size of the stoma. Delrio *et al.*[11] reported the first case following gastroenterostomy alone in 1944. There are 3 other similar cases reported. In a special category are 3 cases of fungus ball formation within the gastric remnant reported by Borg *et al.*[2] in 1966. Thirteen[1,24] additional cases have since been reported. We have no experience with this entity.

MATERIAL

The 10 patients, 5 males and 5 females, in the present series ranged in age from 36 to 71 years, averaging 59.8 years. Three patients had previously undergone a Billroth I type gastric resection; 2 each a Billroth II gastric resection, gastroenterostomy, and hiatal hernia repair with vagotomy and pyloroplasty; and 1 a Whipple procedure for a carcinoma of the ampulla of Vater. All except the latter had a bilateral vagotomy. In 8 the bezoar was gastric in location and in 2 a small bowel obstruction was present. Both of the latter followed gastroenterostomies and vagotomies without gastric resection. The time elapsed between the surgical procedure and

discovery of the bezoar ranged from 4.5 months to 10 years, averaging 30 months. The duration of symptoms in the cases of gastric bezoars varied from 10 hours to 4 months averaging 7.5 weeks. One was entirely asymptomatic. In the 2 cases of small bowel obstruction, symptoms were present for 18 hours and 4 days, respectively. The clinical features are summarized in Table 1. The nature of the offending foodstuff is known in only 3 patients in the present series, oranges in 2 and pineapples and cantaloupes in the other. This is believed to reflect our prior lack of knowledge concerning the necessity of dietary restrictions.

SYMPTOMS

The character of the symptoms depends upon the site and size of the bezoar. Once a small bowel obstruction is established, it is manifest by the typical symptoms of nausea, vomiting, and crampy abdominal pain. On occasion the focus of pain migrates across the abdomen suggesting passage of the bolus; otherwise, there is nothing to suggest the specific etiology of the obstruction.

Bezoars within the gastric remnant may be entirely asymptomatic, particularly when small. Usually they are associated with a sensation of fullness, early satiety, eructation, nausea, vomiting, and/or epigastric or left upper quadrant pain. The symptoms result from a reduction in the gastric volume due to encroachment upon the lumen and retardation of gastric emptying by occlusion of the stoma. The latter may assume a ball-valve mechanism. This may be intermittent accounting for the periodicity of symptoms in some cases, or the bezoar may impact, completely obstructing the gastric outlet. Blood loss,[23,25] either acute or chronic, is rarely encountered and, when present concomitantly, other causes should be excluded. The bezoar may erode the gastric mucosa or become adherent to it, resulting in bleeding as it breaks free.[24] One case in the present series was associated with bright-red bleeding per rectum. This proved to be secondary to colonic diverticular disease. The bezoar was asymptomatic and its discovery was incidental. At gastroscopy there was no evidence of mucosal erosion within the gastric pouch.

Table I

PHYTOBEZOAR FORMATION

Case No.	Age and Sex	Surgical Procedure	Time Elapsed Surgery to Bezoar	Treatment	Remarks
Gastric					
I. JC	62 M	Billroth I	10 years	Diet Only	Oranges
II. WR	36 M	Billroth I	6 months	Papain	Oranges
III. GW	71 F	Billroth I	23 months	Papain	—
IV. BB	46 F	Billroth II	6 months	Papain, cellulose and surgery	Enzymes failed
V. AB	69 M	Billroth II	15 months	Endoscope	Pineapples, cantaloupes
VI. IH	67 F	Vagotomy and pyloroplasty	4.5 months	Papase	—
VII. AS	60 F	Vagotomy and pyloroplasty	4.5 months	Endoscope and lavage	—
VIII. VW	61 M	Whipple	32 months	Endoscope and papain	Carcinoma of ampulla of Vater
Small Bowel					
IX. WC	69 M	Gastroenterostomy	5 years	"Milked" into cecum	Distal ileum
X. TM	57 F	Gastroenterostomy	2 years	Enterostomy	Mid jejunum, several masses present

ROENTGENOGRAPHIC FINDINGS

The radiologist is primarily responsible for the diagnosis of bezoars in the gastric remnant. They present as mottled, movable, filling defects (Fig. 1*A*; 2*A*; and 4*A*) within the barium filled gastric pouch.[20,27,29] In the upright position they float, in an iceberg-like fashion, in the barium column with a small, arcuate edge of the mass protruding above the level of the barium (Fig. 1*B*). If sufficiently large, the barium column may split as it enters the stomach, flowing over the surface of the bezoar. The filling defect is usually sponge-like in character as the barium becomes enmeshed in or adherent to the surface of the bezoar (Fig. 4*B*; and 5*A*). We have found it helpful to have the patient drink 6 to 8 ounces of water or, better yet, a carbonated beverage following the barium suspension. The bezoar then stands out in excellent contrast furnished by the enmeshed and adherent barium against the water and air density of the gastric content (Fig. 5*B*). On delayed roentgenograms the bezoar may be identified by the entrapped barium. They may vary slightly in shape during the examination due to their compressibility and, in part, due to their movement and presentation of varying profiles (Fig. 4, *B* and *C*). More than one bezoar may be present,[28] rarely more than 3. Since the bezoar lies free within the lumen, barium will cover all margins of the filling defect (Fig. 1*A*; 2*A*; and 4*A*). On occasion it will be partially adherent to the gastric wall. This results in a restriction of motion and a contour defect of the gastric wall at the adherent margin of the mass.

The roentgenographic findings in the presence of a small bowel obstruction from a phytobezoar are nonspecific (Fig. 6, *A* and *B*). One one occasion[13] the proximal margin of the occluding bezoar was outlined by a column of air within the bowel. Small bubbles of air may become entrapped within the bezoar allowing its identification, usually retrospectively, within the bowel (Fig. 6*C*).

When first encountered the bezoar might be readily dismissed as retained food particles;[3,28] this, despite repeated denials by the patient of any recent food ingestion. Although rather amorphous in appearance,

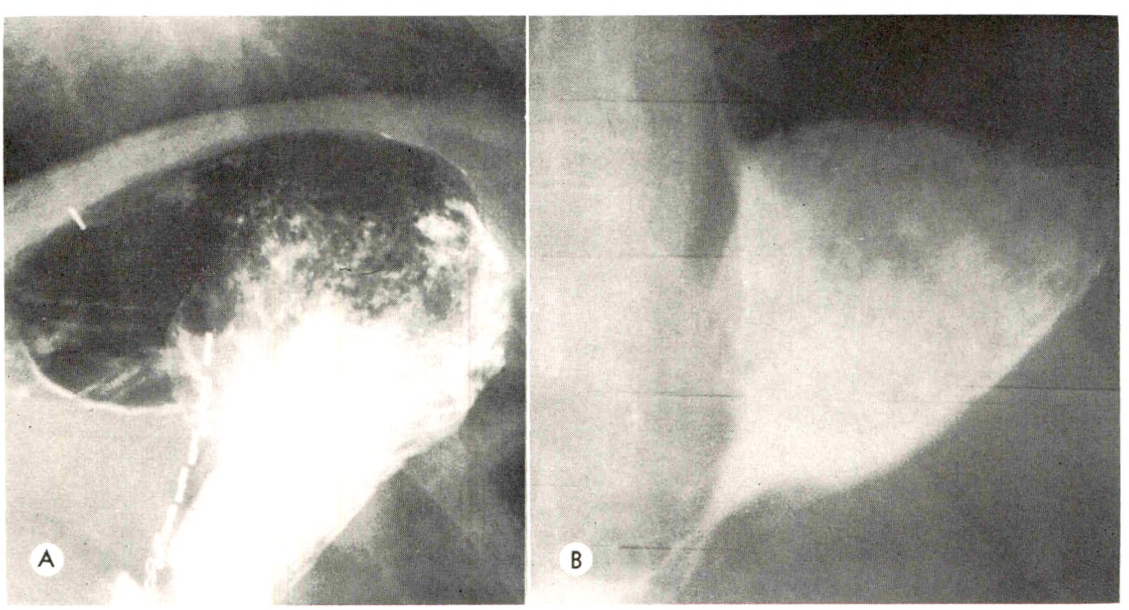


Fig. 1. (*A*) Case 1. Asymptomatic 62 year old male 10 years following Billroth I procedure. Note mottled appearance of bezoar. (*B*) Case 11. A 36 year old male 6 months following Billroth I procedure with 12 hour history of right upper quadrant pain. Upright roentgenogram demonstrates arcuate edge of bezoar protruding above the barium.

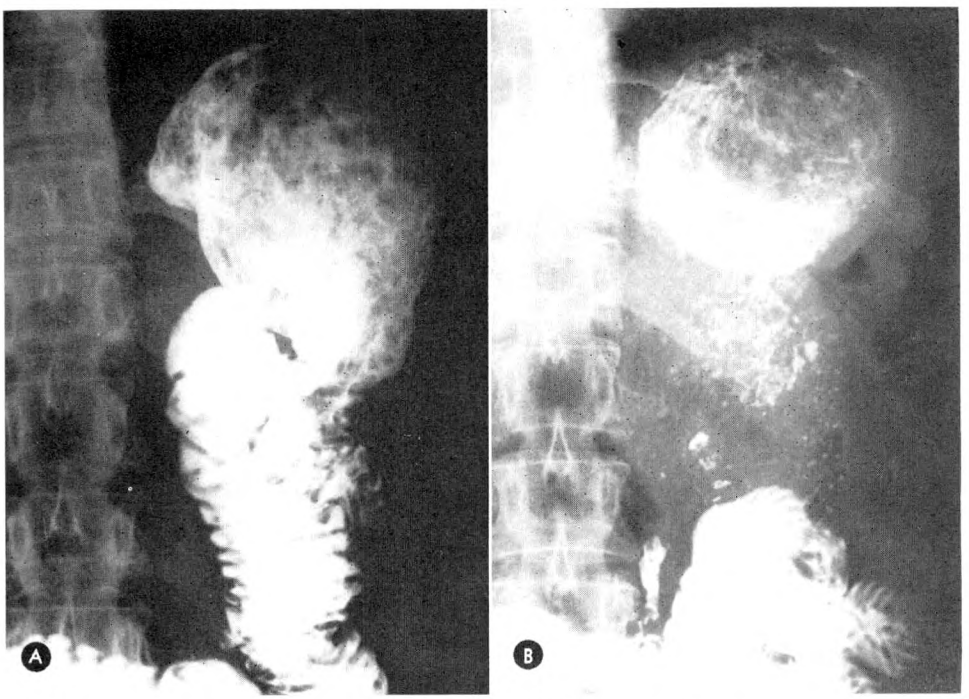


FIG. 2. (*A*) Case IV. A 46 year old female with a 4 month history of intermittent nausea and vomiting 6 months following Billroth II procedure. Note mottled appearance of large bezoar filling gastric remnant. (*B*) A 30 minute delayed roentgenogram demonstrating bezoar by adherent, retained barium.

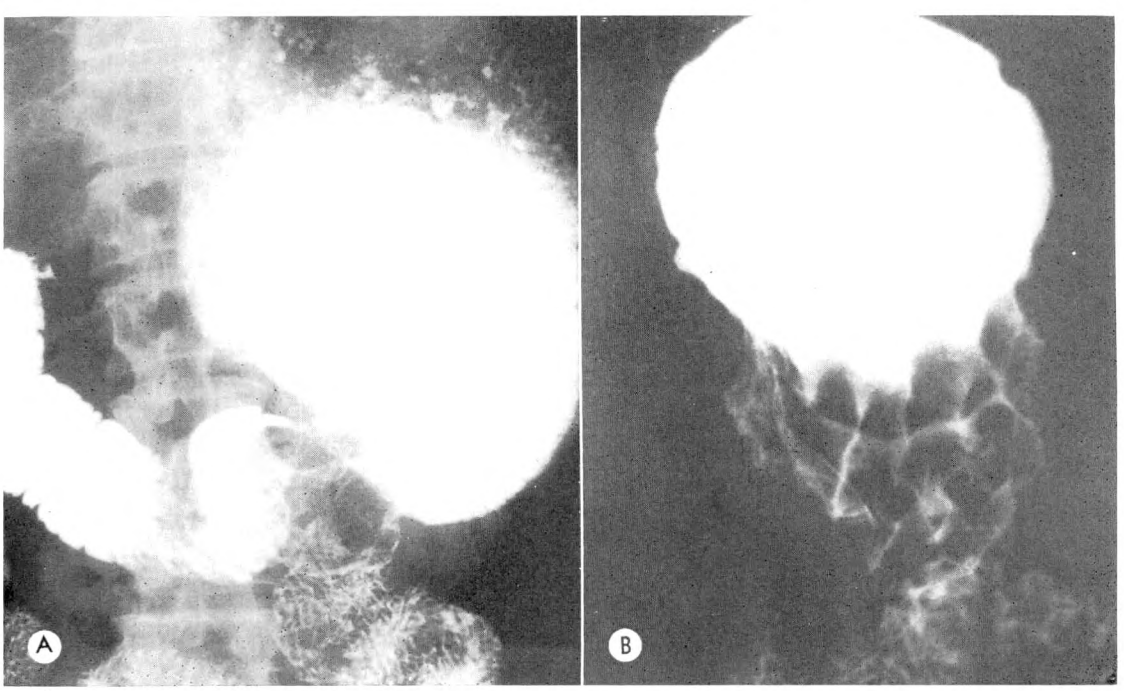


FIG. 3. (*A*) Case V. A 69 year old male with a 3 week history of left subcostal sensation of fullness and early satiety 15 months following a Billroth II procedure. The bezoar has become impacted in the stoma creating a gastric outlet obstruction. (*B*) The bezoar was disrupted and dislodged utilizing the biopsy instrument of a gastroscope. This follow-up examination demonstrates the absence of the bezoar, the adequacy of the stoma and residual mucosal edema. (Courtesy of Dr. Herbert Loyd.)

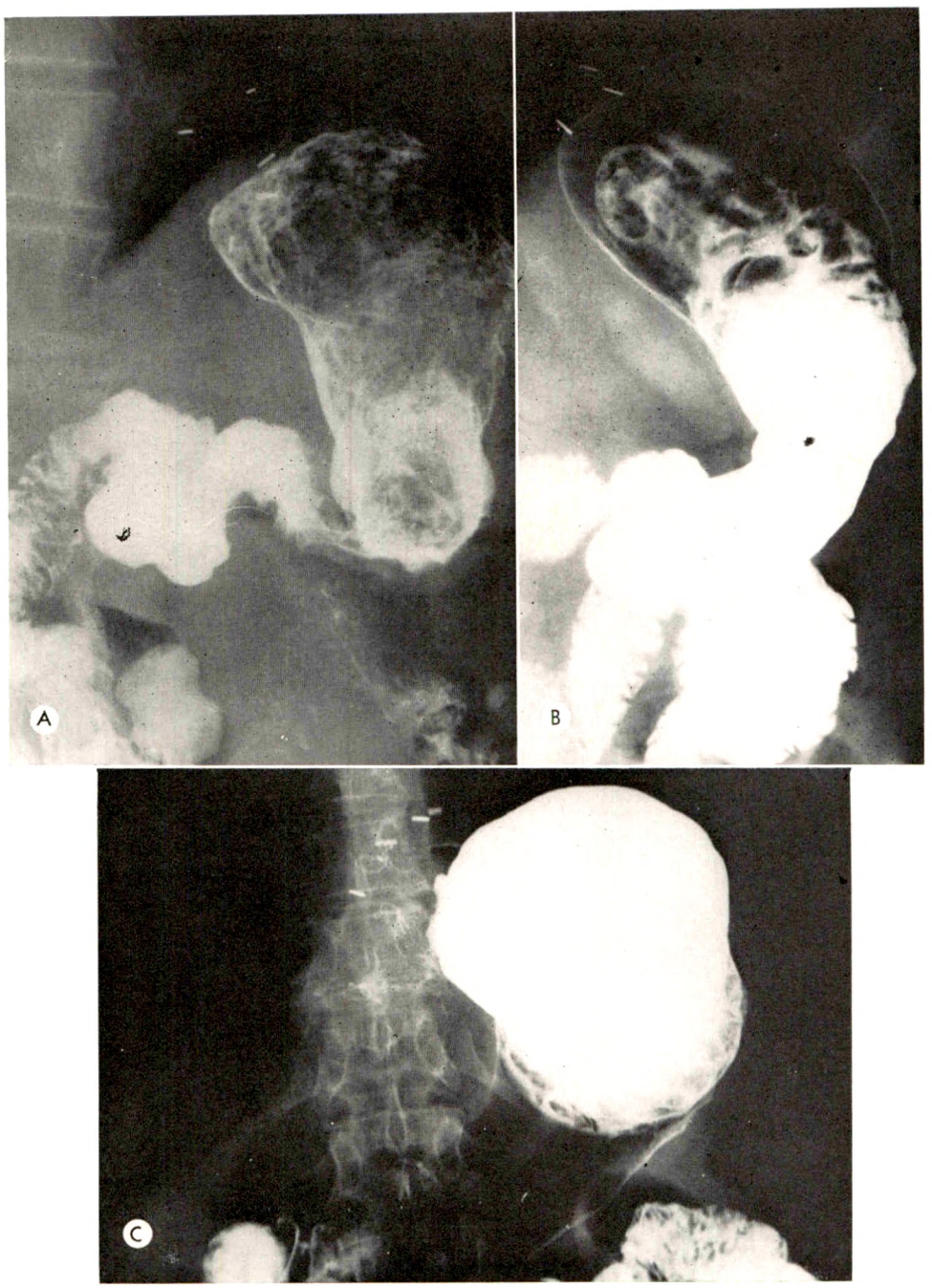


Fig. 4. (*A*) Case vi. A 67 year old female, 4.5 months following vagotomy and pyloroplasty, presented with a 12 hour history of nausea and vomiting. A large, mottled filling defect coated on all surfaces by barium was demonstrated. (*B*) Case vii. A 60 year old female, 4.5 months following vagotomy and pyloroplasty with intermittent nausea and vomiting of 3 months' duration. In the right anterior oblique projection the upper margin of the bezoar stands out well against the fundal air. (*C*) In the anteroposterior projection the inferior margin of the bezoar extends below the barium filled fundus. This case demonstrates the mobility and occasional variability in shape of bezoars. (Courtesy of Dr. Thomas Gillespie.)

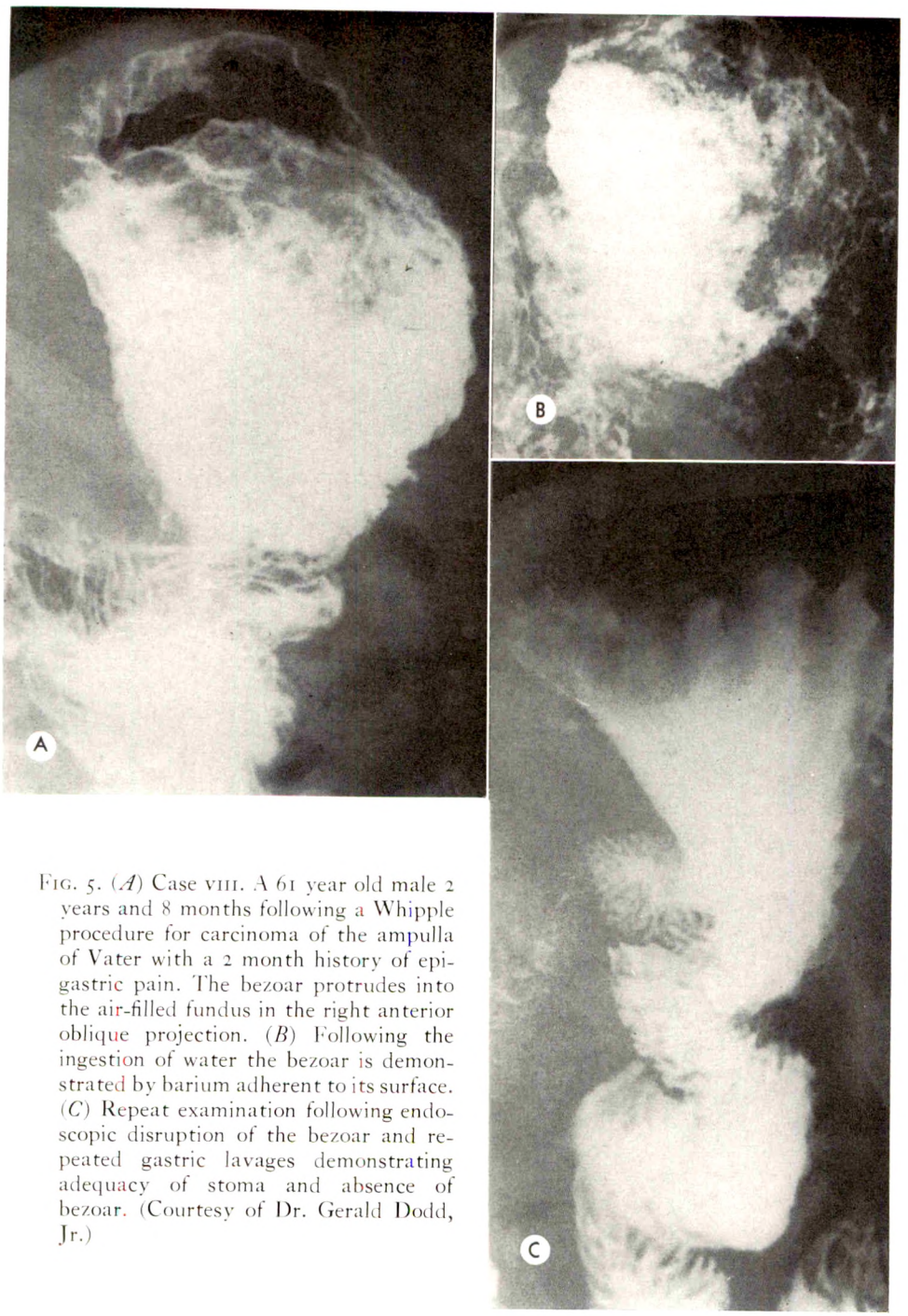


Fig. 5. (*A*) Case VIII. A 61 year old male 2 years and 8 months following a Whipple procedure for carcinoma of the ampulla of Vater with a 2 month history of epigastric pain. The bezoar protrudes into the air-filled fundus in the right anterior oblique projection. (*B*) Following the ingestion of water the bezoar is demonstrated by barium adherent to its surface. (*C*) Repeat examination following endoscopic disruption of the bezoar and repeated gastric lavages demonstrating adequacy of stoma and absence of bezoar. (Courtesy of Dr. Gerald Dodd, Jr.)

the phytobezoar will move as a single unit or ball when palpated. Recently ingested food should divide and mix when palpated at fluoroscopy. Should any doubt exist, the examination should be repeated, preferably following gastric suction and lavage. If the filling defect persists, the diagnosis is confirmed.

The differential diagnosis includes jejunogastric intussusception, recurrent or primary carcinoma of the stomach and sutural defects at the stoma. Jejunogastric

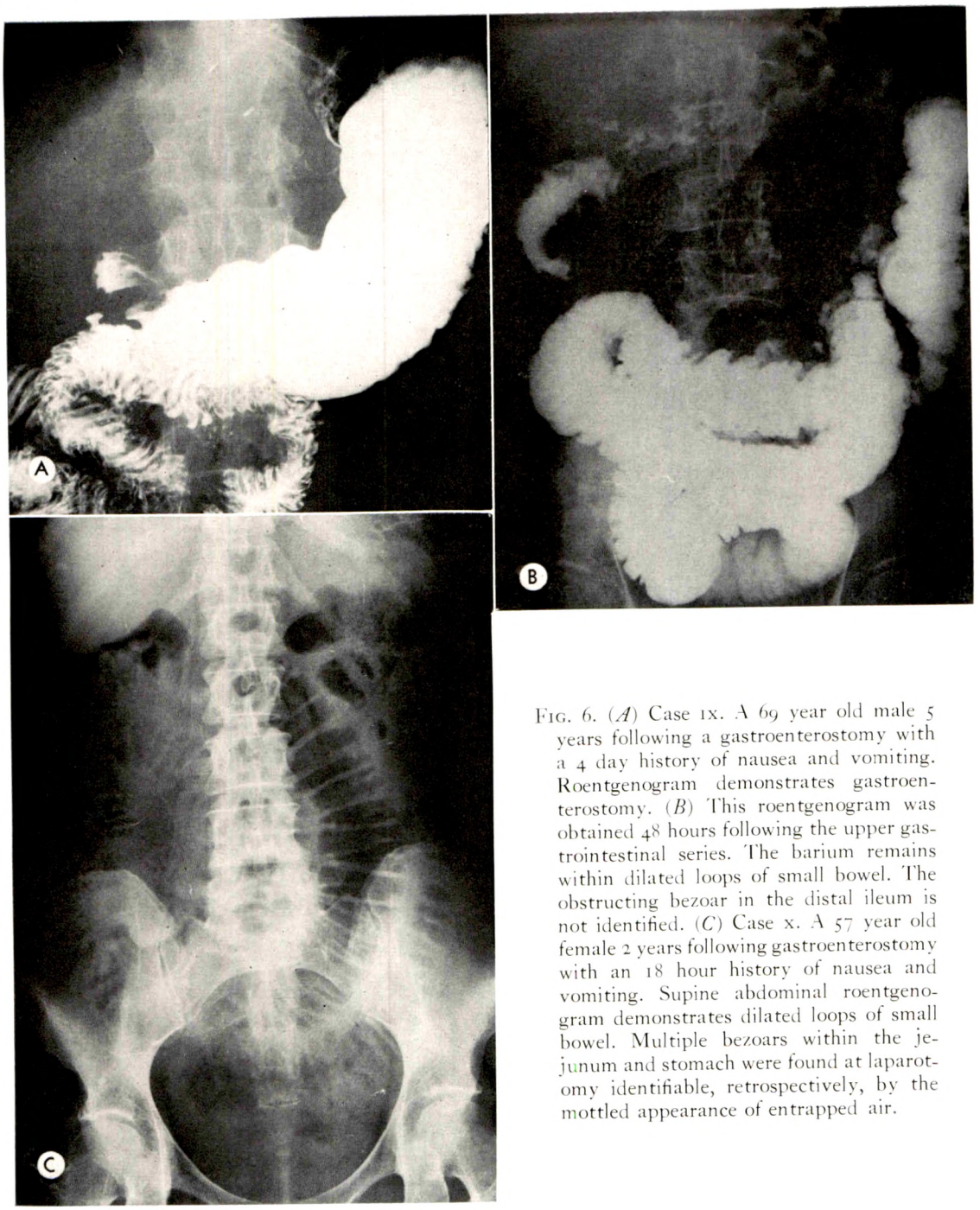


Fig. 6. (*A*) Case IX. A 69 year old male 5 years following a gastroenterostomy with a 4 day history of nausea and vomiting. Roentgenogram demonstrates gastroenterostomy. (*B*) This roentgenogram was obtained 48 hours following the upper gastrointestinal series. The barium remains within dilated loops of small bowel. The obstructing bezoar in the distal ileum is not identified. (*C*) Case X. A 57 year old female 2 years following gastroenterostomy with an 18 hour history of nausea and vomiting. Supine abdominal roentgenogram demonstrates dilated loops of small bowel. Multiple bezoars within the jejunum and stomach were found at laparotomy identifiable, retrospectively, by the mottled appearance of entrapped air.

intussusception is identified by the presence of the coiled-spring appearance of the intussuscepted jejunal loops. Carcinoma is distinguished by the presence of an irregular surface, denoting mucosal destruction, and a broad contour defect at its base indicating that it has arisen from the gastric wall. Sutural defects are smooth, rounded, small, filling defects located at and fixed to the stoma. Fungus balls[2] appear to mimic phytobezoars exactly, requiring gastroscopy and culture for identification.

Gastroscopy is the next logical step in confirming the diagnosis. The phytobezoar

presents as a movable, greenish-yellow mass covered with a gel-like membrane. Confirmatory biopsies may be obtained. Gastroscopy was performed in 6 of the 8 cases of gastric phytobezoars in the present series.

TREATMENT

Once diagnosed there are many approaches to the treatment of phytobezoars, dependent, in part, upon the urgency of the clinical situation. A small bowel obstruction must be relieved by surgery. Upon locating the obstructing bolus it may either be removed *via* enterotomy or "milked" into the cecum manually,[9] thus obviating opening the bowel. The only precaution is to search for and exclude other bezoars or boli located more proximally,[22] as is occasionally the case. It is conceivable that these might cause a second obstruction in the postoperative period.

In one case in the present series the offending bezoar was located in the distal ileum and "milked" into the cecum, and in the other case in the mid-jejunum. The latter was associated with adhesions requiring lysis and the bezoar was removed *via* enterostomy.

The treatment of gastric phytobezoars has been highly variable, as in the present series. Surgery is reserved for only those who fail to respond to more conservative measures. These measures are dietary restrictions, administration of enzymes, and endoscopic disruption and lavage. The most common treatment is the administration of an enzyme, usually raw papain in the form of Adolph's meat tenderizer as first suggested by Dann *et al.*[8] Refined papain, papase,[19] may also be utilized. Its effectiveness is increased by the simultaneous administration of sodium bicarbonate,[21] a mucolytic agent. This dissolves the mucoid capsule of the bezoar and allows more rapid contact between the enzyme and the mass. Papain is a proteolytic enzyme and it is reasonable to question why it should be efficacious in the enzymatic destruction of a mass composed primarily of cellulose. Certainly, experience has proven it effective. It has been suggested that there must be protein bonds between the cellulose fibers of the phytobezoar which are disrupted by papain.[7] Ananase[27] and cellulase[19] have been utilized successfully in the treatment of gastric phytobezoars. The enzymatic action of both is directed against cellulose.

The biopsy instrument of the gastroscope can be utilized to tear apart a bezoar. The resulting small fragments will then either pass into the small bowel or are removed by subsequent repeated lavages. Finally, placement of the patient on a low residue diet has also proven effective in some cases.[20] This is not readily explained; possibly it represents a "spontaneous disappearance" into the small bowel or indicates that the gastric remnant is capable of digesting very small amounts of fiber daily. In any event it would seem that the use of enzymes and sodium bicarbonate would be equally as conservative and more judicious.

Since gastroscopy is commonly employed in the diagnosis, it has become common practice to attempt to disrupt the bezoar at the time of gastroscopy and follow this with repeated gastric lavages and/or enzyme administration. A repeat barium examination is then utilized to confirm the disappearance of the bezoar (Fig. 3*B*; and 5*C*).

In the present series 3 were broken up by the biopsy instrument and were eliminated by subsequent passage. In 1 this was assisted by lavage. Four patients were treated with enzymes, 3 successfully, two utilizing Adolph's meat tenderizer and one papase. In the unsuccessful case the bezoar was quite large, papain was used initially and then cellulase. Sodium bicarbonate was not utilized. This might have led to the failure. The bezoar was subsequently removed by gastrotomy.

DISCUSSION

The performance of a partial gastrectomy with or without an associated vagot-

omy results in profound changes in gastric physiology. Hydrochloric acid secretion is markedly reduced if not eliminated. The pyloric and antral portions of the stomach normally function as a churn, mixing and mechanically disintegrating food particles. The body and fundus participate very minimally in this function. Therefore, this function is eliminated when the pylorus and antrum are excised. Vagotomy results in a further reduction in acid secretion and decreases gastric tone for prolonged periods. Schlang and Mc Henry[22] have demonstrated *in vitro* that both HCl and mixing or agitation of gastric content are essential for the digestion and disintegration of fibrous foods, particularly oranges. These undigested fibers may then congeal to form masses which become coated by the mucus secretions of the stomach forming a phytobezoar.

Proper mastication also plays a role.[4] Many individuals found to have a phytobezoar within either stomach or small bowel have poor dentition or prove to have recently swallowed or bolted whole segments of oranges or large portions of other offending foodstuffs prior to the onset of symptoms. Finally, an injudicious diet or dietary indiscretion creates or compounds the problem.[4] The single or repeated ingestion of excessive amounts of foodstuff is not required. The frequent or even occasional ingestion of fibrous foods, such as a single orange 3 times per week, may suffice. Fibrous foods, particularly citrus foods, should be completely excluded from the diet. Many patients are unaware of this danger and have not been forewarned of the possible complications. Should the radiologist mistake a phytobezoar for recently ingested food, the patient may unknowingly be allowed to continue eating fibrous foods and ultimately suffer the consequences.

In short, phytobezoars are formed in the gastric remnant due to physiologic alterations created by the surgery and compounded by dietary indiscretion and improper mastication of fibrous foodstuffs.

In most cases the size of the stoma is perfectly adequate for the passage of food that is 3.0 cm. to 4.0 cm. in diameter (Fig. 3*B*; and 5*C*). Stenosis or a small stoma is the exception[4,6] and does not appear to be a common factor in phytobezoar formation. Similarly, adhesions[9] play only a small role in the obstruction of the small bowel caused by phytobezoars or food boli. In an exceptional case the lumen will be sufficiently compressed by an adhesion to prevent passage of the bezoar or food bolus (Case x).

Many authors[4,9,23] believe that postgastrectomy patients are particularly prone to transient obstructions of the small bowel during the passage of food boli. These respond to conservative measures; *i.e.*, bed rest, intravenous fluids, and nasogastric suction, and never require surgical intervention. We have encountered 3 such instances in the past 6 months, 1 of which was known to have been preceded by the frequent ingestion of grapefruit. An awareness of this possibility will certainly bring more cases to light and alert the clinician to dietary indiscretions.

SUMMARY

Ten cases of phytobezoar formation following various forms of gastric surgery are reviewed.

The pathogenesis, symptomatology, roentgenographic findings, differential diagnoses, and treatment of this disorder are reviewed.

The necessity for the exclusion of fibrous food, particularly oranges, from the diet of individuals who have undergone gastric resection is emphasized.

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SOME UNUSUAL ROENTGENOLOGIC FINDINGS ASSOCIATED WITH MARKED GASTRIC DILATATION*

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GASTRIC dilatation may occur as an acute or chronic phenomenon, and may or may not be associated with an organic obstruction. The accumulation of gas, secretions and retained food material within the obstructed stomach is generally easily recognized on plain roentgenograms of the abdomen (Fig. 1, *A* and *B*), and the nature of the obstruction can usually be demonstrated by a barium examination after gastric emptying has been obtained by means of repeated or continuous nasogastric suction.

Acute gastric dilatation, in the absence of an actual organic obstruction, is also a well-recognized phenomenon, which is associated with a marked accumulation of gastric and intestinal juices which are not reabsorbed.[2] While the precise etiology and pathogenesis of this condition is still undetermined, most cases either follow surgical operation, trauma, immobilization (especially in a body cast) or are associated with a wide variety of medical disorders such as uncontrolled diabetes, uremic or hepatic coma, cardiovascular and circulatory disorders, pneumonia and poliomyelitis. In recent years this type of acute gastric dilatation has been observed much less frequently, especially in surgical patients, due to improved pre- and postoperative care, the common usage of gastric in-

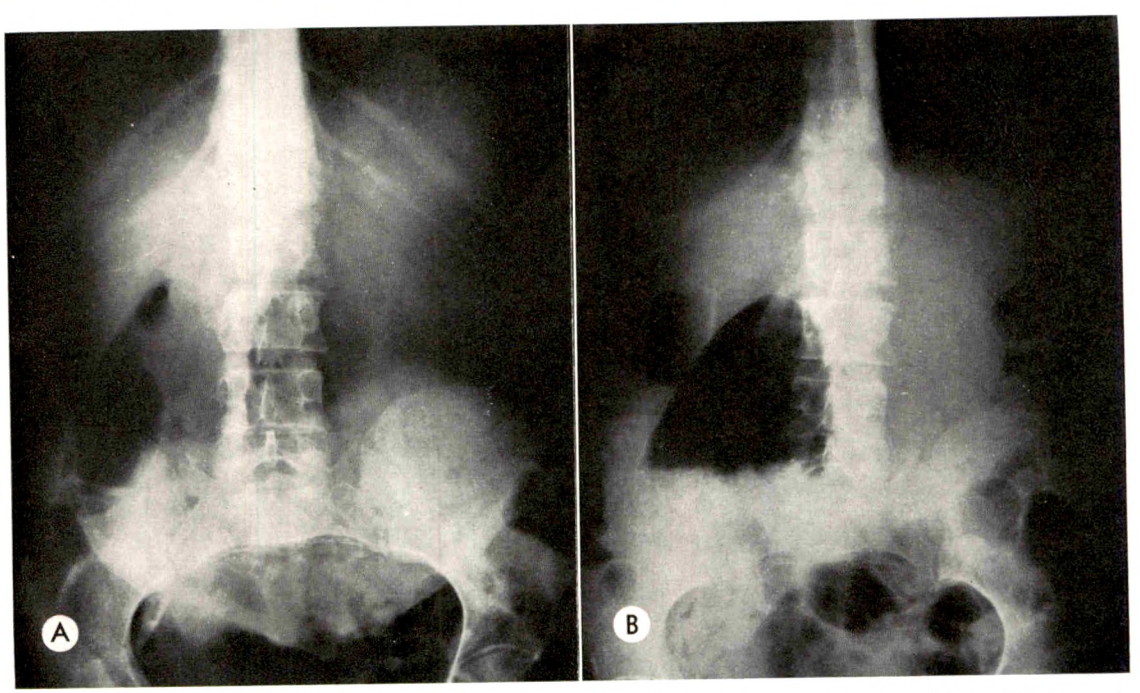


FIG. 1. (*A*) Supine abdominal roentgenogram showing typical findings associated with marked gastric dilatation secondary to obstruction. Note the caudally convex greater curvature crossing the lower abdomen. (*B*) Upright roentgenogram of same patient showing large collection of gas and fluid in the dilated stomach with air-fluid levels in the fundus and antrum.

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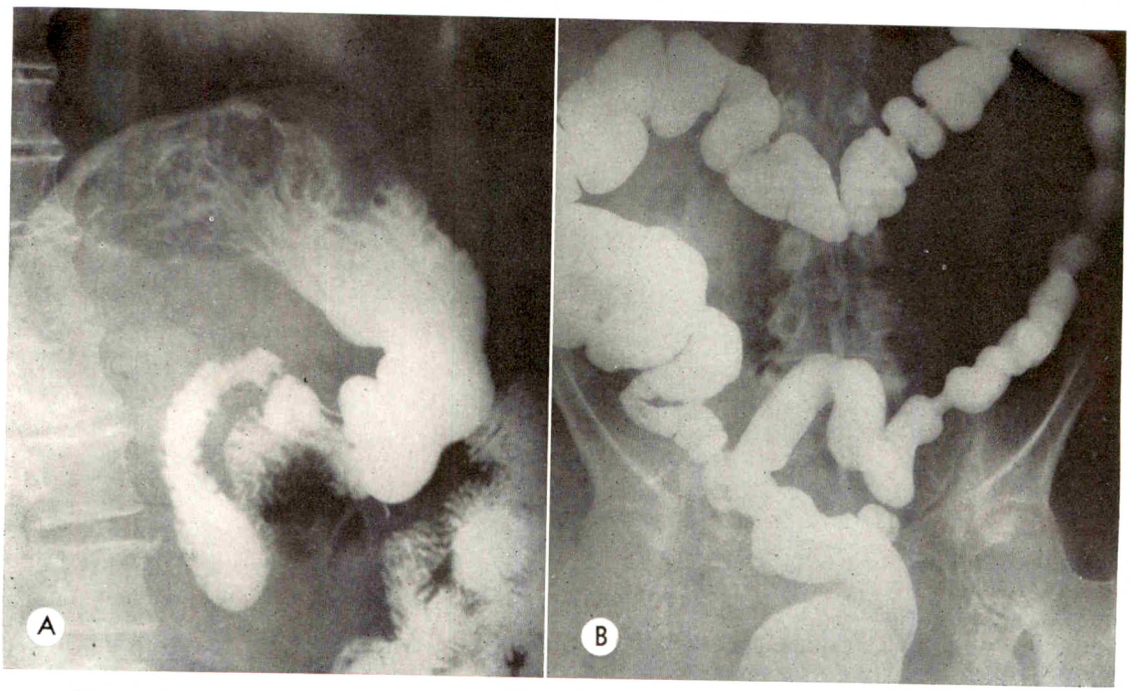


Fig. 2. Case 1. (*A*) Normal stomach and (*B*) colon 2 weeks prior to coronary arteriography.

tubation during and after surgery, improved anesthetics and a better understanding and control of fluid and electrolyte balance.

Although marked gastric dilatation is generally easily recognized, it may occasionally present a diagnostic and/or management problem to the clinician and radiologist. In the present paper some illustrative examples of such cases will be discussed.

Report of Cases

Case 1. A 74 year old white female, A.P., was hospitalized in order to evaluate symptoms suggestive of coronary insufficiency. She had, in addition, complained of vague epigastric distress for several years but repeated gastrointestinal examinations had been normal, including upper gastrointestinal and barium enema studies 2 weeks prior to hospitalization (Fig. 2, *A* and *B*). Approximately 24 hours following coronary arteriography, she developed acute epigastric pain, vomiting and abdominal distention. A plain roentgenogram of the abdomen (Fig. 3) revealed marked dilatation of the stomach as well as a distended, contrast-filled bladder. A diagnosis of "acute gastric dilatation" was made, and immediate intubation of the stomach resulted in the withdrawal of 2 liters of fluid. Following intravenous feeding and further nasogastric suction there was a diminution in the abdominal distention and a disappearance of all symptoms. Despite the

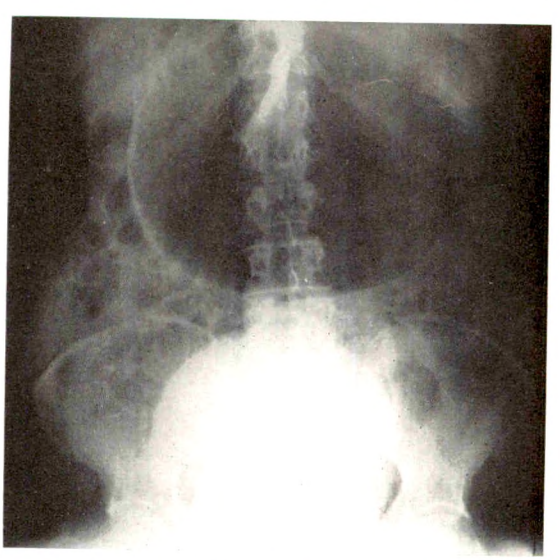


Fig. 3. Case 1. Plain abdominal roentgenogram 24 hours after coronary arteriography showing marked gaseous dilatation of the stomach and distended, contrast-filled bladder.

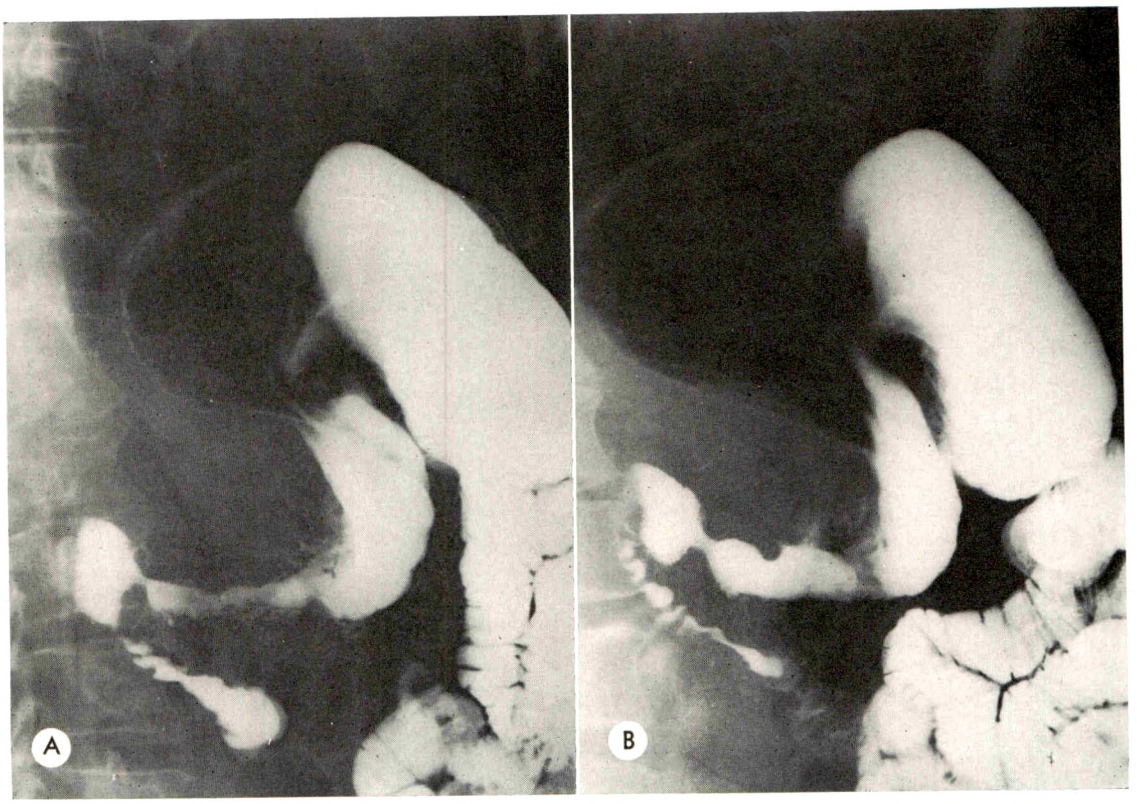


Fig. 4. Case 1. (*A* and *B*) Oblique views from upper gastrointestinal series showing marked elevation, angulation and twist of a segment of the mid-portion of the body of the stomach.

diagnosis of acute "functional" gastric dilatation, her doctor requested a further upper gastrointestinal examination and this, surprisingly, demonstrated a marked elevation, angulation and twist of a segment of the midportion of the body of the stomach (Fig. 4, *A* and *B*). Delayed roentgenograms several hours later (Fig. 5, *A–C*) revealed that this localized twist of the stomach was secondary to transdiaphragmatic herniation of the distal transverse colon through the foramen of Morgagni.

Case II. A 44 year old white female, M.B., was admitted to hospital complaining of increasing abdominal girth. Apart from constipation and one episode of vomiting 3 days previously, she had no other symptoms and generally felt well. There was a past history of an appendectomy during childhood. Physical examination revealed marked abdominal distention with signs suggestive of ascites. Plain roentgenograms of the abdomen (Fig. 6, *A* and *B*) showed a diffuse hazy density with loss of soft tissue detail and obliteration of the hepatic angle. Apart from a small amount of gas and fecal residue in the right colon, there was a general absence of gas in the bowel, and the relationship of the colon to the properitoneal fat lines could not be determined. An upright roentgenogram revealed an increased density shifting to the pelvic cavity. The roentgenograms were interpreted as being consistent with gross ascites, and a diagnostic abdominal paracentesis was performed, resulting in the withdrawal of a small amount of thick, greenish material. After a careful review of the abdominal roentgenograms, the possibility of a massively distended stomach was raised and following intubation of the stomach and the introduction of gastrografin (Fig. 7) this was confirmed. The stomach, in a position of organoaxial rotation, was seen to occupy almost the entire abdominal cavity. Following the withdrawal of approximately 14 liters of fluid over a period of 1 week, a repeat study (Fig. 8) revealed some reduction in the degree of gastric distention. Although further improvement was obtained, some dilatation persisted and with

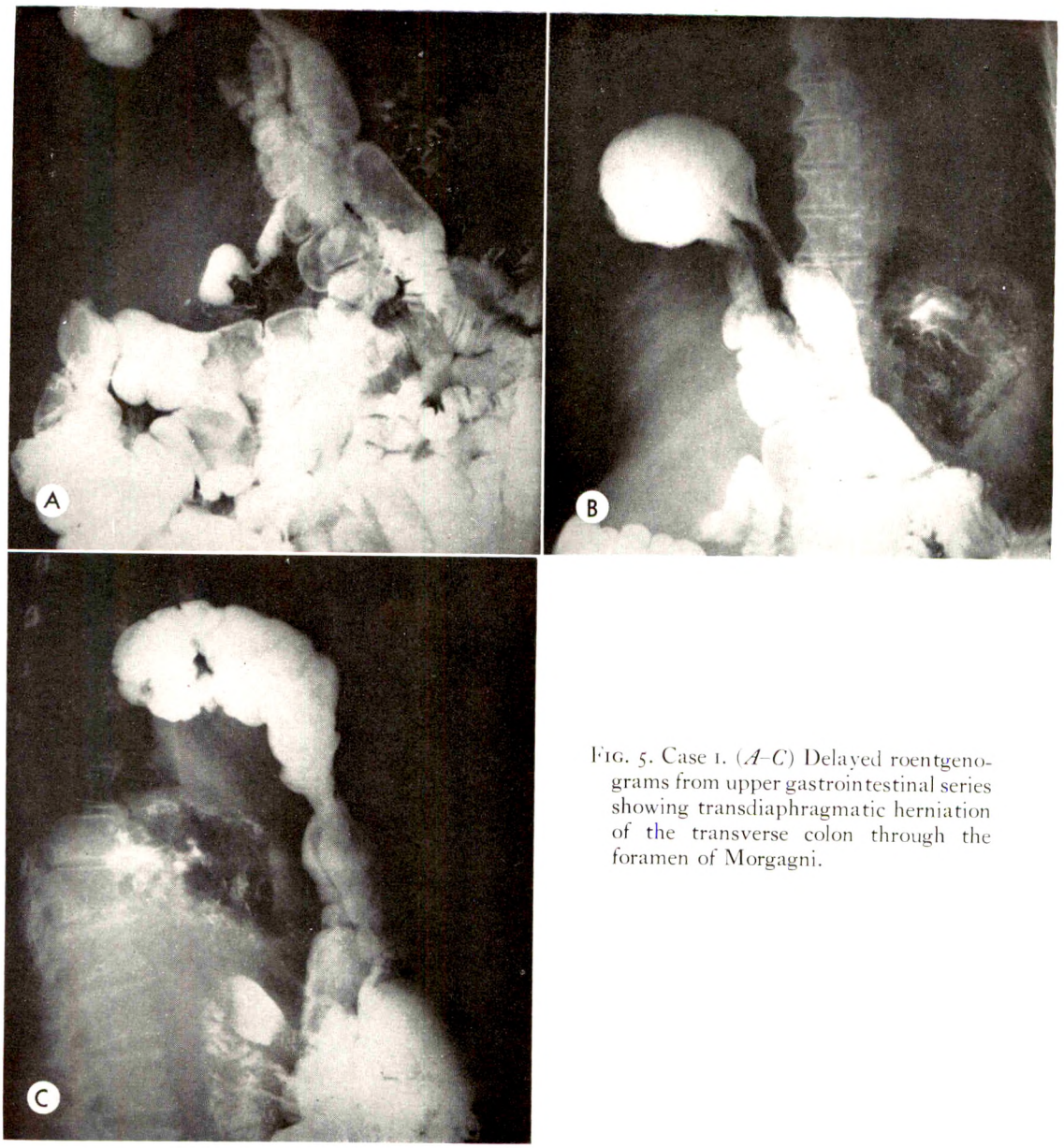


Fig. 5. Case I. (*A–C*) Delayed roentgenograms from upper gastrointestinal series showing transdiaphragmatic herniation of the transverse colon through the foramen of Morgagni.

the development of progressive dilatation of proximal jejunal loops, surgical exploration was performed. This revealed numerous dense, fibrous adhesions producing partial obstruction of the small bowel and fixing the dilated stomach in a position of organoaxial rotation. Following lysis of the adhesions, the patient made a slow but uneventful recovery over a period of 2 weeks, and the roentgenologic findings reverted to normal.

Case III. A 75 year old white male, J.M., was admitted to hospital complaining of increasing abdominal distention, constipation and persistent retching. Physical examination revealed diffuse abdominal distention with active bowel sounds, but no palpable, discrete masses. Plain roentgenograms of the abdomen (Fig. 9, *A* and *B*) showed marked gastric dilatation, gas-distended loops of small bowel and gross fecal loading of the colon. Intubation of the stomach was attempted, but was unsuccessful as a nasogastric tube could not be passed beyond the distal end of the esophagus. A gastrografin examination revealed total obstruction to the passage of contrast material in the

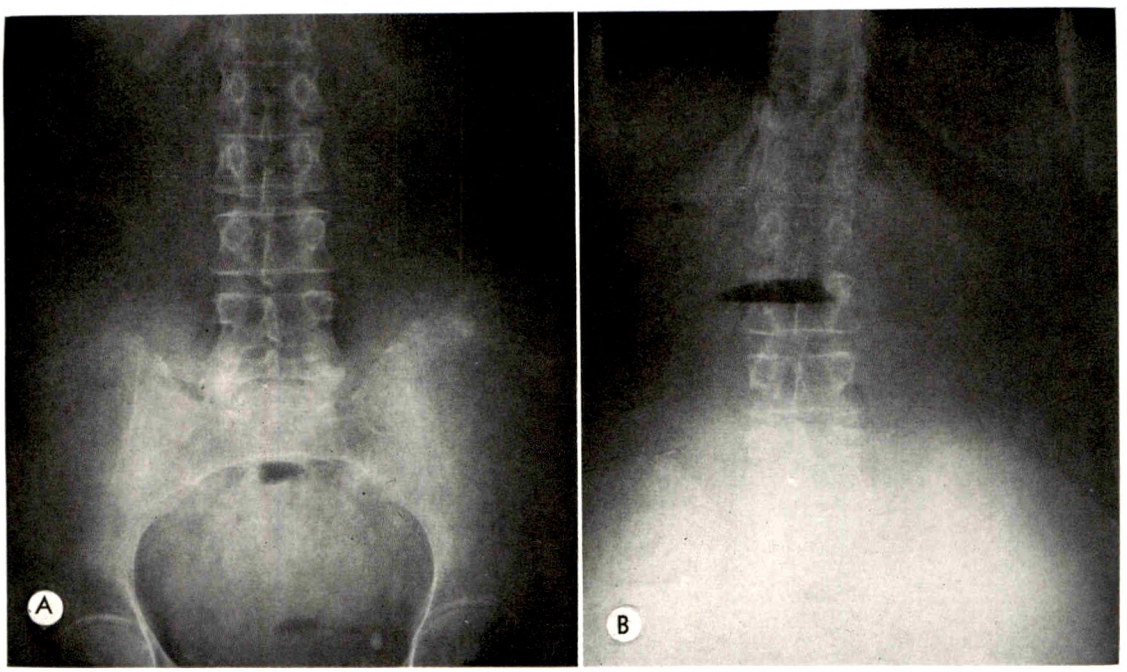


Fig. 6. Case II. (*A*) Supine abdominal roentgenogram showing diffuse, hazy density with obliteration of the hepatic angle and minimal gas in the bowel. (*B*) Upright roentgenogram showing shift of increased density to the lower abdomen and pelvis.

region of the esophago-gastric junction (Fig. 10, *A–D*) and the distal portion of the esophagus was tapered in appearance. This obstruction to the passage of contrast material was present with the patient in the supine, prone and upright positions. In view of increasing abdominal distention, persistent retching, and the inability to intubate the stomach, the possibility of acute gastric volvulus was considered and exploratory surgery performed. Following the administration of a general anesthetic, further efforts to intubate the stomach by means of a nasogastric tube were made, but were unsuccessful. At surgery there was evidence of gross dilatation of the stomach but no signs of gastric volvulus. In addition there was dilatation of the small bowel and marked fecal loading and impaction in the colon. Immediately following decompression of the stomach by means of a gastrostomy, the nasogastric tube was easily passed beyond the esophago-gastric junction into the stomach.

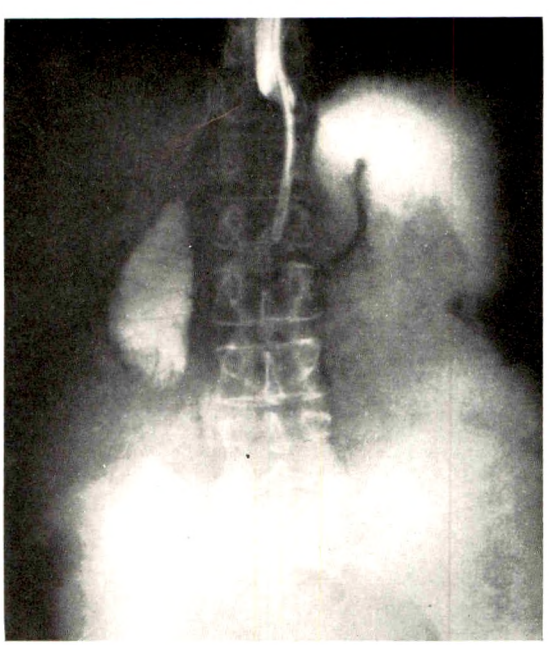


Fig. 7. Case II. Contrast material introduced *via* nasogastric tube outlines the massively distended stomach in position of organoaxial rotation.

DISCUSSION

Each of the above cases was selected in order to illustrate different problems associated with the diagnosis and/or management of marked gastric dilatation.

In Case I the sudden onset of marked dilatation of the stomach following coronary arteriography in a patient in whom

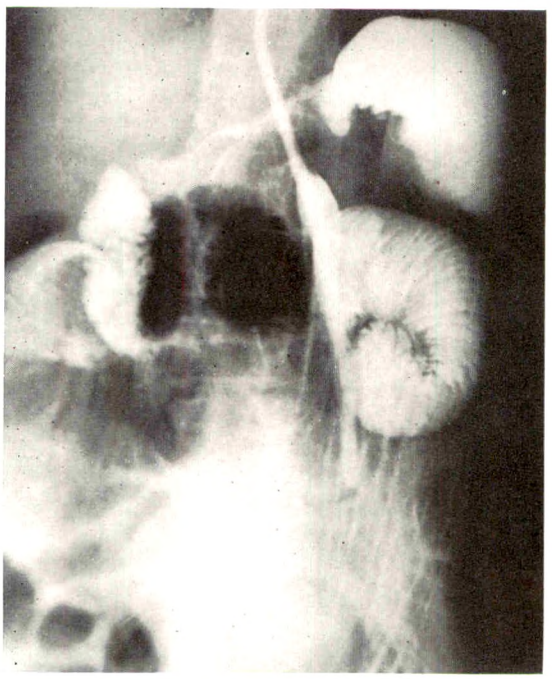


Fig. 8. Case II. Repeat barium study 1 week later showing residual gastric distention. The cardia and fundus are situated in the lower abdomen with the antrum in the left upper quadrant and the duodenum directed to the right and downwards. Note also dilatation of the proximal jejunum.

pre-angiographic barium studies of the upper and lower gastrointestinal tract were normal, suggested a diagnosis of acute "functional" gastric dilatation. In actual fact a partial volvulus, in which a segment of the body of the stomach was elevated and twisted secondary to transdiaphragmatic herniation of the transverse colon through the foramen of Morgagni, was demonstrated. Volvulus of the stomach is generally described as being total or partial. In total volvulus there is a rotation of the stomach around its longitudinal axis (organoaxial volvulus) or the rotation is from right to left or left to right about the long axis of the gastrohepatic omentum (mesenteroaxial volvulus).[2] In the former, the stomach may be regarded as having rotated around a line that connects the cardia and pylorus, while in the latter, the twist occurs about a line that connects the middle of the lesser and greater curvatures. It is generally agreed that the one factor which is essential for the development of total gastric volvulus is a relaxation or lengthening of the ligamentous supports of the stomach, especially the gastrohepatic and gastrocolic omenta. This may be congenital in origin. Gastric atony is probably a principal predisposing factor in the presence of such lengthening of the ligamentous supports. Conditions which are most commonly associated with secondary gastric volvulus are hiatal hernia, and eventration or paralysis of the diaphragm, although a number of other associated conditions, such as gastric ulcer, benign or malignant neoplasms, massive colonic distention, sple-

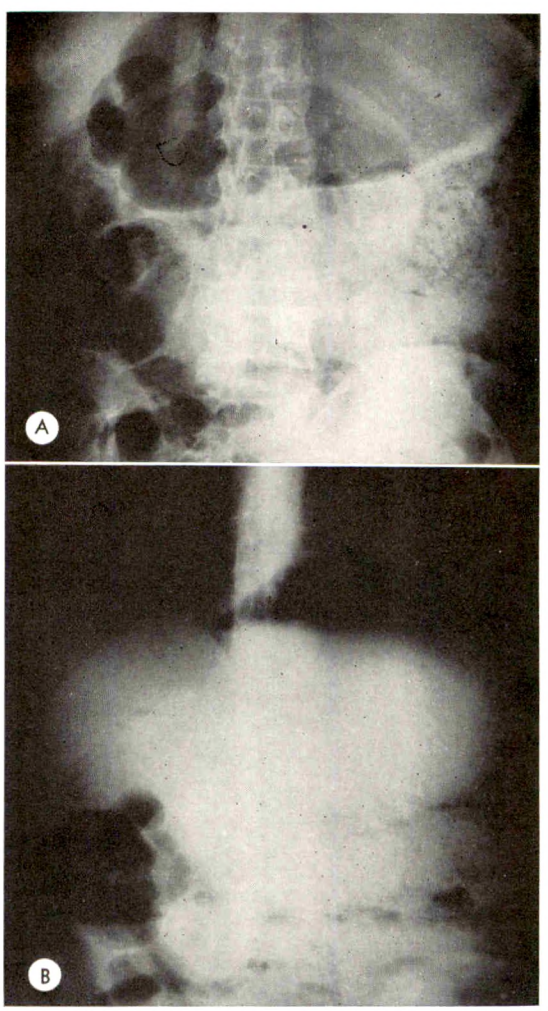


Fig. 9. Case III. (*A*) Plain roentgenogram of the abdomen showing marked dilatation of the stomach. (*B*) Upright roentgenogram showing large gas and fluid collection in the distended stomach.

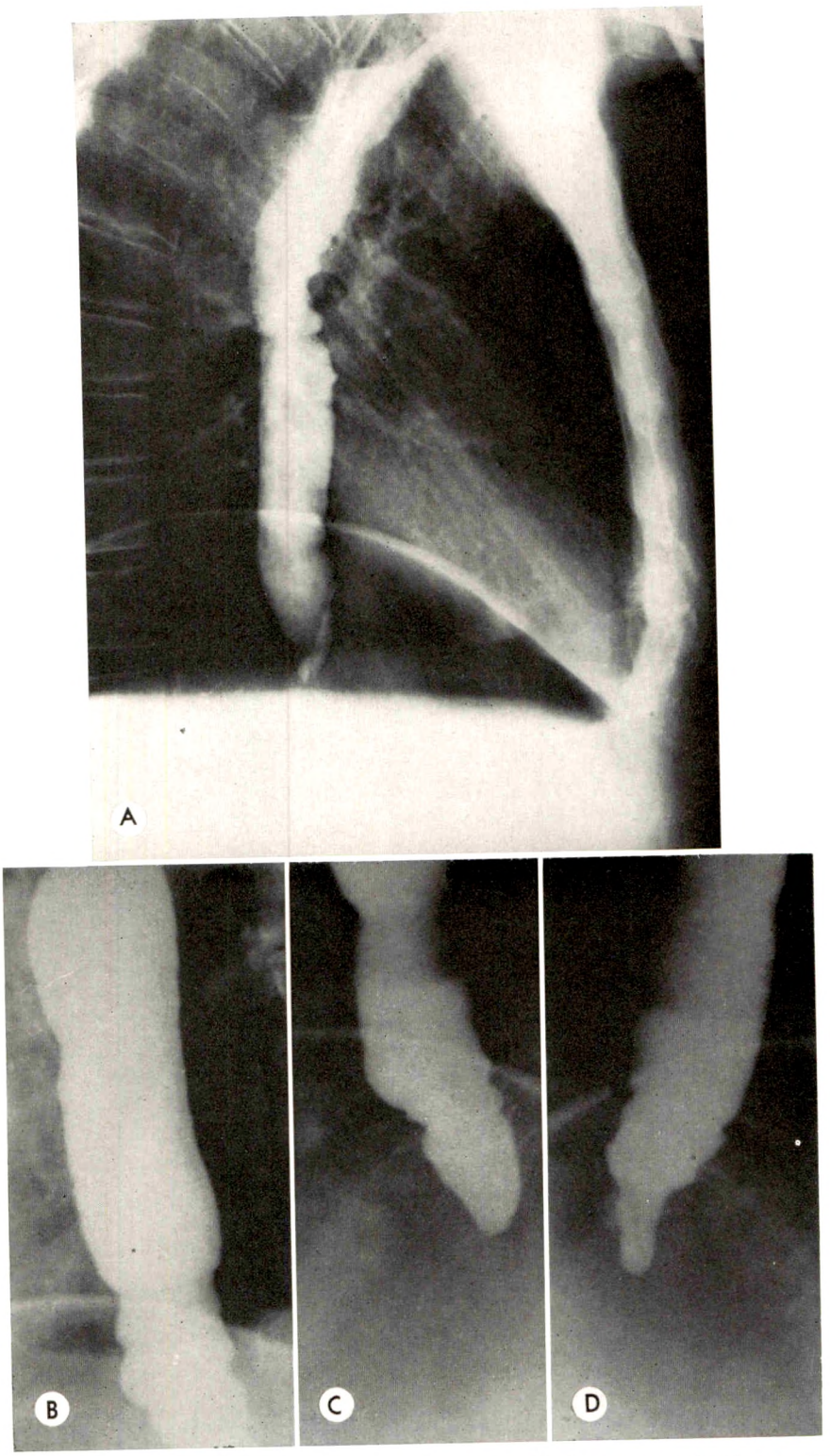


Fig. 10. Case III. (*A*) Lateral upright roentgenogram of contrast-filled esophagus showing total obstruction in the region of the esophago-gastric junction. Note also the long air-fluid level in the massively distended stomach. (*B–D*) Fluoroscopic spot roentgenograms of the esophagus showing total obstruction, and tapered distal portion with active contractions.

nomegaly and splenectomy have been described. These are probably incidental findings as it is difficult to envisage how, in themselves, they can produce gastric volvulus.

Partial volvulus of the stomach is less common. In most cases where the rotation is limited to a localized segment, it is usually the pyloric portion of the stomach which is involved.[2] In a number of such patients an hour-glass stomach has been present, and this is believed to be a definite etiologic factor in this type of volvulus. Usually, only the portion between the constriction and the pylorus rotates.

In the case under discussion, partial or segmental gastric volvulus was secondary to herniation of the transverse colon through the diaphragm at the site of the foramen of Morgagni. This is the opposite of the more usual situation associated with organoaxial rotation of the stomach in which the body of the stomach may be closely related to the anterior margin of the diaphragm in the region of the foramen of Morgagni, and where, in fact, actual herniation of omentum is sometimes present with the transverse colon pulled upward in an inverted V fashion. The attachment of the gastrocolic omentum to the greater curvature of the stomach and the transverse colon probably accounts for this, and for the occasional association of organoaxial rotation of the stomach with hepatodiaphragmatic interposition of the transverse colon.[1] Case I emphasizes the importance of studying the colon in the presence of an otherwise unexplained partial gastric volvulus. Chest roentgenograms may also be helpful in this respect, although in our patient no prior abnormality was present.

Case II illustrates the clinical and roentgenologic difficulties which may be associated with the recognition of gross dilatation of the stomach. In this patient massive gastric distention simulated ascites, and abdominal paracentesis resulted in the withdrawal of gastric content *via* the needle. The gastric dilatation was presumably insidious in onset and chronic in nature, accounting for the virtual absence of symptoms in spite of the remarkable degree of distention of the stomach. As a rule, gastric dilatation is readily recognized on plain roentgenograms of the abdomen, in which the admixture of gas and fluid in the stomach and the caudally convex greater curvature crossing the mid or lower abdomen are diagnostic (Fig. 1, *A* and *B*). However, in the presence of massive distention of a fluid-filled stomach, especially if associated with an organoaxial rotation, in which the greater curvature is located above the lesser curvature with the pyloric portion and duodenal bulb directed downward and to the right, these typical findings may be absent. Moreover, if the hepatic angle is obliterated, as it may be by only a small collection of free intraperitoneal fluid or by fecal material in the ascending and transverse colon,[3] the appearances may simulate ascites. In equivocal cases the introduction of air or barium into the colon as suggested by Keeffe *et al.*[3] is helpful to better evaluate the relationship of the colon to the lateral properitoneal fat lines, since this so-called "flank stripe" sign and the hepatic angle sign[4] are the most useful and reproducible criteria for the diagnosis of ascites. The present case re-emphasizes the unreliability of such nonspecific signs of ascites as diffuse abdominal haziness, poor definition of abdominal organs, separation and floating of bowel loops and increased density shifting to the pelvis in upright roentgenograms.

Case III presented both a diagnostic and management problem. As a rule, the acute onset of gastric dilatation associated with obstruction to the passage of a nasogastric tube in the region of the esophago-gastric junction is highly suggestive of an acute gastric volvulus.[2] In this patient, however, surgical exploration failed to reveal any evidence of a volvulus of the stomach and, although the presence of some degree of a localized twist at the esophago-gastric junc-

tion could not be entirely excluded, it was not associated with any recognizable rotation of the stomach. It is suggested that the obstruction to the passage of both the nasogastric catheter and orally administered contrast medium may have been primarily due to the gross mechanical distention of the stomach. This hypothesis is supported by the ease with which gastric intubation was achieved immediately following simple decompression of the stomach by a gastrostomy opening.

SUMMARY

Gastric dilatation may be the result of organic obstruction or may occur as an acute "functional" phenomenon.

In the vast majority of patients both the presence and nature of the gastric dilatation are easily determined by clinical and roentgenologic means.

Occasionally, marked dilatation of the stomach gives rise to a diagnostic and/or management problem. Some illustrative examples of such cases are presented. These include partial gastric volvulus secondary to transdiaphragmatic herniation of the transverse colon through the foramen of Morgagni, massive gastric distention simulating ascites and obstruction at the esophago-gastric junction associated with gross distention of the stomach.

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THE STRUGGLING ANTRUM*

A NEW SIGN OF PERIGASTRIC MALIGNANCY

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THE diagnosis of pancreatic carcinoma is usually dependent on a nonspecific clinical presentation and roentgen changes in the duodenal sweep. With a conventional gastrointestinal series, the duodenum is often difficult to study effectively due to rapid transit of the barium through this segment. In these circumstances, a definite statement as to the presence of a pathologic process in the pancreas cannot be made. This entails repeat examinations and in doubtful cases further studies such as hypotonic duodenography and visceral (selective) angiography. These latter examinations present the possibility of morbid sequelae, extra hospitalization time and expense. We have found that certain abnormalities of antral mobility are often associated with extrinsic neoplasm, most commonly carcinoma of the pancreas. The appearance of the gastric antrum is therefore one additional factor which may be utilized in deciding which patient should be selected for further investigation.

To understand these pathologic alterations in the antrum and its motor pattern, an accurate knowledge of the normal gross anatomy and peristaltic activity in this region is necessary.

NORMAL ROENTGEN ANATOMY AND MOTILITY

The stomach comprises 2 major anatomic segments, the longitudinal and the transverse. The longitudinal portion consists of the fundus, that portion above the cardiac orifice, and the body, that portion below this orifice down to the level of the incisura angularis. The gross anatomic and roentgen definition of the antrum includes the entire transverse stomach distal to the incisura angularis. Based on its gross anatomy and mechanical activity, the antrum may be conceived of as comprising 2 subdivisions. The proximal $\frac{2}{3}$ of the antrum is then designated the sinus antri and the distal $\frac{1}{3}$, about 2–3 cm. in length, the canalis (Forssell).[7]

The muscularis propria of the longitudinal portion of the stomach consists of 3 layers, the inner oblique muscle with the greatest concentration of muscle fibers along the lesser curvature, the middle circular muscle, and the outer longitudinal layer. In the sinus antri the outer longitudinal muscle layer collects into thick strands in the anterior and posterior walls. The inner circular layer is completely circumferential. In the canalis, on the other hand, the outer longitudinal layer is also complete. Circular muscle fibers are most abundantly developed in this area and are interlaced with fibers from the outer longitudinal layer to form a sphincter-like mechanism.

The peristaltic pacemaker is intrinsic to the gastric wall. The conduction wave is primarily transmitted along the lesser curvature, where there is believed to exist a concentration of the nerve cells of Auerbach's plexus lying between the circular and longitudinal muscle layers.[2–6,8,9]

Normal gastric peristalsis when fully developed proceeds as a smooth broad contraction band approximately 2 cm. in length, at a maximum sequential rate of 3 per minute. It is first roentgenographically visible in the mid-body of the stomach where it is shallow. The prominent wave is detected in the region of the incisura angularis. When first detected, the peri-

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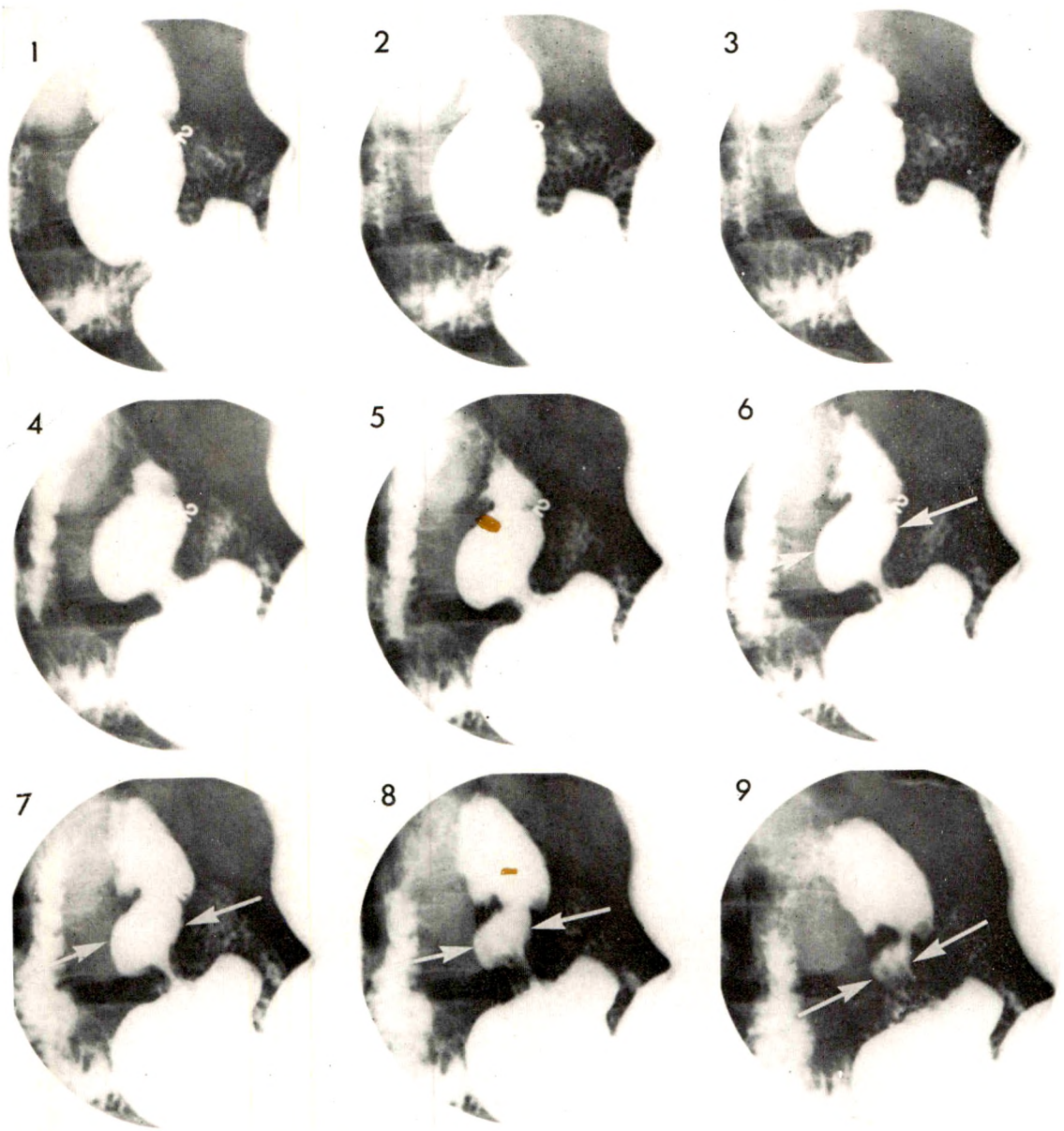


Fig. 1. Normal antral motility. The peristaltic contraction ring is smooth and symmetric; *i.e.*, of almost equal length on both its lesser and greater curvature aspects, and demonstrates smooth, symmetric, sloping proximal and distal borders. The contracted segment is centrally located with respect to the distended proximal and distal portions of the antrum. The folds in the contracted area are parallel, thin, sharp and oriented in the long axis of the antrum. As the contraction deepens, the segment narrows and elongates at the expense of the canalis which now enters the systolic phase. The canalis (arrows) during systole narrows and shortens in symmetric fashion or shows minimal flattening on the lesser curvature with pseudopouch formation on the greater curvature as seen in this case. The lumen of the canalis is centrally located with respect to the distended antrum proximally and the base of the duodenal bulb distally. Just proximal to the contracted canalis, retrograde prolapse of gastric folds is evident with the folds smoothly and symmetrically distributed about the centrally placed lumen of the contracted segment. Note that filling of the bulb (frames *3–9*) occurs during canalis contraction. At the end of canalis contraction, the pyloric ring also narrows (frame *9*).

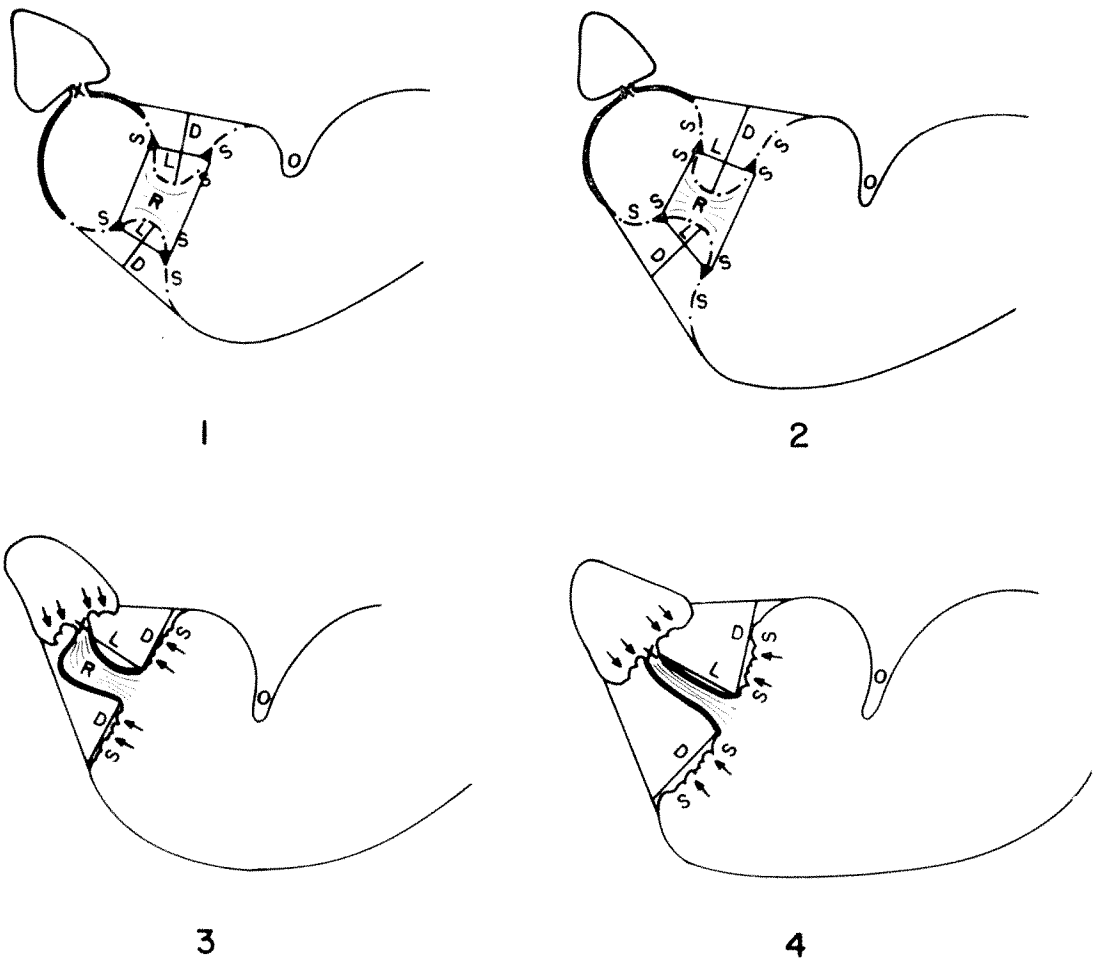


Fig. 2. Schematic representation of antral peristaltic activity and canalis contraction. The canalis is illustrated as having a thicker wall.

(*1*) Early phase of peristaltic contraction with smooth slopes and central contracted segment. (*2*) Deepening of the peristaltic wave with narrowing and elongation of the contracted segment and steeper slopes. (*3* and *4*) Canalis contraction with flattening of the lesser curvature and pseudopouch formation in *3*. The contracted canalis is central with evidence of slight orthograde and retrograde prolapse of the mucosal folds. The distended sinus antri between the incisura (O) and canalis drapes symmetrically over the retropulsed folds.

staltic wave is more prominent on the lesser curvature than on the greater curvature, but then proceeds in smooth continuous symmetric fashion through the sinus and most often ends at the proximal margin of the canalis. As the contraction proceeds through a segment, that segment narrows and shortens. The gastric folds in this segment are compressed and slide unostensibly proximally to be taken up in the proximal distended portion through which the wave has recently passed. Less commonly, the gastric folds slide in orthograde fashion into the distal distended segment. Orthograde and much more commonly retrograde prolapsed folds are identifiable at the junction of the contracted and distended segments. These folds appear unobtrusive, smooth and symmetrically disposed on either side of the markedly contracted lumen. The stomach proximal and distal to the contraction wave balloons smoothly and symmetrically, producing inferior and superior convex margins. Usually, the ballooning walls of these distended segments drape smoothly over the most peripheral margins of the prolapsed folds. The more vertical proximal and distal margins of the peristaltic wave, or the slopes of the peristaltic wave, demonstrate a smooth convex contour in the early phase of contraction. As the contraction ring deepens, the slope assumes a smooth sigmoid configuration, concave nearest to the contracted lumen and convex more peripherally, where the slope blends imperceptibly with the smooth contour of the proximal and distal distended segments.

Peristalsis rarely continues through the canalis. The canalis distends and contracts as a unit. Canalis contraction follows almost every peristaltic wave. Independent contraction of the canalis, however, does not occur. The canalis narrows and shortens in this contraction phase. The gastric folds are evenly compressed and propelled in either retrograde or less commonly orthograde fashion. Retrograde prolapse of the gastric folds is most commonly observed

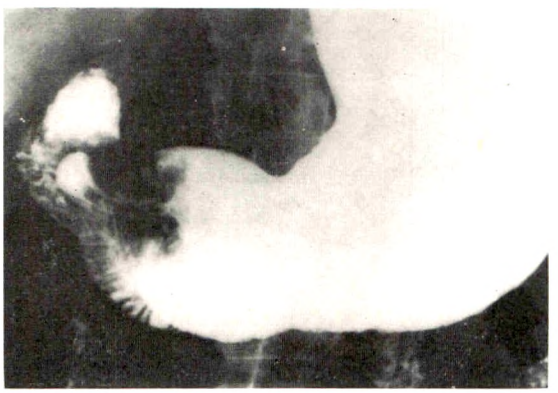


Fig. 3. Retrograde prolapse of gastric folds. Retrograde prolapse of gastric folds is seen normally and is occasionally exaggerated. The contracted canalis is central to the proximal maximally distended sinus antri. The folds within the canalis are thin and smooth. The retropulsed folds are disposed symmetrically and in orderly fashion above and below the contracted lumen which lies centrally. The distended sinus antri drapes smoothly over the retropulsed folds.

proximal to the maximally contracted canalis. Contraction of the canalis is not necessarily associated with pyloric opening and gastric emptying, a phenomenon which is influenced by multiple factors. Contraction of the canalis is often symmetric. When asymmetric, greater shortening along the lesser curvature produces the appearance of a pseudodiverticulum due to a relative bulging of the greater curvature (Fig. 1; 2; and 3).

Normal antral motility may not always be represented by this smooth pattern, especially in older patients. In this older age group, incomplete, ineffective contractions may occur and result in the presence of transient shallow smooth grooves and indentations in the sinus antri. In addition, complete canalis contraction occurs less frequently (Fig. 4).

PATHOLOGIC ROENTGEN FINDINGS

It is well known that carcinoma of the pancreas may affect the upper gastrointestinal tract. Involvement of the duodenum has been stressed in the literature. Involvement of the stomach also occurs at the level of the body and the antrum, espe-

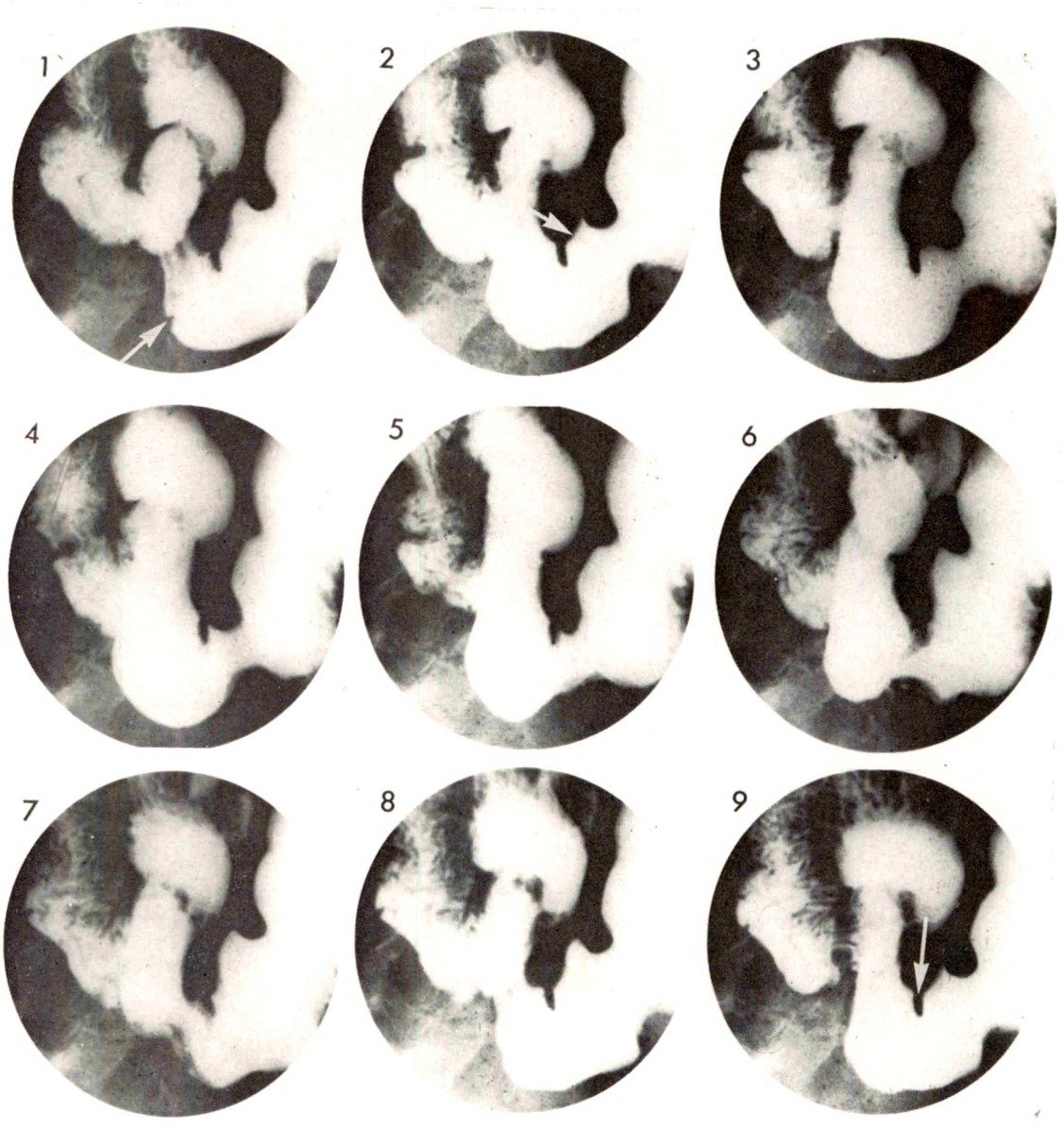


Fig. 4. Normal variations of antral motility often seen with aging; 81 year old female. Several differences from the prototype normal are apparent. The filled antrum is tubular and elongated. The peristaltic pattern is somewhat masked by numerous smooth, transient grooves and indentations (arrows) which may represent irregular relaxation or contraction. The canalis does not contract or empty completely and fails to shorten.

cially along the posterior wall.[1] Partial or complete gastric outlet obstruction is not uncommon in advanced cases. In most of these cases, an obvious perigastric or retrogastric mass is evident.

Fixation of the posterior wall of the gastric antrum only by tumor which does not involve the mucosa will cause abnormalities of distensibility and motility. When the mucosa is penetrated as well, there are, in addition, fold changes which are more readily apparent in the contracted segment.

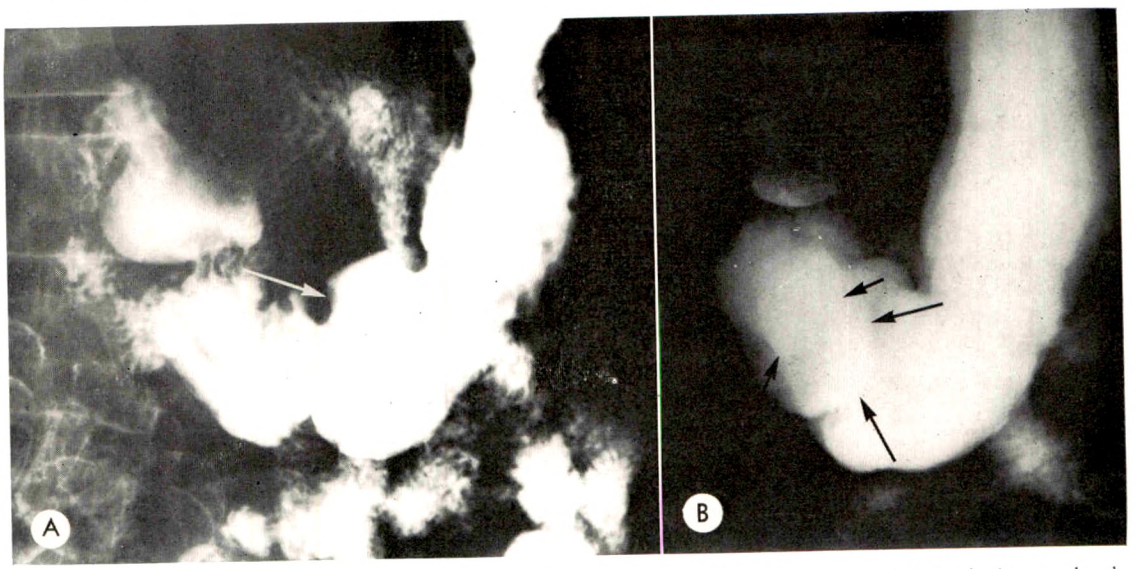


Fig. 5. Carcinoma in the region of the pancreas invading the outer wall of the antrum. (*A*) In the incompletely distended antrum, the phenomenon of multiple loculations due to irregular distention is not apparent. The antrum appears normal but close inspection reveals that the peristaltic wave is abnormal, being broader and irregular along the lesser curvature with flattening of the superior slope (arrow). (*B*) A complex pseudodiverticulum is created in the barium distended antrum. This chamber demonstrates several loculations delineated by overlapping contours (arrows). This is due to fixation and infiltration of the posterior superior wall of the antrum with normal distention of uninvolved wall. There is no obvious perigastric mass.

I. ABNORMALITIES OF DISTENSIBILITY

A. *Multi-loculation*

In the partially filled or contracted state, the circumscribed fixation of one wall of the antrum may not be roentgenographically evident (Fig. 5*A*). When the antrum is filled with a large amount of barium, the normal portion of the antrum, generally the anterior wall and the greater curvature,

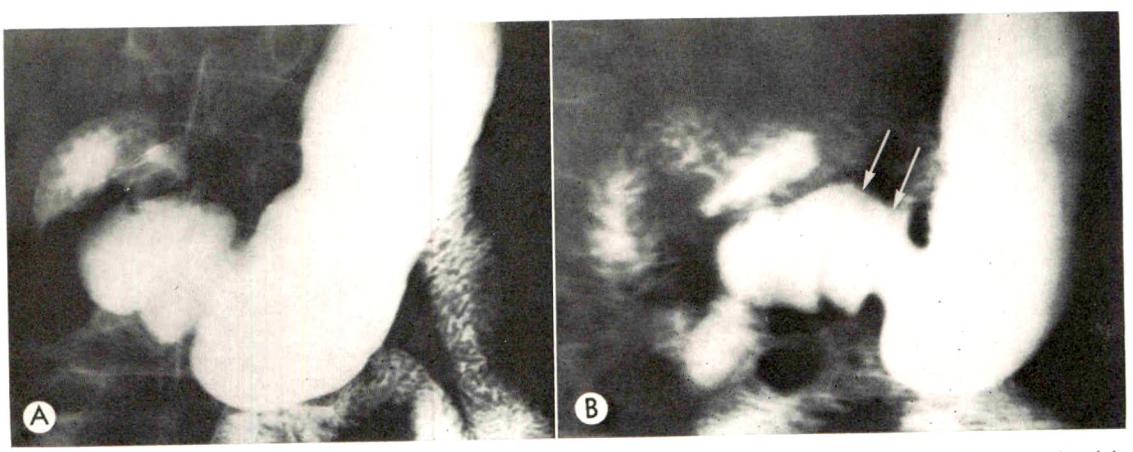


Fig. 6. Carcinoma of the pancreas. (*A*) Vertical ridging in the distended antrum simulates two peristaltic contractions. There are no recognizable slopes and the rings are too narrow to be peristaltic waves. Nodular infiltration of the lesser curvature of the stomach above the incisura angularis is obvious. (*B*) During a phase of partial contraction, the ridges are accentuated. Angulation and fixation of the superior aspect of these ridges are evident (arrows). This may go on to produce pseudodiverticulum formation.

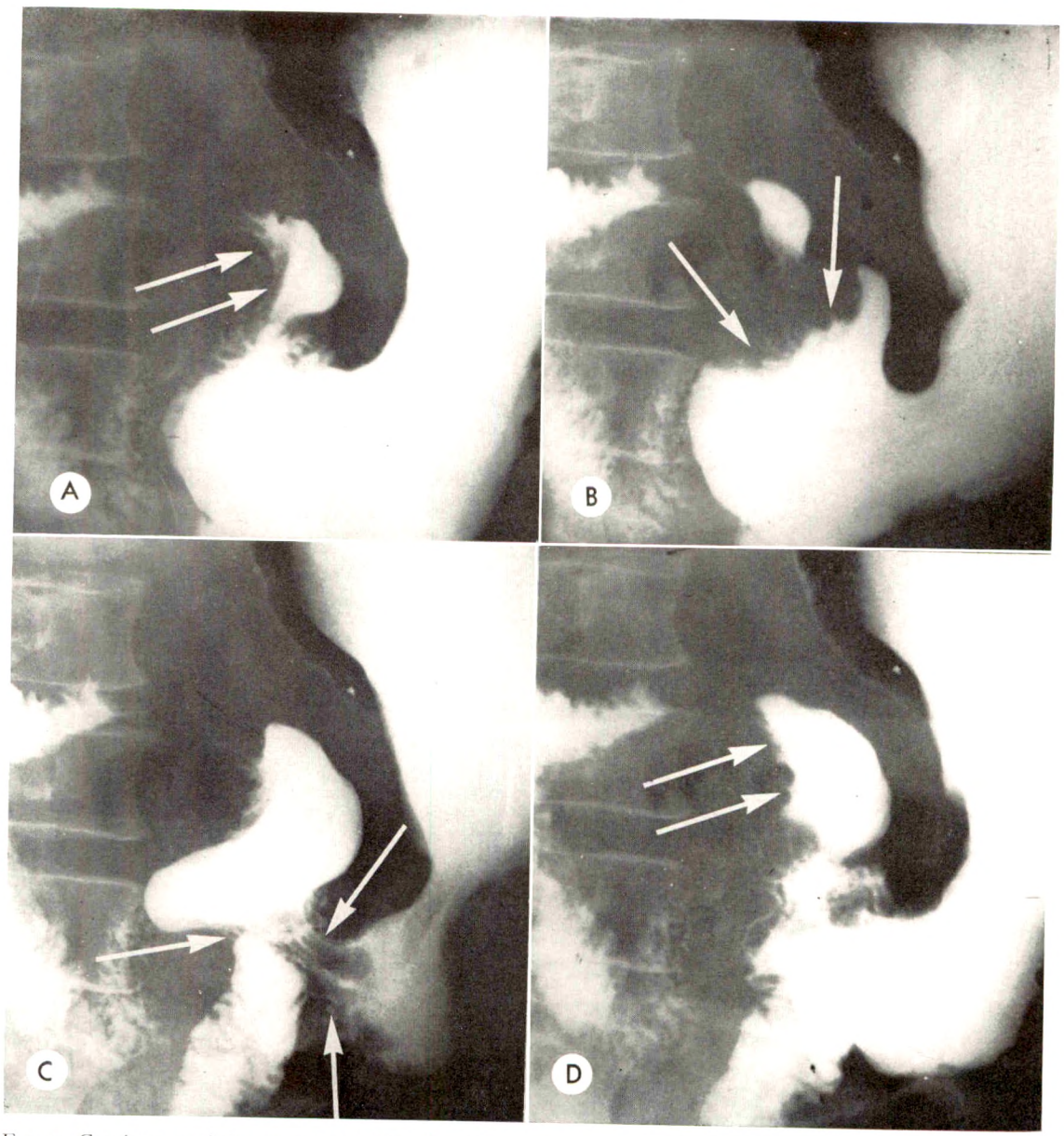


FIG. 7. Carcinoma of the pancreas infiltrating the antrum and duodenum. Polygraph (4 frames) demonstrating abnormal motor activity of the antrum. (*A*) There is a mass infiltrating the duodenal bulb and indenting the canalis primarily along the greater curvature. The mucosal folds in the canalis (arrows) are widened, stretched and fixed in an arcuate configuration by the mass. The duodenal bulb is not filled. The duodenal sweep is incompletely filled. There is flattening and rigidity of the incisura angularis. (*B*) The canalis is contracting in eccentric fashion. The infiltrated folds (arrows) at the proximal margin of the canalis are distorted and crowded and there is failure of draping. (*C*) The nodular infiltration of the region of the incisura is well seen. The distended canalis clearly shows evidence of extrinsic irregular impression on the greater curvature (arrows). (*D*) The peristaltic ring (arrows) is markedly asymmetric. Indentation of the mass is seen flattening the distal slope along the greater curvature. The folds in the contracted segment are broad and stretched.

A small myoma, hardly visible, in the body of the stomach is an incidental finding.

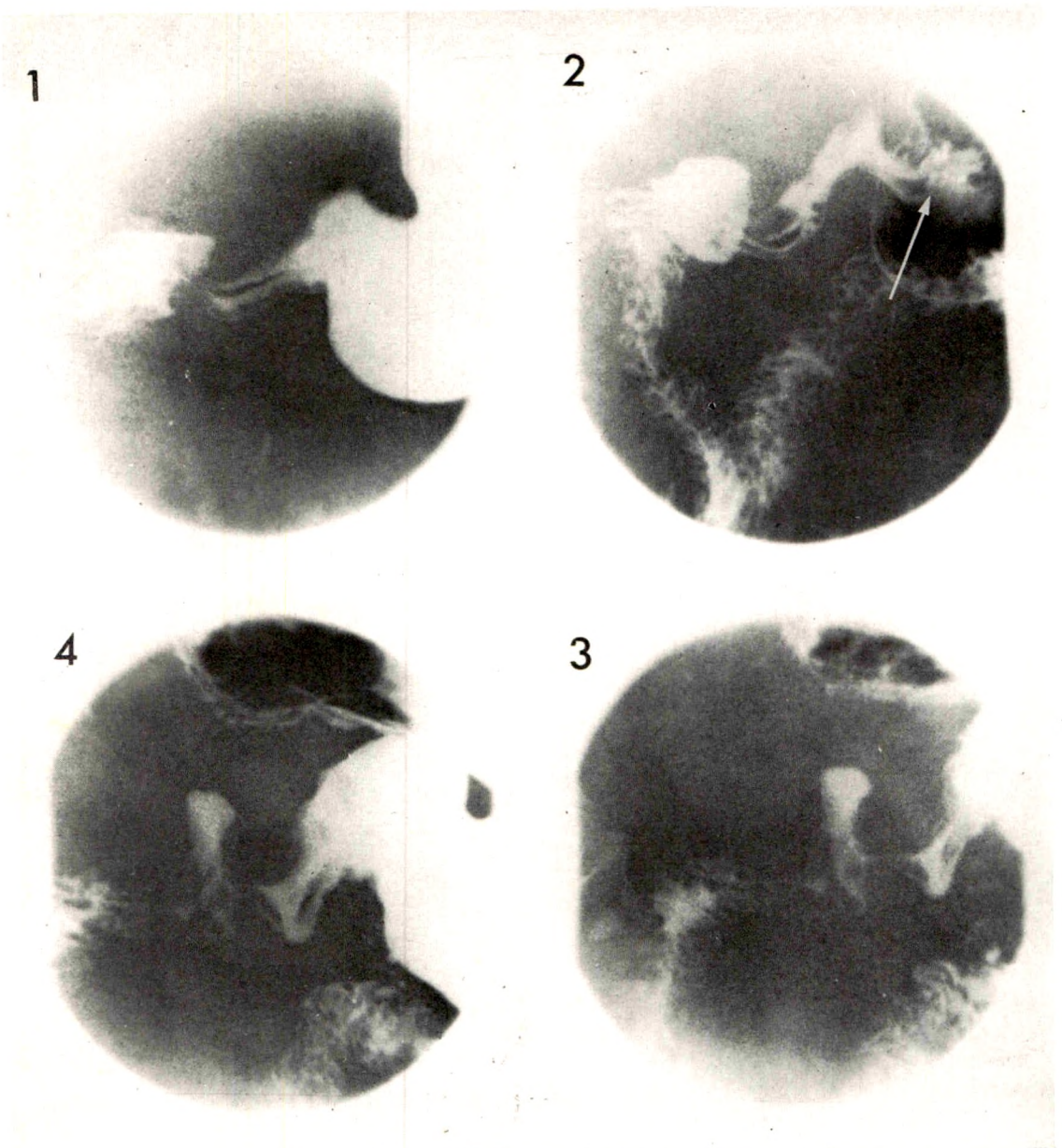


Fig. 8. Carcinoma of the pancreas. Multiple 70 mm. frames obtained a few seconds apart show abnormal contraction and relaxation of the antrum. During its contraction phase, the canalis at its junction with the sinus antri is angulated with uneven proximal ballooning of the distended sinus (frames *1* and *2*). The folds proximal to the contracting canalis are distorted with failure of normal draping (frame *2*, arrow). The folds in the canalis are also uneven in thickness. The descending duodenum is poorly filled in all frames.

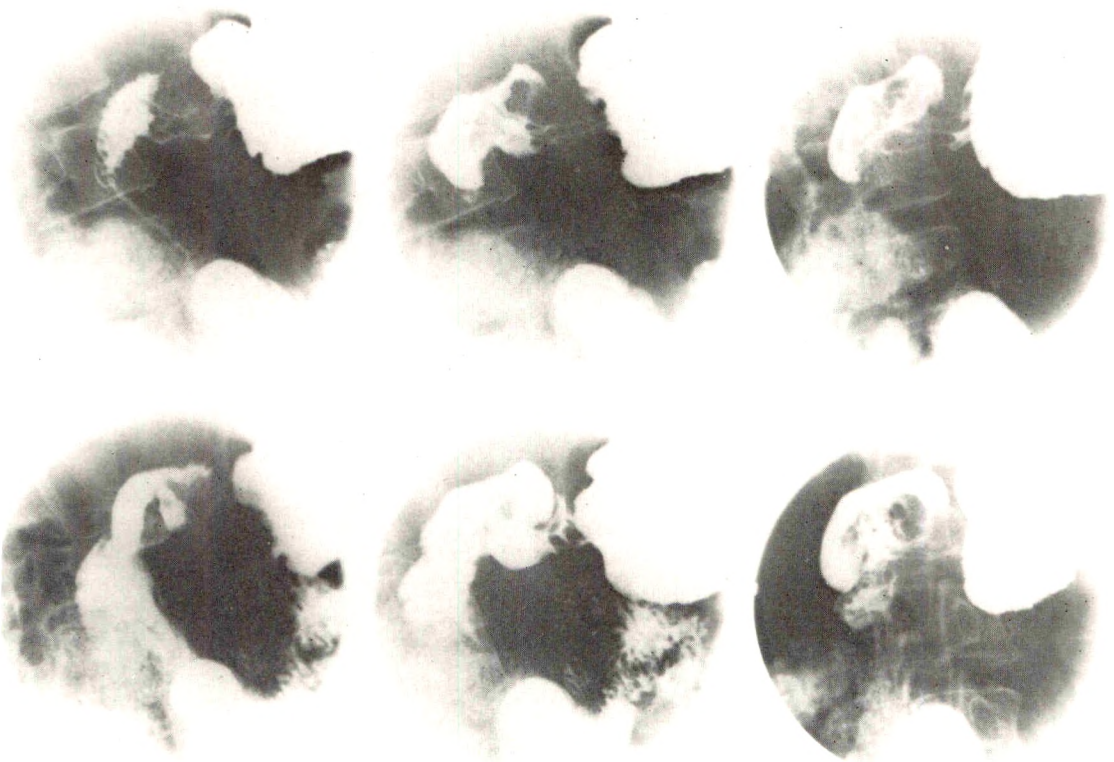


FIG. 9. Carcinoma of the pancreas. All frames show varying degrees of canalis contraction. The folds in the canalis are thick and nodular. The contracted canalis is slightly eccentric. The distended sinus antri angulates at the point of fixation due to uneven distention. The uninvolved portion of the antrum, in distending, drapes over and obscures the fixed segment. In so doing, the axis of the stomach is altered. At no phase does the entire antrum appear normal.

distend. This, in conjunction with the fixed, nondistensible posterior wall creates a pseudodiverticulum. Therefore, the antrum presents as two or more superimposed locules instead of a single smooth chamber (Fig. 5*B*). This is particularly evident in the erect position because the fixed component does not descend to the same extent as the uninvolved portion.

B. *Flattening and ridging*

With the antrum filled with barium, one wall, the wall fixed by the tumor, may fail to distend. This will be reflected in a flattened or slightly concave configuration most often seen along the lesser curvature. Slightly more extensive involvement results in a more complicated pattern of uneven distention. In addition to the lesser curvature flattening, this may result in the presence of fine vertical radiolucencies which simulate a peristaltic wave (Fig. 6*A*). This phenomenon, primarily due to limited distensibility, may be exaggerated during the contraction phase (Fig. 6*B*).

II. ABNORMALITIES OF THE PERISTALTIC WAVE AND CANALIS CONTRACTION

A. *The contracted segment*

(1) Eccentricity

Asymmetry in the depth of the contraction ring is reflected in eccentricity of the contracted segment with respect to the proximal and the distal distended stomach (Fig. 7, *B* and *D*). The contracted segment deviates more often toward the lesser curvature of the antrum because of its

more frequent involvement by the tumor.

Asymmetry of the contracted segment may also be reflected in the length of the segment on its lesser curvature and its greater curvature aspects (Fig. 7*D*). If unevenness of length is evident during contraction, perigastric abnormality can be suspected.

(2) Slopes of the peristaltic wave and canalis contraction

The proximal and/or distal slope of the peristaltic wave and canalis contraction may demonstrate a flat or straight appearance instead of the smooth convex contour or sigmoid configuration seen during passage of the contraction. In addition, there may be irregularities of these slopes seen only during the contraction phase (Fig. 7*D*).

Failure of the normal smooth transition from the convex to the sigmoid phase of the slopes of the contraction may be observed.

B. *Angulation, elevation and rotation of the stomach with the apex at the point of fixation*

This is most evident with the passage of the peristaltic wave through the involved portion of the antrum (Fig. 8). The angulation is then apparent as an abnormality in the relationship of the contracted segment to the proximal distended stomach. Occasionally, striking rotation of the normally distensible anterior wall and greater curvature aspect of the stomach is observed. This is roentgenographically manifested by progressive overlapping of the contracted segment by the caudad draping of the distensible proximal stomach (Fig. 9). In the absence of effectual peristalsis or canalis contraction, the orientation of these 2 gastric regions may at a glance appear normal. Elevation in addition to rotation of the fixed portion of the antrum due to infiltration by tumor may also become more evident during the contraction phase. The depth of the peristaltic wave along the greater curvature is increased because of the lack of mobility of the lesser curvature. This makes the elevation of the antrum more apparent roentgenographically.

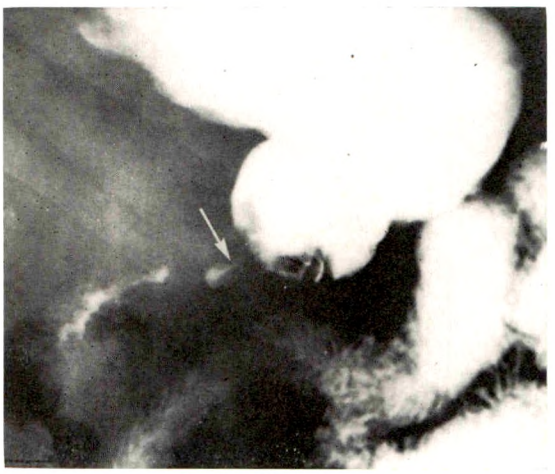


Fig. 10. Carcinoma of the pancreas. The canalis is in systole (arrow). The lumen of the canalis is eccentric in relation to the distended sinus antri. The folds surrounding the entrance to the canalis are bunched, distorted and asymmetric. The sinus antri funnels distally. An obvious nodular infiltration of the upper lesser curvature is noted.

C. *Mucosal fold abnormalities*

When the mucosa is penetrated, there are in addition mucosal fold changes which are more readily apparent in the contracted segment. Infiltrated folds will tend to resist compression in the maximally contracted segment and remain prominent (Fig. 9). The folds may be irregular and nodular. These thick or angulated folds are evident along the margins of the slopes and persist through the entire contraction phase. Proximal to canalis contraction especially, these gastric folds appear unduly prominent (Fig. 10). Instead of the normal minimal retrograde prolapse, the folds appear thickened, distorted and crowded and disposed in an eccentric and irregular manner about the lumen. The wall of the distended stomach proximally may fail to drape over these folds in a smooth and symmetric fashion.

The abnormalities of motility and distensibility described above may be sufficiently characteristic that the diagnosis of a perigastric lesion, most commonly carci-

noma of the pancreas, may be suggested in the absence of other roentgen findings. Abnormalities in the peristaltic pattern may also be seen in intrinsic neoplasms of the antrum. Absence of mucosal ulceration or destruction would exclude an intrinsic process.

SUMMARY

Subtle but analyzable changes occur in the appearance of the antrum during filling and emptying whenever a portion of the wall is fixed, particularly as a result of infiltration by carcinoma of the pancreas.

Abnormalities of distensibility include pseudoloculation, and flattening and ridging of the antrum. Abnormalities demonstrated during the contraction phase include asymmetry and eccentricity, specific alteration of the slopes of the peristaltic wave, fixation, angulation, elevation and characteristic mucosal changes.

These abnormalities of antral distensibility and motor activity reflect the attempts by the uninvolved portion of the antrum to maintain adequate mechanical function. The resultant appearance of a "struggling antrum" may be sufficient to suggest the diagnosis of perigastric tumor.

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Vol. 119, No. 2

A METHOD FOR THE DEMONSTRATION OF THE MUCOSAL PATTERN OF THE DUODENAL CAP*

By HRAYR A. KABAKIAN, M.D.
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THE only reliable evidence for a duodenal ulcer is the roentgenologic demonstration of an ulcer crater.[4,8,9,11–13]

GENERAL CONSIDERATIONS

Stein *et al.*,[12] in their evaluation of conventional roentgenologic techniques for the demonstration of duodenal ulcer craters, state that "no examination for peptic ulcer of the duodenum is complete without exposures of the cap made in the supine position with air contrast and in the prone oblique position with compression that is adequate to reveal the mucosal pattern." In their series, the one single view that alone showed the highest percentage of ulcers (24.5 per cent) was the supine air contrast exposure. On the other hand, 12.5 per cent of the duodenal cap ulcers were seen only on the prone oblique exposure with compression by means of a pneumatic paddle,[3] while none of the ulcers were seen in this position alone without compression. In only 1.8 per cent of the cases was the ulcer seen in the erect position alone. The supine air contrast view, therefore, although singly showing a greater percentage of ulcers, is not a substitute for the prone oblique view with compression. The necessity for an adequate demonstration of the mucosal pattern by compression becomes self evident. It is well known, however, that the mucosal pattern cannot be shown when the cap is high under the ribs (Fig.

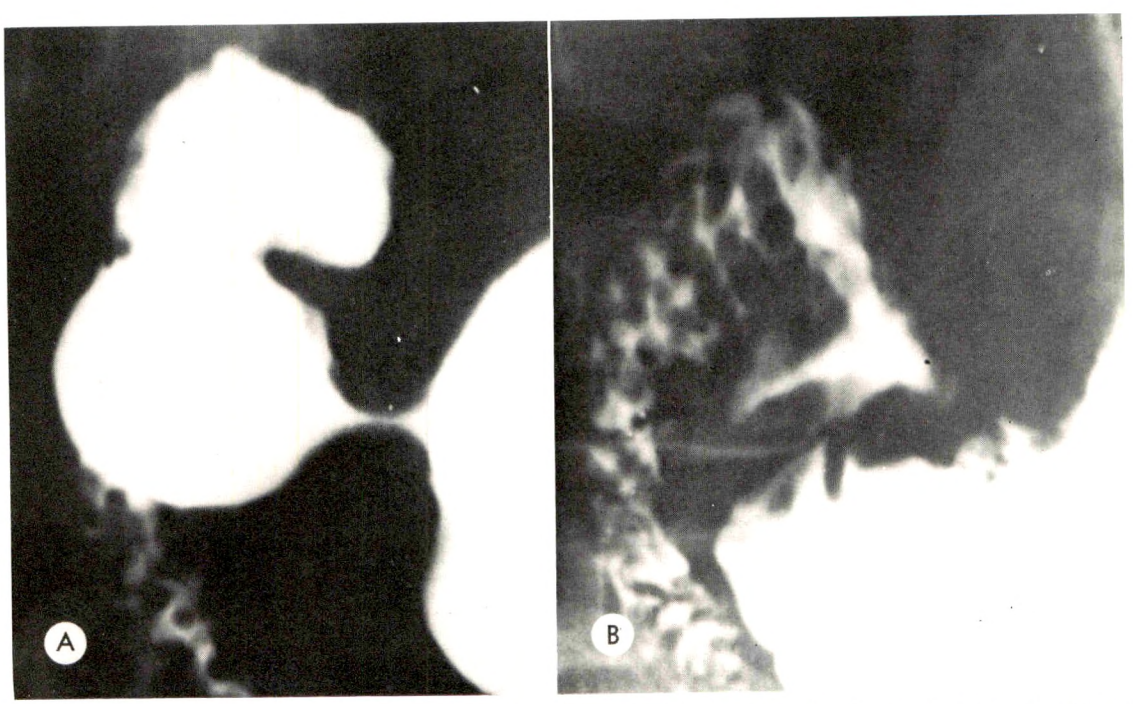


FIG. 1. (*A*) In the prone position, the duodenal cap is situated high under the ribs and compression is not effective in demonstrating the mucosal pattern. (*B*) Deep inspiration makes the cap available to compression and the mucosal pattern is identified.

* From the Department of Radiology, American University Medical Center, Beirut, Lebanon.

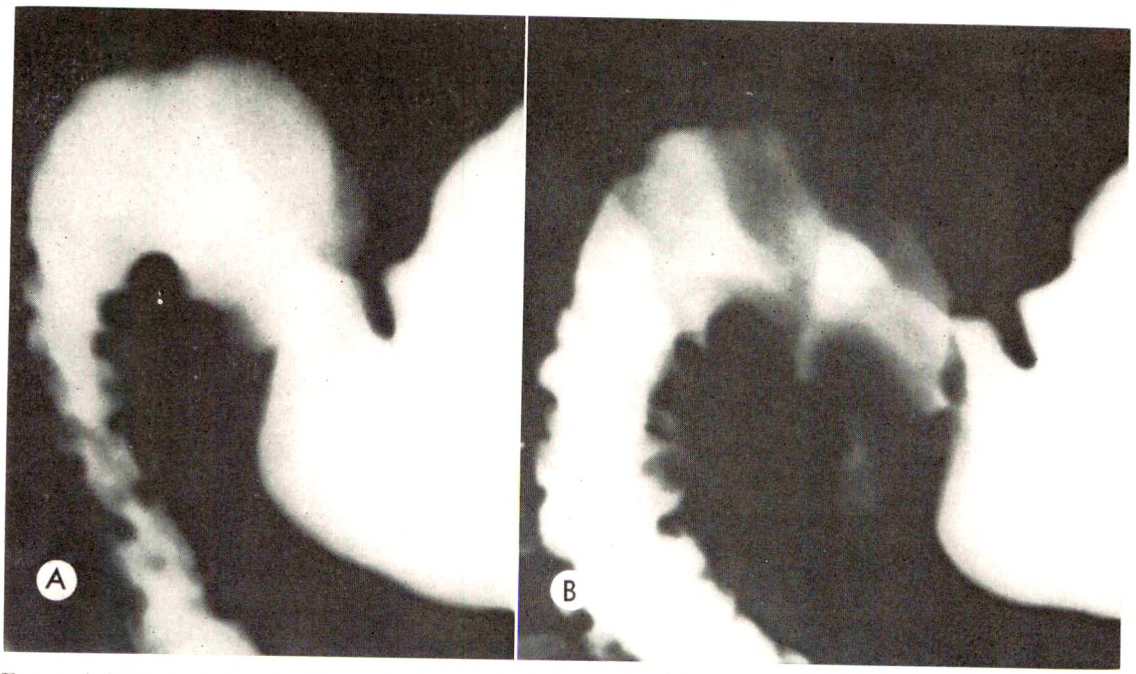


FIG. 2. (*A*) In the prone position, the duodenal cap is situated high under the ribs and compression is not effective in demonstrating the mucosal pattern. (*B*) Deep inspiration makes the cap available to compression and the mucosal pattern is identified. An ulcer is clearly seen in the mid cap.

1*A*; and 2*A*) and therefore inaccessible to compression, or when it is directed posteriorly and concealed by the overlapping barium filled antrum (Fig. 3*A*; 4*A*; 5*A*; and 6*A*).[6–9,11]

In an attempt to demonstrate in these cases the mucosal pattern of the cap in the prone position, we have taken advantage of the fact that the stomach and duodenum are significantly displaced inferiorly and slightly anteriorly in deep inspiration. During this process, the cap, which is situated

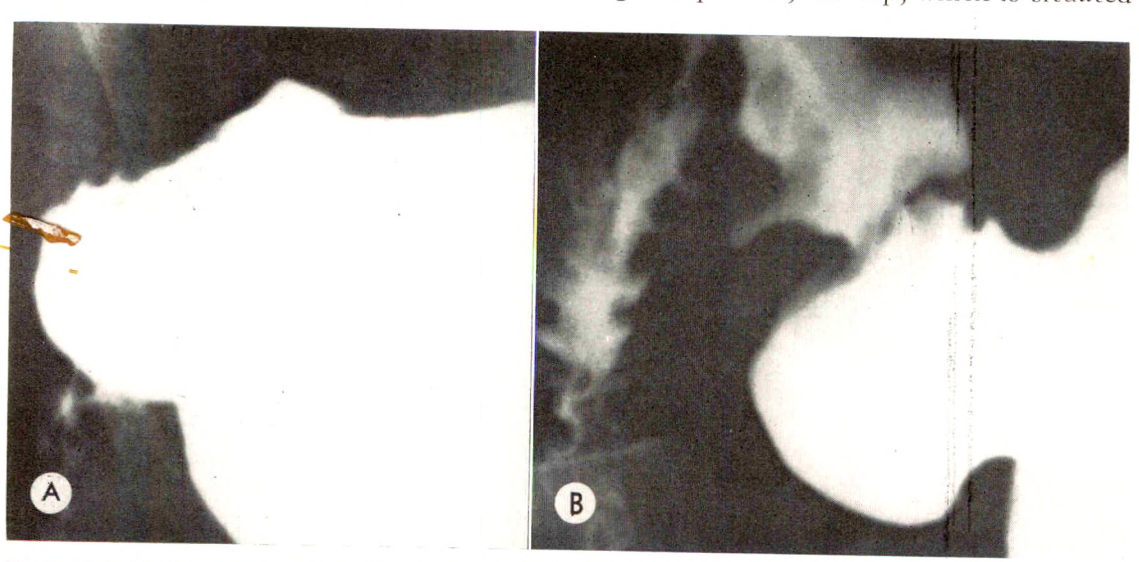


FIG. 3. (*A*) In the prone position, the duodenal cap is obscured by the overlying barium filled antrum and cannot be compressed. (*B*) Deep inspiration displaces the antrum inferiorly and slightly anteriorly, more than it displaces the duodenal cap, thus exposing the latter. The cap is also made available to compression and its configuration and mucosal pattern are identified.

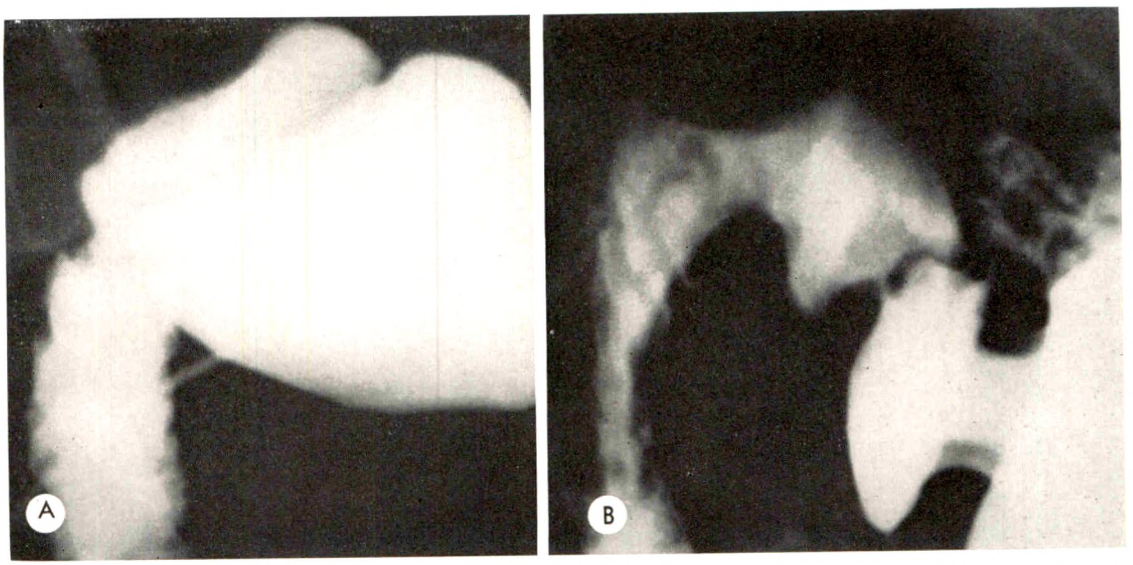


Fig. 4. (*A*) In the prone position, the duodenal cap is obscured by the overlying barium filled antrum and cannot be compressed. (*B*) Deep inspiration displaces the antrum inferiorly and slightly anteriorly, more than it displaces the duodenal cap, thus exposing the latter. The cap is also made available to compression and its configuration and mucosal pattern are identified.

high under the ribs, becomes available to compression by the pneumatic paddle (Fig. 1*B*; and 2*B*). Furthermore, deep inspiration effectively separates the cap from the antrum in cases where it is obscured by the latter, at the same time making the cap

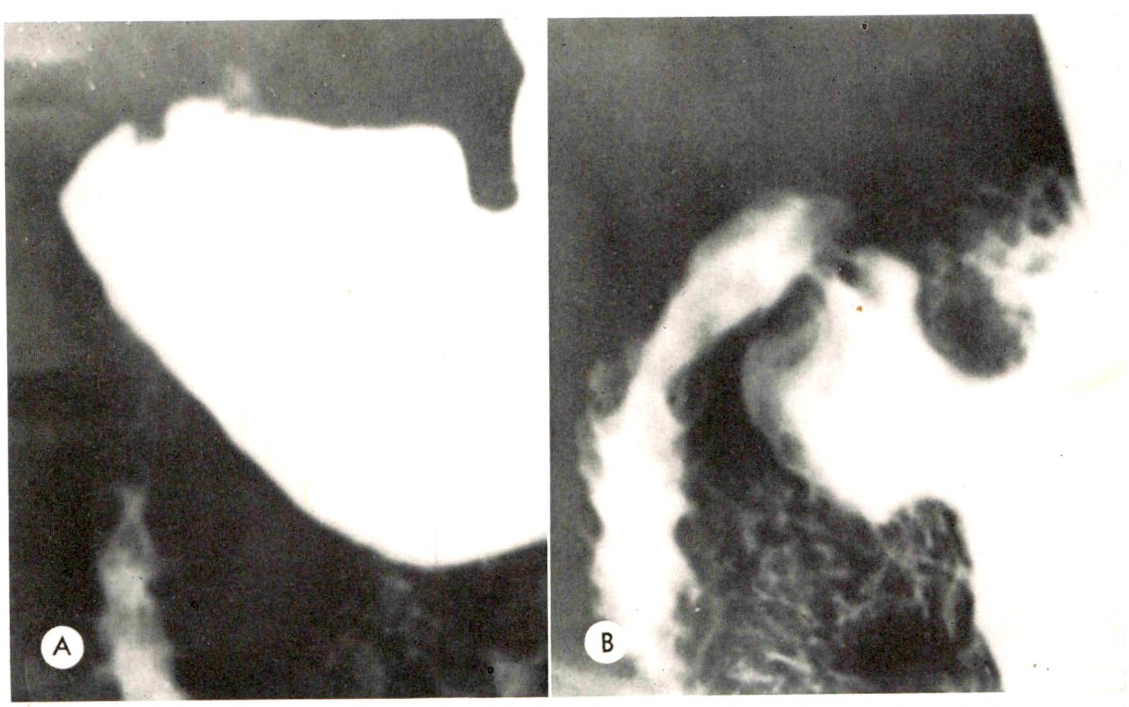


Fig. 5. (*A*) In the prone position, the duodenal cap is obscured by the overlying barium filled antrum and cannot be compressed. (*B*) Deep inspiration displaces the antrum inferiorly and slightly anteriorly, more than it displaces the duodenal cap, thus exposing the latter. The cap is also made available to compression and its configuration and mucosal pattern are identified.

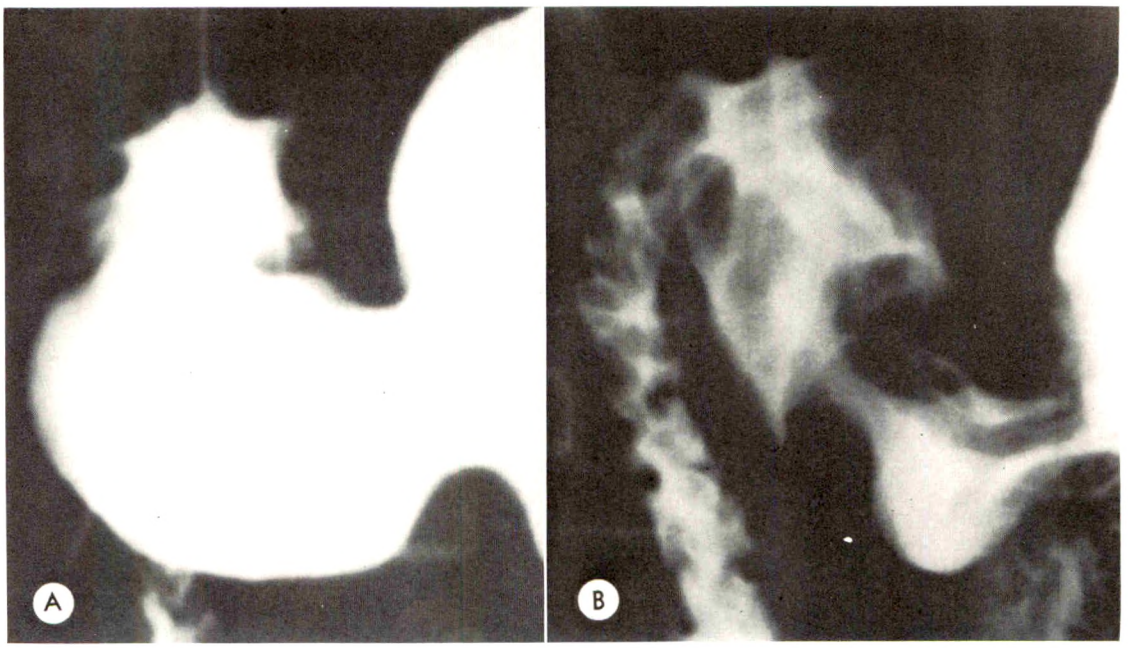


FIG. 6. (*A*) In the prone position, the duodenal cap is obscured by the overlying barium filled antrum and cannot be compressed. (*B*) Deep inspiration displaces the antrum inferiorly and slightly anteriorly, more than it displaces the duodenal cap, thus exposing the latter. The cap is also made available to compression and its configuration and mucosal pattern are identified.

available to compression (Fig. 3*B*; 4*B*; 5*B*; and 6*B*).

A lesser problem is the case of the air filled antrum and the duodenal cap overlapping each other in the supine right anterior oblique position. In such cases,

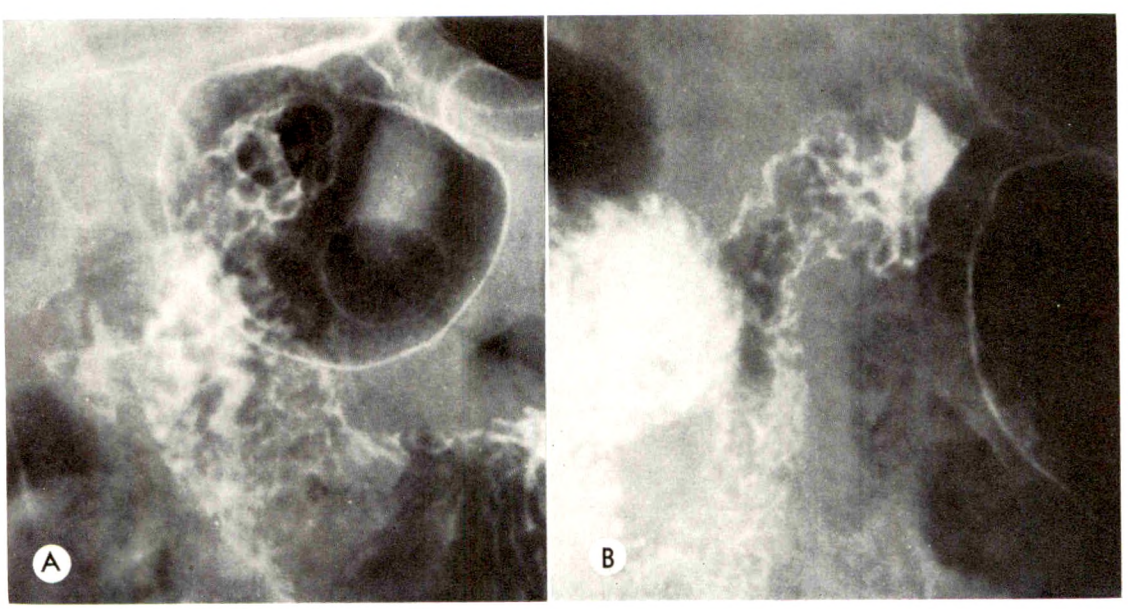


FIG. 7. (*A*) In the supine right anterior oblique position, the duodenal cap is seen through the air filled antrum. (*B*) A clearer visualization of the duodenal cap is obtained in deep inspiration when the antrum is displaced inferiorly and anteriorly.

similarly, deep inspiration often separates these 2 structures and a clearer visualization is thus obtained (Fig. 7, *A* and *B*).

TECHNIQUE

The patient assumes the prone oblique or lateral position and the pneumatic compression paddle is placed at a distance caudad to the cap. Following deep inspiration, the cap comes to lie over the center of the paddle. The patient is then asked to hold his breath in deep inspiration, while the pneumatic paddle is being inflated and the required degree of compression obtained for a satisfactory demonstration of the mucosal pattern.

SUMMARY

A technique is described to demonstrate, in the prone position, the mucosal pattern of the duodenal cap which is either situated high under the ribs or is obscured by the overlying barium filled antrum.

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ULCERS IN THE DESCENDING DUODENUM*

POSTBULBAR ULCERS

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ULCERS in the descending portion of the duodenum can be easily missed in upper gastrointestinal series unless careful attention is paid to this area.

Our experience with ulcers in this location was reviewed for a period of 12 months, and the clinical and roentgenographic findings are described.

An interesting anatomic finding in patients with postbulbar ulcer disease was noted and is reported.

MATERIAL AND METHOD

A total of 4,669 upper gastrointestinal series was performed during a 12 month period and the incidence of duodenal ulcers and postbulbar ulcers during this period was obtained. Routine upper gastrointestinal examinations were done and whenever a questionable stricture or ulceration was noted in the postbulbar area, hypotonic duodenography was added if possible.[3]

Initially, hypotonic duodenography was performed by the method of Martel *et al.*[8] Later, however, this was modified, and Pro-Banthine* was given intravenously instead of intramuscularly. This modification was instituted in an effort to obtain better control of the degree of filling of the duodenum during the examination. Fifteen to 30 mg. was given intravenously when it was fluoroscopically determined that the duodenum was filled with barium. The relaxation of the duodenum was almost immediate and the possibility of the duodenum emptying while waiting for the intramuscular dose to take effect was obviated. Intravenous Pro-Banthine is used routinely by the gastroenterologists at our institution whenever hypotonicity is needed at endoscopy, and they report no significant complications with its use. We have had no serious complications since the institution of this modification in hypotonic duodenography, and the side effects (dryness, blurring of vision, etc.) have been no different than when Pro-Banthine was used intramuscularly. The side effects do, however, wear off more quickly and commonly small bowel motility returns prior to the end of the examinations. Obviously, the examination was not performed in patients with contraindications to the use of Pro-Banthine, *i.e.*, cardiac arrhythmias, glaucoma or urinary retention usually secondary to prostatic enlargement. In 1 patient hypotonic duodenography was performed after placing a tube in the duodenum.

ANATOMY

The first portion of the duodenum normally is directed slightly upward and backward and then bends downwards. The duodenal bulb comprises, on an average, $\frac{5}{8}$ of the first portion of the duodenum,[1] although it can comprise from $\frac{1}{5}$ to $\frac{5}{6}$ of the entire extent of the first portion of the duodenum. By definition, any ulcer beyond the apex of the duodenal bulb should be considered "post-bulbar." However, since the apex cannot be accurately located in patients with inflammatory changes in this area, we have only considered in this study ulcers in the descending duodenum as postbulbar ulcers.

* Propantheline Bromide (Searle & Co.).

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RESULTS

The results are summarized in Table 1. Our incidence of 3.6 per cent of duodenal ulcers being postbulbar in location correlates with previous reports of approximately 5 per cent of roentgenologically demonstrated ulcers.[1,7] Reports on surgical material, on the other hand, suggest that the actual incidence may be higher than 5 per cent if the posterior wall of the duodenum is carefully examined.[1] Autopsy series have reported an even higher incidence, ranging from 5 to 20 per cent.[1]

Our ratio of 17:1 male to female (Fig. 1; and 2, *A* and *B*) correlates with that described by Cooke and Hutton.[5] This, however, is in sharp contrast to the great number of females described by Bilbao *et al.*[3]

The average age of our patients with a postbulbar ulcer was 49 years as compared with 39.8 years for those with duodenal bulb ulcers. This correlates well with what has been reported previously.[3,5]

CLINICAL FINDINGS

Incidence. The significance of the higher incidence of postbulbar ulcers in males

TABLE I

SUMMARY OF CLINICAL DATA

Total number of upper gastrointestinal series: 4,669	
Females	2,021 (43%)
Males	2,448 (57%)
Total number of patients with evidence of duodenal ulcer disease: 497 (11%)	
Number of patients with postbulbar ulcers: 18 (3.6% of ulcers)	
Sex incidence in patients with duodenal ulcer disease:	
Males	76%
Females	24%
Sex incidence in patients with postbulbar ulcers:	
Males	94% (17)
Females	6% (1)
Average age of patients with duodenal ulcer disease:	
Total patients	39.8 years of age (average)
Males	38.8 years of age
Females	42.8 years of age
Average age of patients with postbulbar ulcers: 49 years of age	

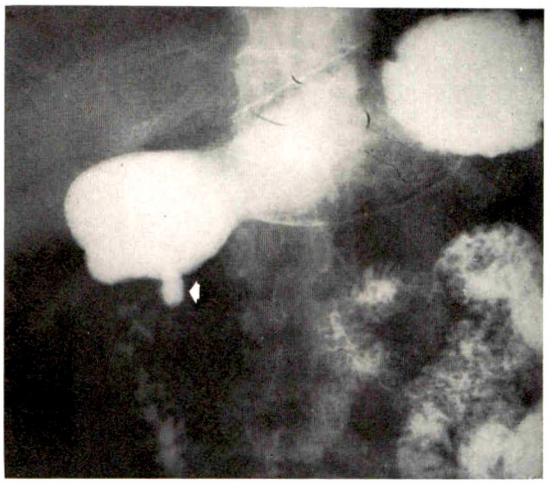


FIG. 1. G.G., a 66 year old male with a 10 year history of ulcer symptoms including one previous upper gastrointestinal bleeding episode. Ulcer crater (arrow) well seen in the proximal descending duodenum.

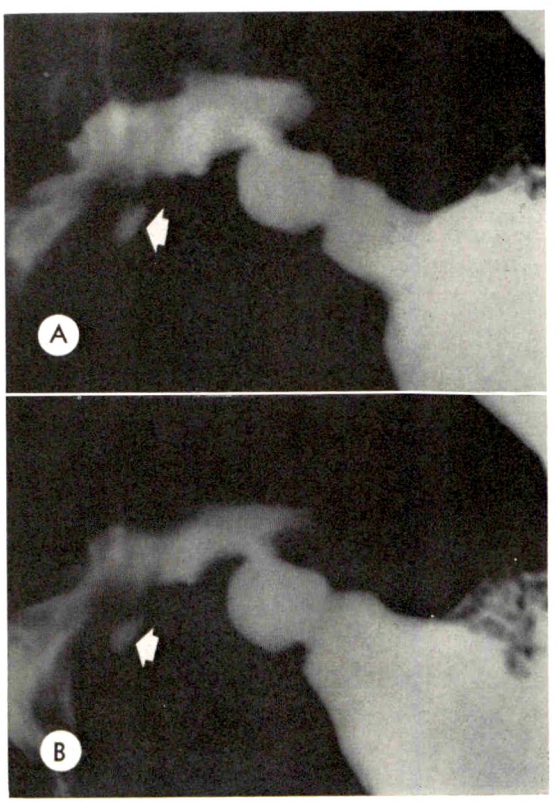


FIG. 2. A.R., a 44 year old female with a 5 year history of ulcer symptoms.

(*A* and *B*) This was the only woman with a postbulbar ulcer (arrows) in our series. She also represented the only patient in our series that did not have a "posterior bulb."

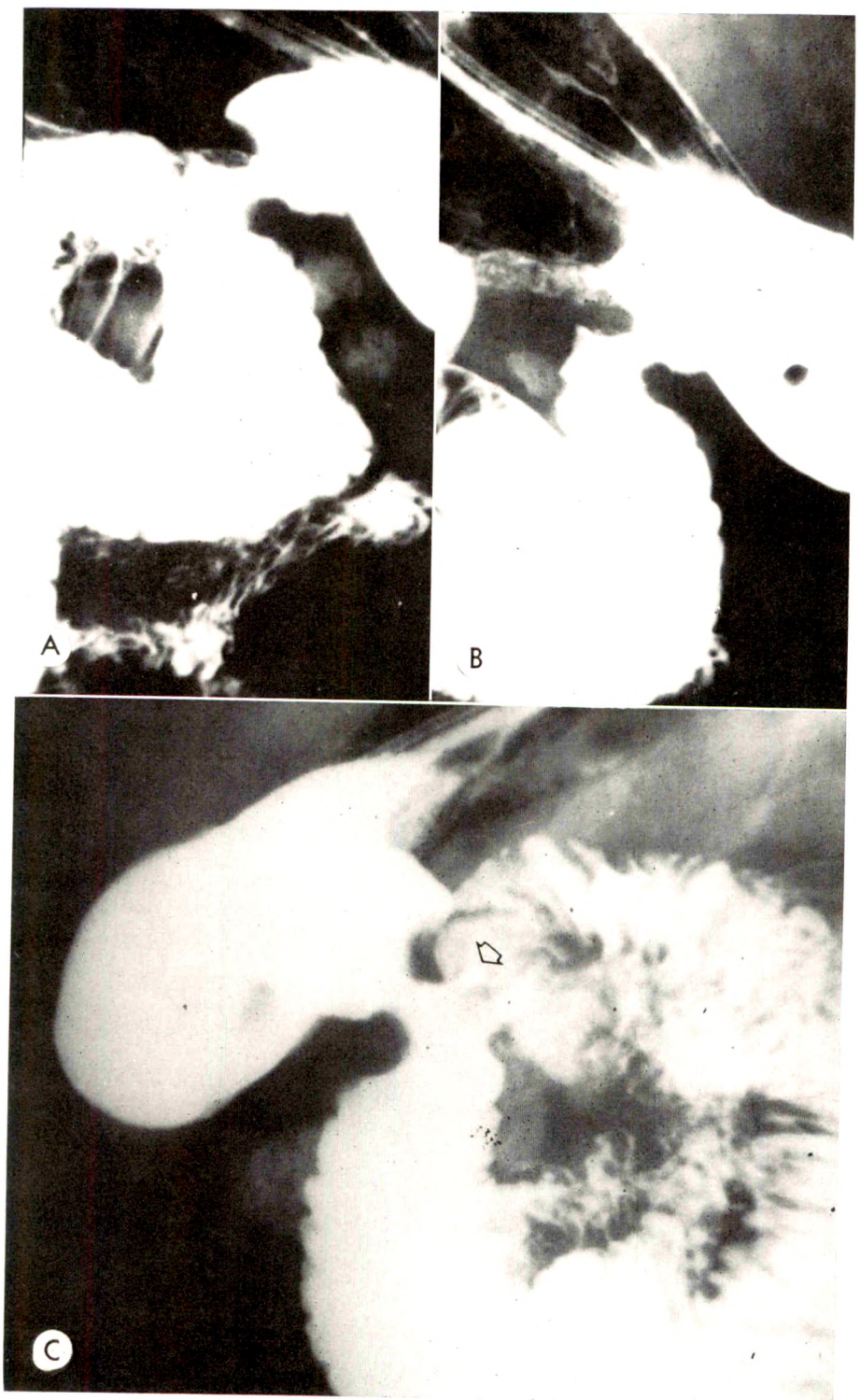


FIG. 3. B.S., a 69 year old male with a 3 year history of ulcer symptoms.
(*A–C*) In this hospitalization the patient presented with evidence of gastric outlet obstruction. At surgery prominent fibrosis and a duodenal stricture were present, with an active postbulbar ulceration (arrow).

(94 per cent) in our series is not known. It is conceivable that it is related to patient selection, but that does not seem likely since our population subjected to upper gastrointestinal examination was 57 per cent males and 43 per cent females. Admittedly, ours is a relatively small series including a total of only 18 patients with postbulbar ulcers.

The high average age of our patients correlates well with previous studies[3,5] and is probably related, at least in part, to delay in diagnosis. Symptoms have been present in our patients for an average of 9 years prior to the diagnosis of postbulbar ulcer.

Signs and Symptoms. Pain was by far the most prominent symptom in our patients. The pain was usually located in the epigastrium or right upper quadrant and was usually relieved by antacids. Seventy-two per cent reported awakening with pain 2 to 3 hours after retiring at night. Although vomiting did occur in a few patients, it was not a prominent feature. Weight loss also occurred, but it certainly was not a significant finding.

A high incidence of gastrointestinal hemorrhage in patients with postbulbar ulcers has been previously reported.[1] Seven (39 per cent) of our patients reported one or more episodes of gastrointestinal hemorrhage. This is in contrast to the 19.1 per cent incidence of bleeding in bulbar ulcers.[7]

Gastric Acid Secretion. Five of our patients had gastric secretory studies consisting of a 1 hour basal acid collection followed by a peak histamine stimulation. The basal acid output (BAO) ranged from 0 to 23.0 mEq./hr. (mean 5.84) and the peak acid output (PAO) ranged from 32.4 to 74.9 mEq./hr. (mean 54.3). The BAO is certainly within the range considered compatible with bulbar ulcers, and therefore a differential diagnosis between bulbar ulcers and postbulbar ulcers cannot be made on the basis of the basal secretion. The PAO, on the other hand, is quite elevated (normal mean = 24.5 mEq./hr.), but some individuals with bulbar ulcers also have peak secretory responses that are just as elevated. Because of this we hesitate to attach any etiologic significance to the elevated peak acid secretory response.



Fig. 4. J.P., a 39 year old male with a 6 year history of ulcer symptoms. Ulcer (arrow) is present along the medial wall of the descending duodenum in the prevaterian portion.

Roentgenographic Findings

The characteristic feature of postbulbar ulcers is a stricture-like narrowing of the descending duodenum (Fig. 3, *A–C*) with or without an associated ulcer niche being present. In general the area of narrowing is short, and there is usually some distortion of the mucosal pattern of the descending duodenum and possibly the duodenal bulb. None of our cases, however, had an associated ulcer crater noted in the duodenal bulb. The narrowing could be due to spasm

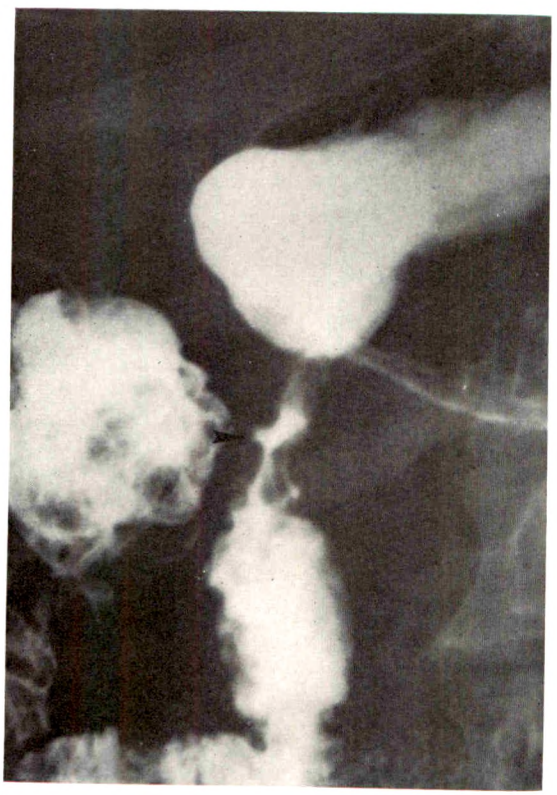


FIG. 5. E.O., a 46 year old male with a 2 week history of epigastric pain. An upper gastrointestinal series revealed a persistent narrowing in the proximal part of the descending duodenum.

A hypotonic examination was performed following the routine examination (reason for barium in the colon) and an ulcer crater (arrow) was noted in the lateral wall of the prevaterian portion of the duodenum.

but it is usually due to prominent fibrosis[3] present in these patients, most likely secondary to the long standing inflammatory changes. The ulcer niche is usually identified at the time of hypotonic duodenography. The ulcers and strictures are always located in the prevaterian portion of the descending duodenum, either in the medial (Fig. 4) or lateral walls (Fig. 5). Ulcers beyond the level of the ampulla of Vater are uncommon and, if present, a hypersecretory state such as Zollinger-Ellison syndrome should be suspected.

In reviewing our cases we noted that one of the reasons postbulbar ulcers were so difficult to identify was that the antrum of the stomach and the duodenal bulb overlapped the area in question (Fig. 6, *A–C*). The duodenal bulb was directed posteriorly in 17 of our 18 cases, making the delineation of the postbulbar area very difficult unless lateral projections were obtained or the patient was given effervescent substances* allowing the postbulbar area to be visualized through the air filled antrum and bulb. The duodenal bulb was even directed in a leftward direction in some cases (Fig. 7, *A* and *B*).

A "posterior bulb" is a normal variation in the anatomy of the duodenum and is more commonly seen in hypersthenic individuals. In reviewing our cases we found this variation in approximately 10 per cent of all our patients.

The reason for the correlation between postbulbar ulcers and posterior bulbs is uncertain; however, we have considered two possibilities. One is that the prominent fibrosis associated with these cases[4,9,10] involves the duodenal bulb, so that it becomes fixed in a posterior position. Therefore, when the stomach is distended with barium, it projects anterior to the bulb and postbulbar area. The other possibility is that patients with the "posterior bulb" variation are predisposed to postbulbar ulcers. Interestingly, Barton and Cockshott[2] also associated a high incidence of postbulbar ulcers with the increased incidence of redundant duodenums in their series, so that an anatomic predisposition is a possibility even though we do not know the reason. The association of this anatomic variation in 17 of our 18 patients with postbulbar ulcers seems more than coincidental and should lead us to examine the postbulbar area more carefully in patients with "posterior bulbs."

SUMMARY

The clinical and roentgenographic findings in patients with postbulbar ulcers are reviewed.

The typical patient is a male of approxi-

* Seidlitz Powder (Garfield & Co.) or Unik-Zoru Tablets (Unik Medical Labs. Inc.).

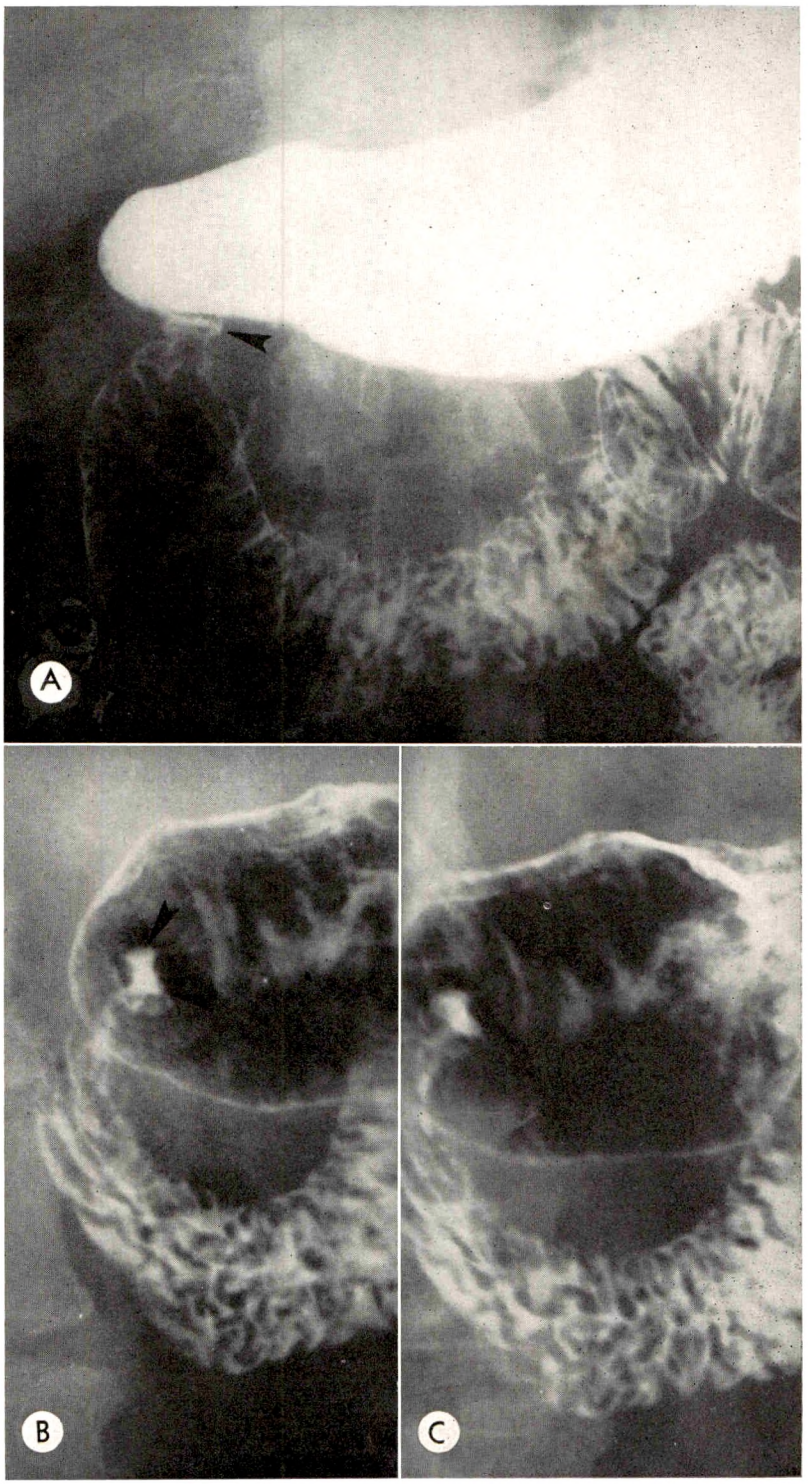


Fig. 6. W.J., a 54 year old male with a 19 year history of ulcer symptoms.
(*A*) In the prone position the postbulbar area is covered by barium in the antrum of the stomach. (*B* and *C*) The ulcer crater (arrowheads) is well seen in this steep left posterior oblique position.

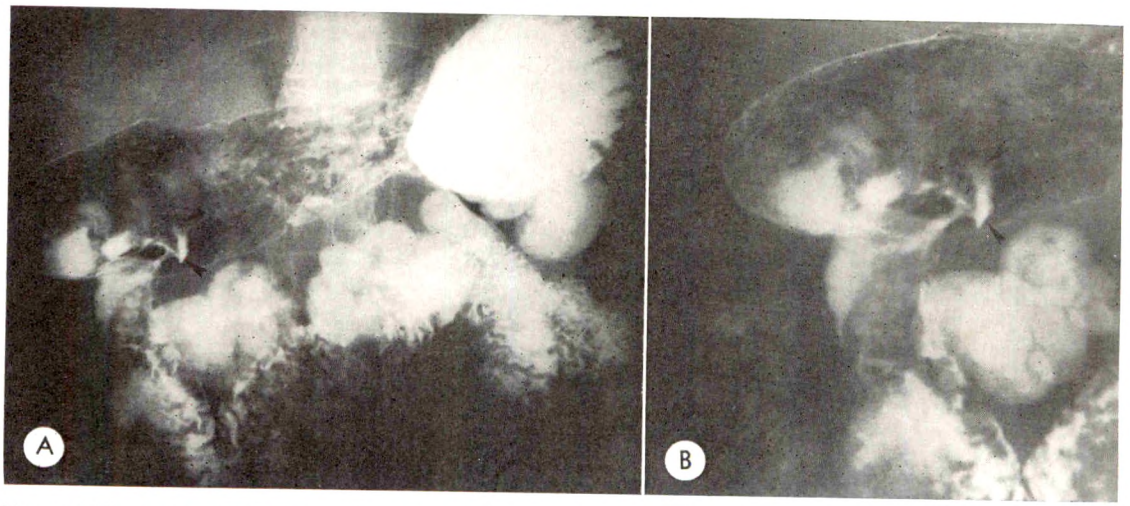


Fig. 7. H.P., a 55 year old male with a 24 year history of ulcer symptoms.
(*A* and *B*) The duodenal bulb is actually directed towards the left. The prominent stricture and ulcer crater in the proximal portion of the descending duodenum are well demonstrated and were confirmed at surgery.

mately 50 years of age with several years of ulcer symptoms.

A stricture with an associated ulcer niche in the descending duodenum is the classic roentgen finding.

The association of "posterior bulbs" in patients with postbulbar ulcers is emphasized, since this anatomic appearance makes the examination of the postbulbar area more difficult.

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LIPOMATOSIS OF THE ILEOCECAL VALVE*

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LIPOMATOSIS of the ileocecal valve is characterized by enlargement of the valve due to submucosal infiltration with fat. Lack of a distinct capsule differentiates the fat in this condition from a true lipoma arising from the lips of the valve. A number of synonyms have been applied including hypertrophy of the ileocecal valve, lipohyperplasia, fatty degeneration and the ileocecal valve syndrome. In most cases the ileocecal area is otherwise normal, although lipomatosis of the valve is a regular feature of Crohn's disease of the terminal ileum.[4]

Judging how big a valve must be, before it qualifies as being enlarged, frequently presents a problem. Rigid measurements are not satisfactory because of differences in magnification depending on the roentgenographic technique. Lasser and Rigler[8] measured normal valves and determined that the range of normal for either lip is up to 1.5 cm. in thickness. Hinkel[6] suggested that 4 cm. is the upper limit of normal for the valve including both lips.

Although lipomatosis of the ileocecal valve may produce symptoms which can be alleviated by resection of the valve, it is often asymptomatic and operation for an enlarged valve alone must be avoided. Therefore, accurate differentiation between lipomatosis and carcinoma or lymphoma of the valve is mandatory in order to avoid unnecessary surgery. Careful radiologic studies with appropriate compression roentgenograms usually allow an accurate distinction to be made. However, we have recently seen 2 patients with enlarged valves in whom we were unable to exclude the presence of a significant lesion. One patient was an elderly woman with lipomatosis of the valve that appeared nodular in shape in certain projections of the barium enema studies. The other was a young woman with granulomatous ileocolitis and concomitant lipomatosis of the ileocecal valve, presenting roentgenologically as a grape-like cluster of tumors arising from the valve.

REPORT OF CASES

CASE I. The patient was a 70 year old woman who had no symptoms. She was referred for a follow-up barium enema examination because of the previous detection of 2 villous adenomas. The first lesion was removed from the descending colon by a partial colectomy 17 years earlier. A second polyp was removed per rectum 1 year before. Neither tumor showed evidence of malignancy. Physical examination and laboratory studies were normal. Barium enema examinations were performed twice, 6 weeks apart, with no change in the findings. The lips of the ileocecal valve were thickened (Fig. 1, *A–C*). Both oblique projections showed definite nodularity of the valve, although the surface was smooth. The irregularity of the valve was not present on a barium enema examination done 1 year earlier, although similar projections were not available on the previous study.

Because of the history of previous villous adenomas of the colon and because of the presence of nodularity of the enlarged valve (a sign usually interpreted as indicating the presence of a tumor), a laparotomy was performed to rule out an adenoma or carcinoma of the valve. At operation the cecum was opened and a symmetrically enlarged ileocecal valve was identified (Fig. 2). A biopsy showed fatty infiltration of the submucosa. An incidental appendectomy was performed. The postoperative course was uneventful.

CASE II. The patient was a 26 year old woman with the chief complaint of diarrhea and abdominal pain of 2 months' duration. Although she had noted loose stools during the past 6 years, the diarrhea recently became more severe and was associated with crampy abdominal pain. Physical examination showed tenderness in the right lower quadrant of the

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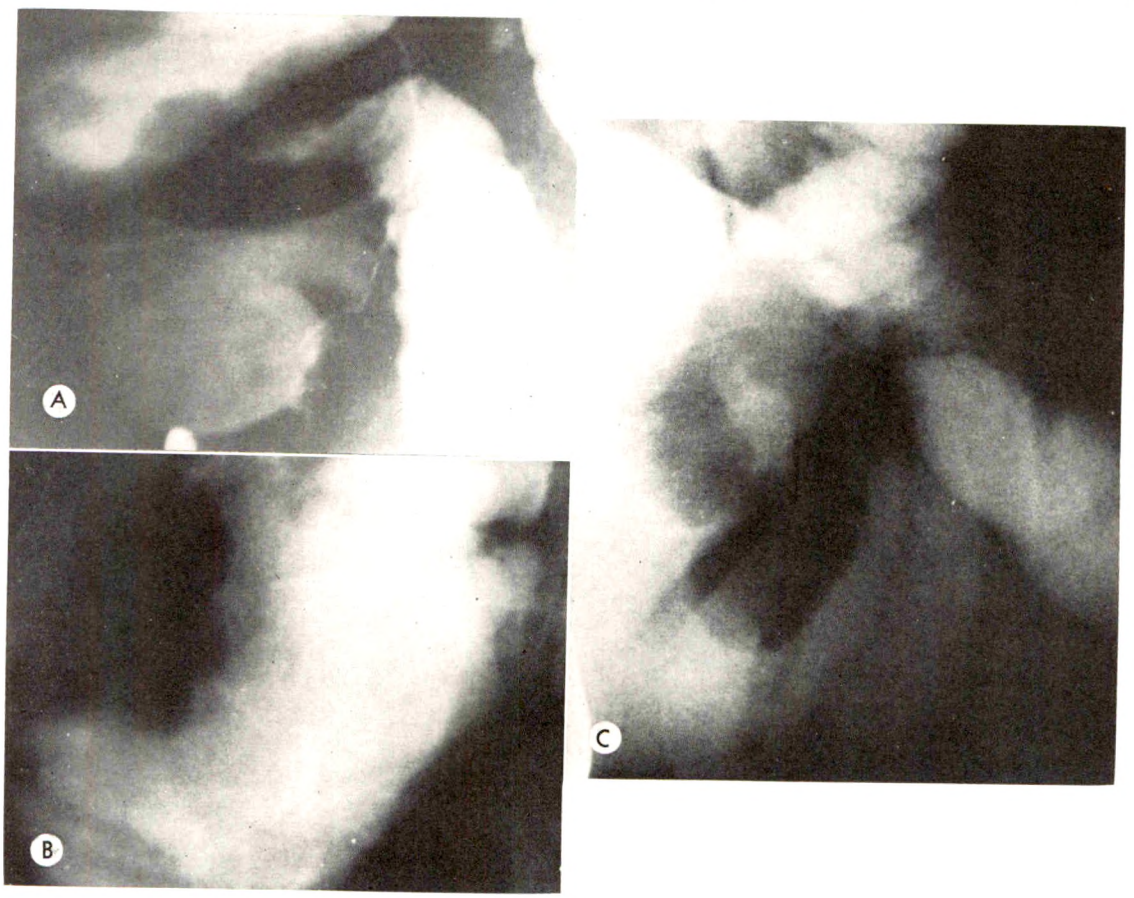


FIG. 1. Case 1. (*A*) Supine, (*B*) left posterior oblique, and (*C*) right posterior oblique roentgenograms of the ileocecal valve made during barium enema examination. Note the smooth nodularity of the valve, particularly in *B* and *C*.

abdomen. Laboratory findings included hemoglobin, 12 gm./100 ml.; hematocrit, 37 per cent; total proteins, 6.7 mg./100 ml.

Colonoscopy to the cecum, performed by Peter M. Loeb, M.D., showed normal mucosa as far as the splenic flexure, where a few round, discrete superficial ulcers were noted. Numerous similar ulcers were present in the transverse colon. Deep longitudinal ulcers were present in the ascending colon. Multiple, distinct polypoid lesions covered with erythematous mucosa were detected attached to the ileocecal valve. The valve itself appeared to be symmetric and non-inflamed.

Barium enema examination showed marked ulceration of the cecum and ascending colon. There was slight loss of haustration and minimal ulceration of the transverse colon. The mucosa of the last 12 cm. of the ileum was edematous. At least 5 nodules were present attached to the ileocecal valve. These intermittently prolapsed backward into the ileum (Fig. 3, *A–D*). Surgery was undertaken because of intractability of symptoms despite medical therapy, and because of the uncertain nature of the polypoid lesions.

At operation the distal ileum, cecum and ascending colon were thickened and inflamed. Numerous enlarged lymph nodes were present in the mesentery. An ileo-colectomy and transverse ileo-colostomy were performed.

Pathologic examination showed marked panenteritis involving the cecum, ascending colon and terminal ileum with deep ulcerations and diffuse chronic inflammation with numerous granulomata in the submucosa. Several distinct nodules arose from the ileocecal valve (Fig. 4).

Histologically these were composed of unencapsulated fat completely covered with mucosa (Fig. 5, *A* and *B*). The final diagnosis

was Crohn's ileo-colitis with lipomatosis of the ileocecal valve. Whether or not the lipomatosis was caused by the Crohn's disease or was only coincidental remains uncertain.

DISCUSSION

Lasser and Rigler[8] proposed an etiologic classification of conditions producing enlargement of the ileocecal valve. This includes edema (idiopathic and post-traumatic), submucosal fat accumulation, herniation of ileal mucosa, tumor (benign and malignant) and inflammatory lesions involving the valve. The cause of edema is obscure, although occasionally it is due to intermittent intussusception. Prolapse of ileal mucosa similar to herniation of gastric mucosa through the pylorus has been recognized at surgery and at autopsy.[5] Radiologically in these cases, a mass can be identified protruding through the lips of the ileocecal valve. When tumors involve the valve, a distinct mass may be identified attached to an otherwise normal valve. Nodularity and irregularity of the valve are usually interpreted as an indication that a tumor is present. The most common tumor is a lipoma, which differs from lipomatosis of the valve because it is well encapsulated. Rarely, a metastasis to the valve occurs. There is difficulty in distinguishing carcinoma of the valve from carcinoma arising in the cecum or ascending colon. In one series of 91 cases of carcinoma of the cecum and ascending colon, 15 per cent of the tumors began at the valve.[1] Lymphosarcoma involving the valve is not uncommon. The lesion usually involves the terminal ileum also, whereas ileal extension is rare in cases of carcinoma of the ileocecal valve.[9]

Lipomatosis is uncommon before the age of 40 and is usually seen between 50 and 70 years of age. The mean age in one series was 42.5 years.[2] Females predominate 2 to 1. Kelby[7] found that 50 per cent of patients with lipomatosis have clinical manifestations, whereas 82 per cent of Lasser and Rigler's series had abdominal complaints.[8] Castleman suggests that the symptoms



Fig. 2. Case 1. Photograph of the ileocecal valve taken at operation. The cecum has been opened showing the symmetrically enlarged valve.

may be due to chronic intussusception, the enlarged valve producing intermittent colocolic intussusception.[3] Lasser and Rigler[8] propose that a cycle is established in which the enlarged valve tends to cause intussusception and each intussusception causes further enlargement of the valve. Another possibility is that the enlarged valve produces partial obstruction at the ileocecal junction. Whether or not the patient's symptoms are actually related to the enlarged valve is difficult to determine. However, Lasser and Rigler[8] suggest that a definite syndrome exists consisting of bloating, right-sided abdominal pain and tenderness over the ileocecal valve.

In Case 1 presented here, surgery was advised because nodularity of the enlarged valve seen in certain projections of the barium enema examination raised the possibility that the enlargement was due to a

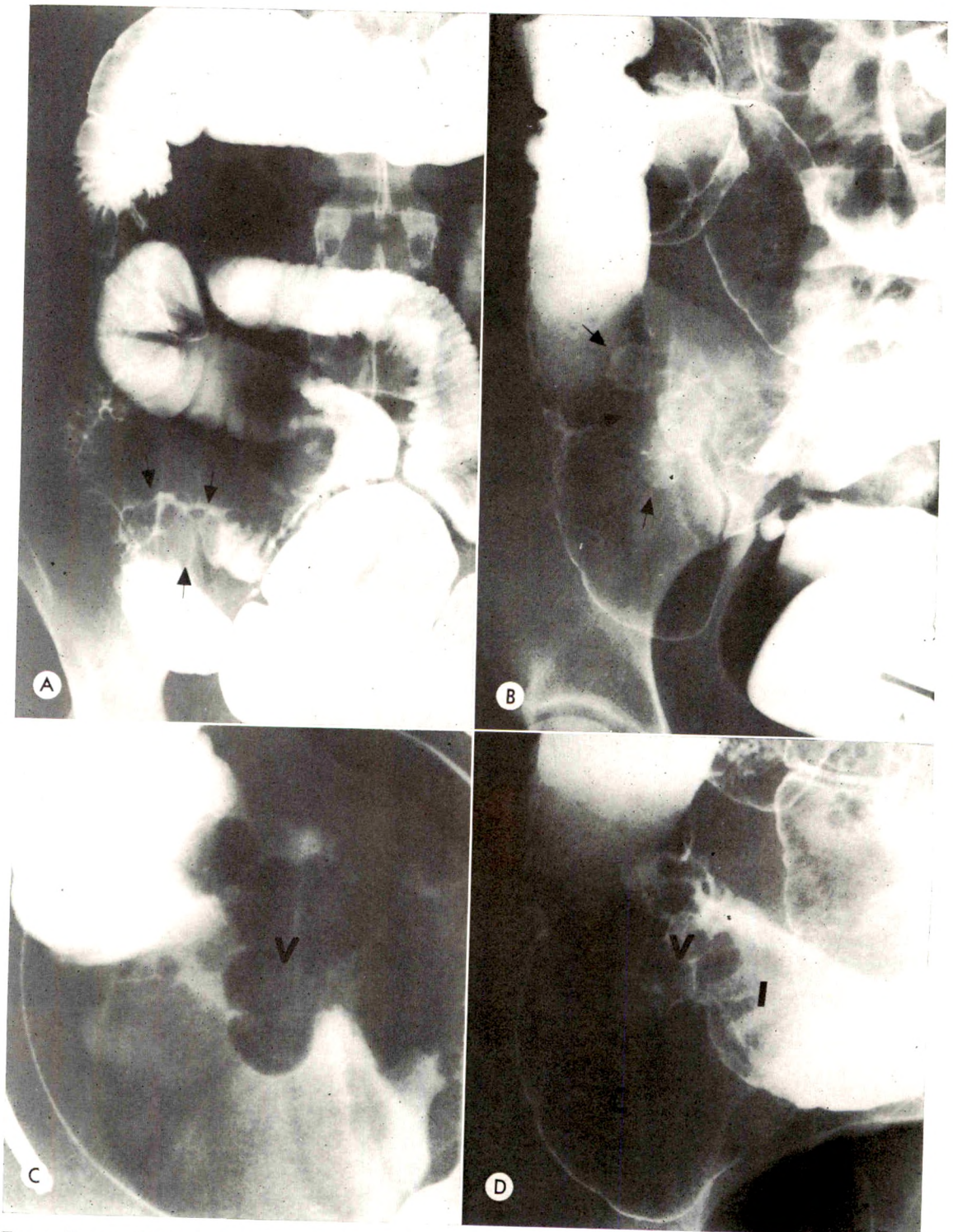


FIG. 3. Case II. Multiple roentgenograms made during barium enema and air contrast examinations. (*A*) Arrows show multiple polyps prolapsing backward through the ileocecal valve. (*B*) Arrows indicate grape-like cluster of polyps attached to the ileocecal valve. Note ulcerations in the ascending colon and edema of the terminal ileum. (*C*) Compression roentgenogram shows multiple polyps attached to the ileocecal valve (V). (*D*) Roentgenogram made during air contrast study shows the ileocecal valve (V). Cecum (C). Note edema of the terminal ileum (I).

tumor. However, since only lipomatosis of the valve was found, nodularity is not a reliable criterion on which to base the diagnosis of tumor. Lasser and Rigler[8] noted that there is a stellate appearance of enlarged valves when viewed *en face*. They suggest that this wrinkling of the overlying mucosa is evidence that the muscularis mucosae is intact and free to contract. Hence, the stellate configuration and its associated nodularity indicate that the lesion is benign rather than malignant. Furthermore, Boquist *et al.*,[2] studying gross specimens in 10 cases of lipomatosis, noted enlargement of the valve with a polypoid appearance in several cases. Histologically their cases showed diffuse fatty infiltration in the submucosa which often formed polypoid protrusions. Attention should be given to whether or not the surface of the valve is smooth or shaggy, rather than whether there is nodularity.

In Case 11 presented here, barium enema examination disclosed tumor masses in association with the inflammatory changes of granulomatous ileo-colitis. An appearance of multiple grape-like tumors has not been described before with lipomatosis or lipomas of the valve and this finding has not been reported associated with Crohn's ileocolitis. Hypertrophy of the lips of the valve is characteristic of Crohn's disease of the terminal ileum.[4] In fact, the presence of an enlarged valve may be used as an aid in the differentiation between Crohn's disease and ulcerative colitis. In ulcerative colitis the valve is atrophic and patulous. The thickened valve of Crohn's disease is smooth and symmetric without the grape-like protrusions noted in the present case. Multiple polypoid masses in association with inflammatory disease of the colon may be due to pseudo-polyposis, inflammatory polyps or colitis cystica profunda. However, in the present case the masses were due to lipomatosis of the valve. The relation of the lipomatosis to the concomitant ileo-colitis is uncertain and, while the 2 could be causally related, the association may be coincidental.



Fig. 4. Case 11. Photograph of the resected specimen. Ileum (I). Ileocecal valve (V). Cecum (C). Thin arrows indicate polypoid lesions attached to the ileocecal valve. Heavy arrows show ulceration in the terminal ileum.

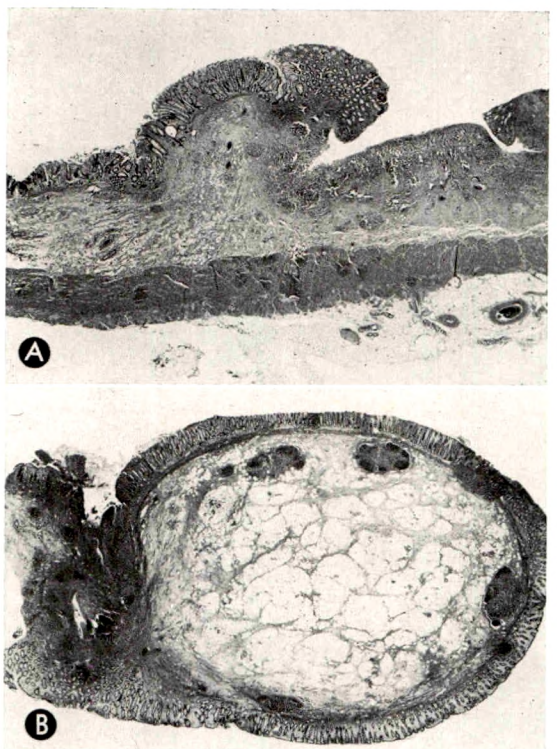


Fig. 5. Case 11. (*A*) Photomicrograph of the terminal ileum (H and E ×8). Note the deep ulceration. The submucosa is thickened and contains multiple small granulomata. (*B*) Photomicrograph of one of the multiple polyps attached to the ileocecal valve (H and E ×8). Normal mucosa completely covers unencapsulated fibroadipose tissue which is contiguous with the chronically inflamed submucosa.

SUMMARY

Two patients with lipomatous infiltration of the ileocecal valve are reported.

Surgery was undertaken in Case I when tumor was suspected because the valve was nodular in shape in certain projections of the barium enema examination. The fact that no tumor was found indicates that the nodularity of the valve is not a reliable sign of carcinoma. Greater importance should be given to whether the surface of the valve is smooth or irregular.

Case II had Crohn's ileo-colitis and grape-like masses attached to the ileocecal valve due to lipomatous infiltration of the valve. No similar case has been reported either with lipomatosis of the valve or associated with Crohn's ileo-colitis. The relationship, if any, between the ileo-colitis and the lipomatosis of the valve is obscure.

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RIGHT ILEOCOLITIS SECONDARY TO ANISAKIASIS*

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THICKENING of the bowel wall of the ascending colon or terminal ileum is a nonspecific roentgenologic change in many inflammatory processes. An accurate and specific history may suggest the differential preoperative diagnosis in most of these patients.

An unusual type of ileocolitis was observed recently in a young woman with a history of eating raw fish.

REPORT OF A CASE

A 32 year old Swedish woman was admitted to the Peter Bent Brigham Hospital with the complaint of right lower quadrant pain and fever of 5 days' duration. An episode of viral pneumonia had resolved completely 1 month prior to admission. There was no diarrhea or melena. Appendectomy had been performed in 1953. The patient had been in the United States for 5 years and had not recently traveled. She and her husband had the habit of eating raw fish.

Examination revealed a temperature of 101°F. There was tenderness and guarding in the right lower quadrant as well as a palpable mass. The right ovary was not palpable, but pelvic examination was otherwise negative.

Laboratory examination revealed a white blood cell count of 19,000/mm.^{3}, with a differential of 72 per cent neutrophils, 25 per cent lymphocytes, 2 per cent eosinophils, and 1 per cent basophils. Stool examination was not performed.

Plain film abdominal roentgenograms revealed thickening of the wall of the ascending colon (Fig. 1). Upper gastrointestinal and small bowel series as well as barium enema examination revealed the thickened wall of the ascending colon and showed involvement of the terminal ileum with this inflammatory process (Fig. 2, *A* and *B*).

Laparotomy was performed on the day following admission because of an acute abdomen. At surgery the impression was right-sided colitis of uncertain etiology; a right hemicolectomy was performed. The pathologic specimen, consisting of the ascending colon and terminal ileum, revealed an inflammatory process involving the submucosa, muscle and pericolic fat with necrotizing granulomata and prominent eosinophilic and fibroblastic infiltrate. There was marked thickening of the ileocecal valve. The wall of the colon and terminal ileum was edematous but the mucosa was intact. The *Anisakis* organism was identified in the histologic specimen.

DISCUSSION

Several cases of larval anisakiasis have been reported in Holland[5] and in Japan.[1,3,6,7] The disease has not been reported in the United States, but larval anisakid nematodes have been identified in market fish purchased in Connecticut, Maryland, and New York[2] The *Anisakis* parasite is an

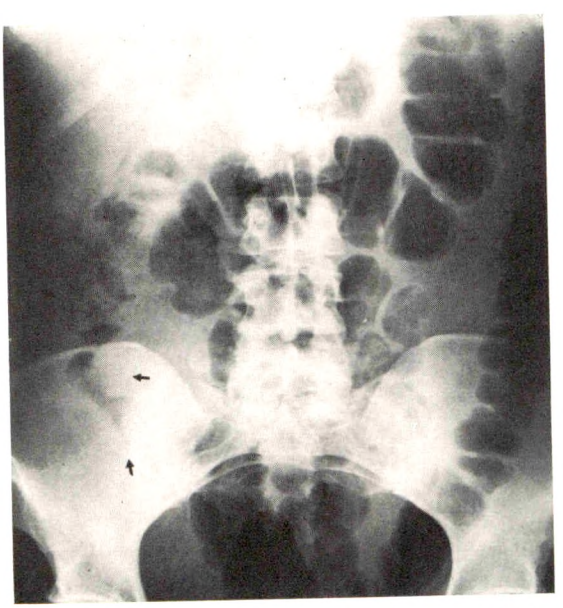


FIG. 1. Preliminary supine roentgenogram of the abdomen. The colon is clearly outlined with air in its entirety. The lumen of the cecum and ascending colon is narrowed by thickened bowel wall (arrows). An adjacent soft tissue mass is suggested.

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This work was supported in part by USPHS grant GM18674.

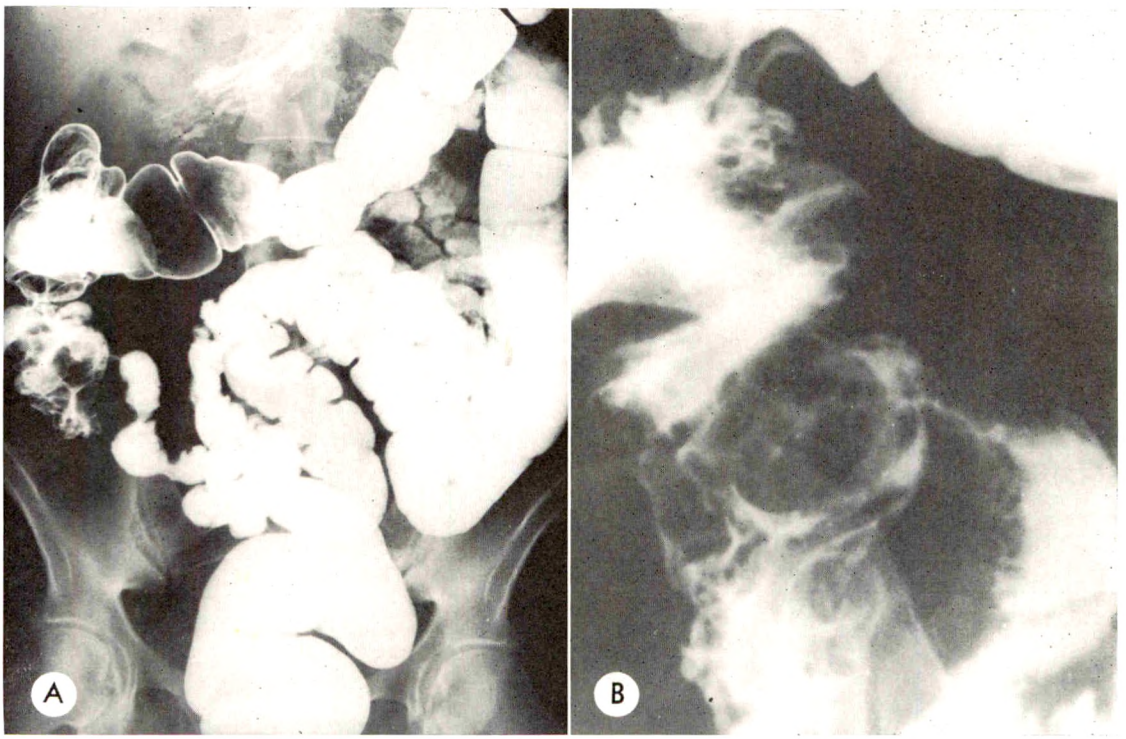


FIG. 2. (*A*) Prone frontal barium enema roentgenogram. The stomach and small bowel are opacified from a small bowel series performed immediately prior to the colon study. The cecum and ascending colon show limited distensibility because of marked bowel wall thickening. The ileocecal valve is markedly enlarged. The terminal ileum is drapped over an adjacent soft tissue mass. No ulcerations of the colonic or ileal mucosa are visible although the folds are thickened. (*B*) Detail fluoroscopic roentgenogram of the ileocecal area.

ascaris-like nematode.[4] The final hosts for this parasite are marine mammals, including whale, dolphins and sea lions. *Euhausiid* crustaceans prey upon the second stage larvae which have hatched in sea water. Various sea fish, including herring, cod, mackerel, bonito, pike and squid, consume the crustaceans with their larvae which are now in the third stage. The larvae are able to penetrate the alimentary tracts of the fish and then survive and develop in their body cavities and muscles.[4] Marine mammals then consume these fish with their larvae, thereby completing the life cycle. Man becomes infected with the larvae through ingestion of raw, slightly salted or vinegar-pickled fish.[7]

In humans the *Anisakis* larvae have mainly been found in the wall of the intestine (30 per cent) or stomach (65 per cent).[7] Clinically, there may be a long, nonspecific history of several months or an acute history of only 1 day. Intestinal cases usually have a shorter history than gastric cases. Stool examination for ova and parasites is negative, but tests for occult blood in gastric washings or stool are usually strongly positive. A mild eosinophilia is occasionally present, especially when the stomach is involved.

In the past, very few cases of anisakiasis have been diagnosed preoperatively. In a Japanese series of 92 cases,[7] gastric tumor or ulcer was the diagnosis in 16 per cent. Preoperative diagnoses have also included appendicitis, Crohn's disease, cholecystitis, diverticulitis, tuberculous peritonitis and other acute abdominal conditions.[7]

In the case of gastric involvement, live, moving *Anisakis* worms have been seen

directly penetrating the stomach wall on endoscopy.[4] Occasionally, the worm may be seen in the stomach on upper gastrointestinal series.[4] Intestinal cases are more difficult to diagnose and can only be suspected by history. If the diagnosis is definite, conservative treatment with streptomycin combined with erythromycin, antiphlogisca and instillation of isotonic glucose solution may be used.[4] When diagnosis is uncertain, laparotomy is necessary for removal of the lesion.

Pathologically,[1,3,5–7] the outstanding feature is massive eosinophilic infiltration associated with marked edema and, sometimes, fibrosis in the submucosa. The eosinophilic abscess or phlegmon in the submucosa harbors the *Anisakis* larvae and the mucosa usually remains intact. Involvement of the deeper layers suggests an allergic change.

The roentgenologic appearance of a markedly thickened bowel wall in our case reflects accurately the pathologic change, but the findings are nonspecific. Thickening of the bowel wall of the ascending colon or terminal ileum may be seen also in Crohn's disease, tuberculosis, amebiasis, ischemia and neoplasm. Thus, anisakiasis may be added to this list and should be suspected in any patient with the habit of eating raw, pickled or slightly salted fish.

SUMMARY

Roentgenologic and clinical signs of an inflammatory process in the bowel were the only apparent preoperative diagnostic clues in a young woman who also habitually ate raw fish. Laparotomy followed by hemicolectomy led to the histologic diagnosis of anisakiasis caused by an *Anisakis* parasite.

Although anisakiasis is not common in the United States, it should be suspected in patients who present with a markedly thickened bowel wall and a history of eating raw, pickled or salted fish.

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THE "HIDE-BOUND" SMALL BOWEL OF SCLERODERMA: CHARACTERISTIC MUCOSAL FOLD PATTERN*

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IN the past, dilatation has been regarded as the roentgenographic hallmark of scleroderma of the small bowel.[3] However, the differential diagnosis of dilated small bowel loops is quite extensive.

The purpose of this communication is to: (1) document the incidence of another previously reported but neglected roentgenographic sign; and (2) report the occurrence of transient intussusceptions, which we will show to be related to this sign.

The finding is a relative decrease in the distance separating the valvulae conniventes for a given degree of small bowel dilatation. Usually, the valvulae separate longitudinally when the bowel dilates in width, as seen in conditions such as sprue and small intestinal obstruction. However, this study will demonstrate that in a significant number of cases of scleroderma, the opposite occurs; *i.e.*, the valvulae tend to remain abnormally packed together despite bowel dilatation.

MATERIAL AND METHOD

The records of 42 consecutive patients with scleroderma seen at The New York Hospital over the past 5 years were reviewed. The diagnosis was established either by biopsy or clinical findings, such as characteristic changes in the skin. Small bowel series were available in 18. The character of the valvulae conniventes and their relationship to the dilated small bowel loops were tabulated for each patient. No attempt at precise measurement was made, since it is not clear what a normal minimum distance separating the valvulae would be in a dilated loop of bowel. Rather, the over-all roentgenologic appearance was used to judge whether or not the valvulae were abnormally close together for a given degree of bowel dilatation.

RESULTS

Of the 18 small bowel series reviewed, 11 (61 per cent) showed a strikingly narrowed intervalvular distance (Fig. 1). This was most marked in the jejunum and proximal ileum. While the duodenum was almost invariably dilated, it did not consistently demonstrate this altered mucosal fold pattern. Sharp angulation of dilated jejunal loops in which the folds presented the retracted appearance was also occasionally noted (Fig. 1). When barium fully opacified the dilated loops, the decreased intervalvular distance was manifested as a series of sharp "spines" projecting from the margin of the lumen (Fig. 2). The valvulae themselves were not widened or blunted and maintained their normal axis perpendicular to the lumen of the bowel. No nodularity or "thumbprint" type defects were noted.

In 3 cases (17 per cent), the "coiled-spring" pattern of intussusception was noted. This ranged from mild to marked findings with a definite prominent intraluminal defect further dilating the segment (Fig. 3).

DISCUSSION

While dilatation is characteristic of scleroderma of the small bowel, it is by no means specific. In the usual case of dilatation, there is some increase in the length as well as the width of the loop. Consequently, the mucosal folds tend to be separated in proportion to the degree of dilatation. This is true whether the dilata-

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tion is secondary to mechanical obstruction or to conditions characterized by atony or disordered peristalsis as in paralytic ileus or sprue. None of these, however, produces the tightly packed "accordion" appearance to the folds within dilated small bowel loops demonstrated by the cases of scleroderma. The changes are distinct from the "stacked coin" or "picket fence" appearance of mucosal folds in diffuse intramural bleeding. Further distinction from infiltrative diseases of the small bowel is easily established by the normal appearance of the valvulae themselves. They are not widened, blunted or altered in orientation.

The occurrence of this roentgenologic sign has been briefly noted by others,[3] but its incidence has not been previously documented, and it has therefore not received the attention it deserves. Since the finding occurs in over 60 per cent of the cases, it is a clinically useful sign in differentiating scleroderma from other diseases that cause



FIG. 1. Scleroderma of the small bowel. For the degree of dilatation, the mucosal folds are strikingly retracted closer together. Note that the valvulae conniventes themselves are not widened. Sharp angulation of loops is present in the left mid-abdomen.



FIG. 2. Scleroderma of the small bowel. When the dilated loops are filled with barium, the decreased intervalvular distance is evidenced as closely spaced "spines" projecting from the bowel margin.



FIG. 3. Scleroderma of the small bowel. Tightly packed jejunal folds in the left upper quadrant in the "coiled-spring" pattern of intussusception. There is an associated prominent intraluminal defect in the dilated portion of the bowel. The folds distally are also characteristically tightly packed.

small bowel dilatation. In fact, recognition of this characteristic feature may place the radiologist in a position to make the diagnosis in those rare cases where small intestinal involvement occurs prior to other more obvious signs of scleroderma.[4]

This is also the first report of the occurrence of intussusception of the small bowel in scleroderma. The disordered peristalsis occasioned by the sclerodermatous involvement is analogous to that in sprue, in which similar transient nonobstructive intussusceptions have been observed.[6] Identification of the intussusceptions themselves, being secondary to sclerodermatous involvement of the small bowel, is easily revealed by the presence of the characteristically retracted mucosal folds both proximal and distal to the lesion.

The known pathology of scleroderma provides an explanation for the narrowed intervalvular distances as well as the intussusceptions. It is well documented that when scleroderma involves the small bowel, there is replacement of both the inner circular and outer longitudinal layers of muscularis propria by fibrous tissue.[1,3] The fibrous replacement may not be symmetric. Restriction in a longitudinal direction could therefore be expected in those cases in which there is significant collagenous replacement of the longitudinal fibers of muscularis. Dilatation with a predominant increase in width but relative foreshortening would result in disproportionate apposition of the valvulae conniventes. This condition in the small bowel is analogous to the changes that occur in the soft tissues of the hands and face, in which there also is replacement of normal tissue by excess fibrosis, creating a "hide-bound" appearance. It is apparent that the intussusceptions merely represent one more point in the continuum of small bowel foreshortening. In other words, the bowel is so "hide-bound," that, particularly with disordered peristalsis, an actual transient telescoping of one segment of bowel into another may readily occur.

SUMMARY AND CONCLUSIONS

A strikingly narrowed intervalvular distance, disproportionate for the degree of dilatation, is distinctive of scleroderma of the small bowel. It occurs in over 60 per cent of the cases.

Transient, nonobstructive intussusceptions are reported for the first time, occurring in 17 per cent of the cases reviewed.

An explanation for these changes based on the histopathology of the disease is proposed.

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ANEURYSMAL LYMPHOMA OF THE SMALL INTESTINE*

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ANEURYSMAL lymphoma of the small intestine is an uncommon lesion. Fu and Perzin[9] found only 6 cases in a total of 38 lymphomas of the small intestine, seen in their institution, in a period of 25 years. Although aneurysmal lymphoma belongs to 1 of the 5 accepted roentgenographic patterns of lymphoma of the small intestine,[17] it definitely represents a separate entity, with its own clinical problems, unique pathophysiology and specific roentgen manifestations.

Two illustrative cases are reported, one with early and the other with advanced roentgen features.

REPORT OF CASES

Case I. A 57 year old white man entered the hospital complaining of lower abdominal cramping of 6 months' duration, weakness, and a 28 pound weight loss. The cramping would occur ½ to 1 hour after eating and would persist for 1 to 2 hours. It was localized inferior to the umbilicus.

He appeared elderly and emaciated. On rectal examination, a firm, bulging mass was felt against the anterior rectal wall. Stool guaiac was ++ for occult blood. The hemoglobin was 7.8 gm. per cent and the hematocrit 24 per cent.

The intravenous pyelogram showed the right ureter to be deviated laterally by a soft tissue mass measuring approximately 7×12 cm. The same mass caused an impression upon the roof of the urinary bladder. The barium enema examination demonstrated an extrinsically compressed sigmoid colon. The small bowel examination was the most revealing (Fig. 1*A*). A loop of proximal ileum measuring approximately 20 cm. in length was dilated and had a markedly distorted barium-filled lumen. The loop was located in the pelvic area, was fixed, had thick walls and accounted for the mass seen on the pyelogram and barium enema roentgenogram. Six and 24 hour delayed roentgenograms showed the retained barium in only this particular small intestinal loop.

At surgery, there was a mass involving the ileum and located in the pelvic area. The mass represented a segmental dilatation of the bowel with the loops proximal and distal to the mass having a normal appearance. The resected specimen was filled with barium and roentgenographed (Fig. 1*B*). The aneurysmal dilatation in the center of the small intestinal loop corresponded to the mass felt clinically, identified on the roentgenogram and seen in the patient's abdomen at surgery. The barium within the lumen of the aneurysmal dilatation had an irregular, ulcerated and penetrating pattern.

Upon opening, the specimen had the gross appearance of a necrotic tumor having normal appearing proximal and distal small intestinal loops attached to it. The microscopic examination revealed lymphosarcoma of the ileum.

Following surgery, the patient received cobalt 60 teletherapy to the lower abdomen with a total of 2,525 rads delivered in 33 days. He returned 4 years later with extensive abdominal spread of lymphoma.

Case II. A 76 year old cachectic white woman entered the hospital with a 3 month history of diarrhea, weakness, and 18 pounds weight loss. She was having 2–3 semiformed stools per day. Two months prior to admission the diagnoses of pernicious anemia and colitis were made in another hospital. She received 1 unit of blood during her hospitalization.

A lower abdominal mass was felt on physical examination. Laboratory data revealed: hemoglobin 8.5 gm. per cent, hematocrit 25.9 per cent, total protein 5.2 gm. per cent (normal 6.0–8.0 gm. per cent), albumin 2.2 gm. per cent (normal 3.5–5.0 gm. per cent). Stool guaiac + for occult blood.

The intravenous pyelogram showed a large mass in the pelvis extrinsically deforming the urinary bladder, but not the ureters. The barium enema examination was normal. On the

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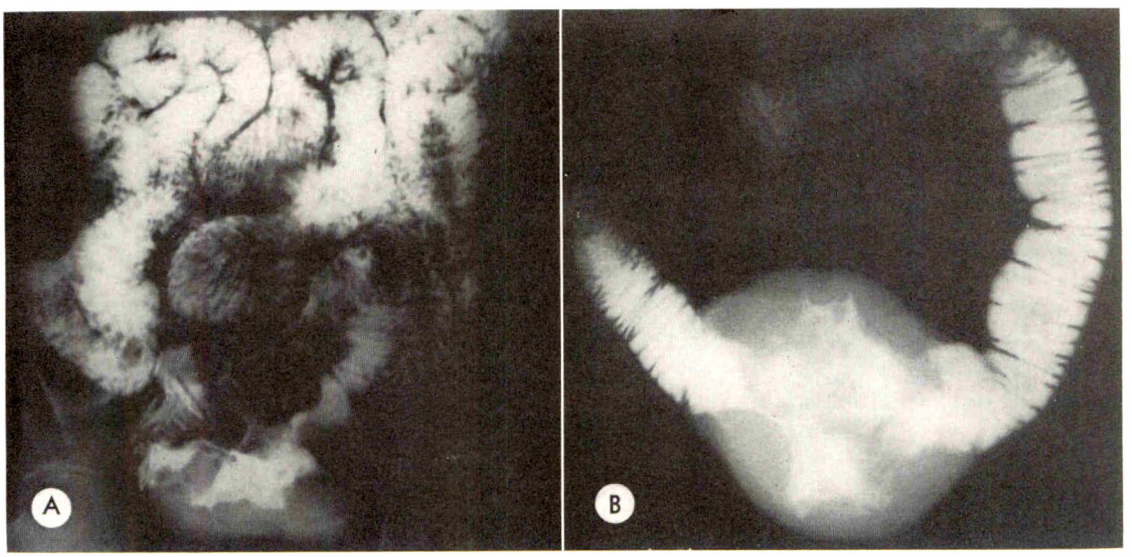


Fig. 1. Case 1. (*A*) Small bowel barium study in a 57 year old man with early aneurysmal lymphoma of the small intestine. A 20 cm. long segment of ileum is seen superimposed on the urinary bladder, still containing contrast material from an intravenous pyelography performed earlier. The barium-filled lumen has a widened, distorted, geographic type of pattern. There is a surrounding mass. (*B*) Barium-filled surgical specimen. The aneurysmal dilatation of the loop of ileum has normal proximal and distal loops. The extent of the ulceration and necrosis within the tumor is well demonstrated. The tumor measures 9×11 cm.

30 minute roentgenogram of the small bowel barium study (Fig. 2*A*) a large 12×10 cm. collection of barium appeared and occupied the entire pelvic inlet. The bag or cavity-like collection had multiple, mottled filling defects in it and appeared to be connected proximally with a normal loop of ileum. An hour later (Fig. 2*B*), after the aneurysmal dilatation had become filled with more barium, a loop of normal appearing ileum was seen distal to it. Decubitus roentgenograms at that time showed an air fluid level in the dilated segment. The 24 hour follow-up roentgenograms showed barium remaining in the dilated segment only. Ten days later there was still barium remaining in the aneurysmal dilatation.

While waiting for abdominal exploration, clinical symptoms of peritonitis developed. At surgery, a perforation of the aneurysmal segment of ileum was found. The large, necrotic tumor was removed in pieces. A second, smaller tumor measuring about 4×5 cm. was removed from the ileum distal to the large aneurysmal segment.

The specimen of the second lesion (Fig. 3) looked like a ballooning of the bowel wall at the antemesenteric border. It had normal proximal and distal ileum attached to it. The cross section revealed a soft, whitish, ulcerated tumor mass partially encircling the bowel wall. The microscopic sections showed malignant lymphoma, lymphangitic type, well differentiated.

Following surgery, the patient had multiple complications and died several weeks later.

DISCUSSION

Dilatation of the small intestine by lymphoma was first described by Moxon in 1873.[19] Ten years later, Bessel Hagen[2] coined the term "aneurysmal" to describe the dilatation of the ileum by lymphoma in a 7 year old boy (Fig. 4). In reviewing the early pathologic descriptions from leading medical centers in Europe during the 1880s and 1890s, Nothnagel and Rolleston[20] stated in 1907 that the majority of lymphomas cause aneurysmal dilatation of the bowel. Recent studies, however, disclosed a lower incidence of aneurysmal dilatation occurring in primary lymphoma of the small intestine.[1,4,5,9] A possible reason for the described decrease in incidence in aneurysmal lymphoma is perhaps the earlier diagnosis of intestinal lymphoma by the small bowel barium examination.

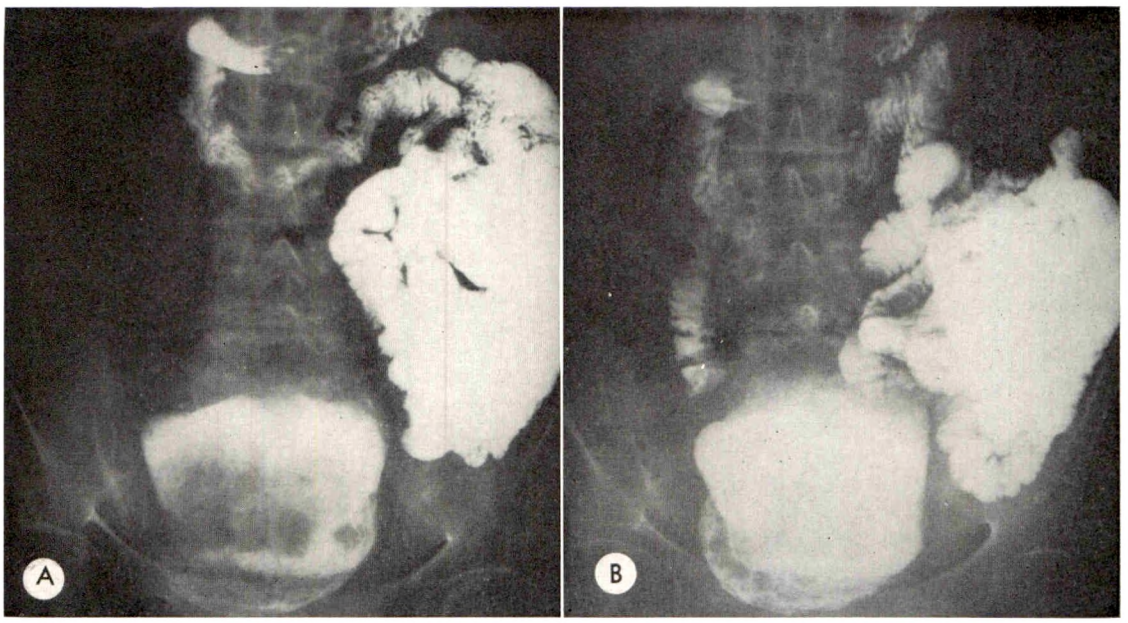


FIG. 2. Case II. Advanced form of aneurysmal lymphoma of the small intestine in a 76 year old woman. (*A*) A 30 minute roentgenogram of the small bowel barium study showing early filling of the aneurysmal dilatation containing secretion and residual food particles. (*B*) A 90 minute roentgenogram of the same small bowel study after the aneurysmal dilatation has accumulated a large amount of barium. There is visualization of the exit normal appearing small intestinal loop. At 24 hours and 10 days follow-up, roentgenograms showed residual barium in the aneurysmal dilatation. Surgery revealed that the aneurysm perforated and was fragmented.

PATHOPHYSIOLOGY

The dilatation of the small intestine involved by lymphoma has been previously explained by the destruction of the myenteric plexuses by the tumor.[3,8,11,14] However, the growth characteristics of bowel lymphoma, and not destruction of the nervous plexuses, allow aneurysmal dilatation to occur.

As the lymphoma grows, it infiltrates peripherally along the long axis of the bowel. This infiltrative process by tumor cells does not cause a desmoplastic reaction as is the case in the growth of an adenocarcinoma of the small intestine.[20–22] Because fibrosis fails to develop in lymphoma, the bowel wall can weaken and dilate. The small intestinal wall weakening occurs in several ways: (a) the infiltrating tumor cells stretch the muscle fibers; once stretched beyond double their length normal contracting ability is lost;[12] (b) the infiltrating tumor cells invade and obstruct the lymphatic and vascular channels, thus resulting in anoxia of the muscle fibers which become necrotic; and (c) the anoxia also causes necrosis of the epithelial lining

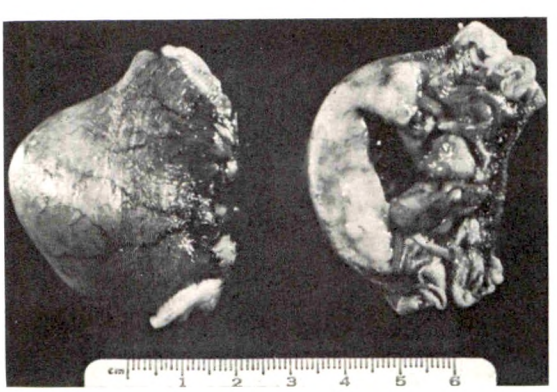


FIG. 3. Case II. Photograph of a second, smaller aneurysmal lymphoma of the ileum found during the surgical exploration in the same patient. The lesion was distal to the large aneurysmal dilatation and was removed intact. There is ballooning of the bowel wall at the antemesenteric border. The cross section reveals a soft, whitish, ulcerated tumor, partially encircling the bowel wall.

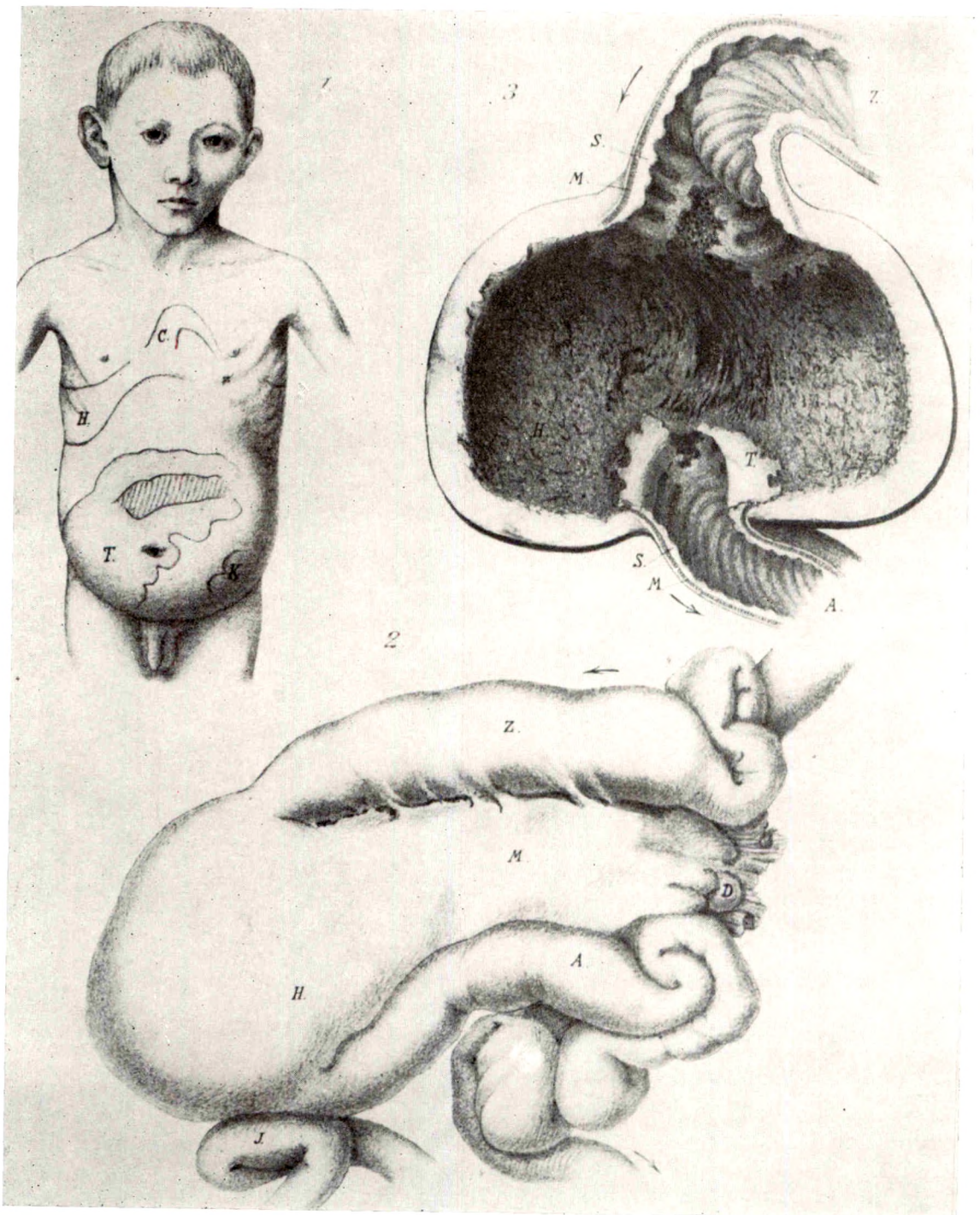


Fig. 4. Aneurysmal lymphoma of the small intestine in a 7 year old boy reported in 1885.[2] 1. Photograph of the patient. 2. Drawing of the gross specimen showing the aneurysmal dilatation (H) between 2 normal appearing small intestinal loops (Z and A). 3. Cross section of the specimen. (With permission of the publisher.)

which is sloughed off as bowel content passes through the lumen. The loss of necrotic tissue surrounding the lumen eventually creates a cavity. Thus, the aneurysmal dilatation is caused by the enlarged lumen and the weakened muscles which cannot contract against the intraluminal pressure.[3,14,21,22]

CLINICAL FINDINGS

Aneurysmal lymphoma can occur in any age group.[9] Bessel Hagen[2] described its occurrence in a 7 year old boy, while our 2 cases occurred in a 57 year old man and in a 76 year old woman. The determining factor is the extent of the tumor growth, not the age of the patient.

Most of the symptoms in patients having aneurysmal lymphoma are similar to patients having other types of lymphoma of the small intestine. The predominant complaints are abdominal pain and weight loss.[4,16,21,24] The pain is usually dull and not radiating, at times crampy. The physical findings can be minimal.

A clinical feature which occurs more frequently in aneurysmal lymphoma than in other types of lymphoma of the small intestine is anemia. It is a significant finding in patients with aneurysmal lymphoma. The above reported patients had a hematocrit of 24 and 25.9 per cent, respectively. Both had blood loss into the bowel lumen.

A palpable abdominal mass is a frequent finding. Obstruction occurs late because the bowel lumen remains open, although markedly distorted and ulcerated.

Aneurysmal lymphoma differs from other types of intestinal lymphoma in that it perforates more often.[25] The perforation can be the result of an ulceration or of necrosis.[3,6,13,16]

ROENTGEN MANIFESTATIONS

Marshak and Lindner[18] give 5 characteristic appearances of lymphomatous involvement of the small intestine: (1) multiple intraluminal nodular defects; (2) polypoid form causing intussusception; (3) endo-exoenteric form with excavation and multiple fistulae formations; (4) invasive form of the mesentery with large extraluminal masses; and (5) infiltrating form. Aneurysmal lymphoma belongs to the fifth form, *i.e.*, when a diffuse infiltration of a segment of small intestine becomes markedly dilated.

The diagnosis of an aneurysmal lymphoma of the small intestine is made on the small bowel study. The barium will outline an acute widening of the bowel lumen (Fig. 1, *A* and *B*; and 2, *A* and *B*). The normal mucosal pattern lining the dilated segments will be replaced by an enlarged and ragged lumen. The other loops of small intestine are usually displaced away from the involved segment because the wall of the dilated loop is thickened by infiltrating tumor.

In early cases, the dilated bowel lumen has a geographic pattern outlined by barium (Fig. 1, *A* and *B*). In advanced cases, the destruction by tumor is so extensive that the lumen, still present within the aneurysmal dilatation, has the appearance of a large bag (Fig. 2, *A* and *B*). The size of this "bag" can be considerable. The segment of bowel entering and leaving the aneurysmal segment is usually normal in caliber and appears to be uninvolved. The visualization of the exit loop can take considerable time, because the aneurysmal segment has lost its peristaltic ability. There might be more than one aneurysmal dilatation of the small intestine.

The barium column in the dilated segment appears mottled on early roentgenograms in a small bowel study. This is the result of retained secretion and food particles in the dilated segment. The dilated segment remains atonic and retains barium for as long as 10 days as demonstrated in Case 11. Decubitus roentgenograms can demonstrate air fluid levels in the aneurysmal dilatation.

Other helpful roentgenographic examinations are: (a) plain roentgenograms of the abdomen which might show the extent of the abdominal mass; (b) intravenous

pyelograms which might show displacement of the ureters and urinary bladder; and (c) barium enema studies which might show displacement of the colon.

Lymphoma of the small intestine can be multicentric,[7] so that one area can show an aneurysmal dilatation, while another can have any other type of roentgenographic appearance of lymphoma.[17,23]

There are definitely other conditions which can cause dilatation of the small intestine and they must be differentiated from aneurysmal lymphoma.

Segmental dilatation of a small intestinal loop can be proximal to an area of narrowing caused by inflammation (regional enteritis), fibrosis (previous infarction) or carcinoma (desmoplastic reaction in adenocarcinoma or carcinoid tumor). The dilated loop preceding the obstruction will have, however, thin but preserved valvulae conniventes. In aneurysmal lymphoma, the mucosal pattern is distorted in early cases and totally destroyed in late cases.

The findings in sprue are: dilatation, segmentation and moulage, which can be confused with aneurysmal dilatation in lymphoma. The dilated loops in sprue are different from aneurysmal lymphoma, for in sprue the valvulae conniventes are still present. Segmentation differs in that there are multiple dilatations connected by strands of barium. Moulage can be similar to aneurysmal lymphoma and can only be differentiated by seeing the other signs of sprue.

Scleroderma and dermatomyositis show dilatation, but the atonic segments are long and contain atrophic valvulae conniventes. Both duplication cyst and Meckel's diverticulum show large collections of barium on the small bowel study. The bowel wall, however, is normal in thickness and the lumen in the dilated segment lacks the ulcerations seen in lymphoma.

It should be kept in mind that other sarcomas of the small intestine such as leiomyosarcomas, fibrosarcomas and neurofibrosarcomas can cause ulceration and dilatation of the bowel.[10,18,23]

SUMMARY

Two unusual cases of aneurysmal lymphoma of the small intestine are reported, one showing early, the other late roentgen features.

The clinical manifestations, the pathophysiology and especially the roentgen findings are discussed.

In the early form of aneurysmal lymphoma, the involved segments show a ragged, geographic pattern of the barium-filled lumen and a companion mass.

In the advanced form, large, aneurysm-like dilatations are demonstrated on the small bowel study. Such aneurysmal cavities accumulate barium and retain it for days.

In both instances, the small intestinal loops entering and leaving the aneurysmal lymphoma are normal in caliber and free of disease.

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DIFFUSE EOSINOPHILIC GASTROENTERITIS*

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DIFFUSE eosinophilic gastroenteritis is a syndrome of peripheral blood eosinophilia, gastric and small bowel nodular deformities (seen roentgenographically), and dense gastrointestinal eosinophilic infiltration (detectable microscopically). This disease has been reported in 56 patients since its description in the late 1930s. Because this disease often has a distinct roentgenographic small bowel pattern accompanied by a gastric deformity, accurate roentgenographic interpretation is frequently possible.

The purpose of this paper is to describe this roentgenographic pattern of diffuse eosinophilic gastroenteritis and to illustrate it by findings in 4 patients evaluated at Walter Reed General Hospital over the past 4 years. The extent of small bowel involvement in conjunction with gastric involvement and the complete roentgenographic reversibility of the condition by means of corticosteroid therapy will be stressed. Etiology and classification of this entity will also be discussed.

REPORT OF CASES

Case 1. A 22 year old Caucasian enlisted man of Mediterranean extraction was seen for evaluation of abdominal complaints, peripheral eosinophilia, anemia, and splenomegaly. He had experienced prandial abdominal cramps, nausea, vomiting and diarrhea associated with a 20 pound weight loss over a period of 2 years. He reported no hematemesis, hematochezia, or melena. Roentgenograms reportedly were normal shortly after the onset of his illness. The patient denied that he had traveled outside of the United States. Although he admitted to occasional episodes of "hay fever," no food allergies, asthma, or drug allergies were noted. Physical examination on admission revealed palor, a palpable spleen tip, and minimal axillary and inguinal lymphadenopathy.

Laboratory studies included an initial hematocrit of 30 per cent and a white blood cell count of 15,000/mm.^{3} with 48 per cent eosinophils. Peripheral smear, blood indices, serum, and bone marrow iron studies indicated severe iron deficiency. An elevated level of A_2 hemoglobin (5.6 per cent) was present on hemoglobin electrophoresis. Occult blood was present intermittently in stools; however, multiple stools and duodenal drainage were negative for parasites. Routine skin tests and blood chemistries were within normal limits. Serum albumin was initially 2.9 gm. per cent. Proctosigmoidoscopy and barium enema examination were normal. Upper gastrointestinal roentgenograms revealed thickened valvulae conniventes and separation of the small bowel loops (Fig. 1).

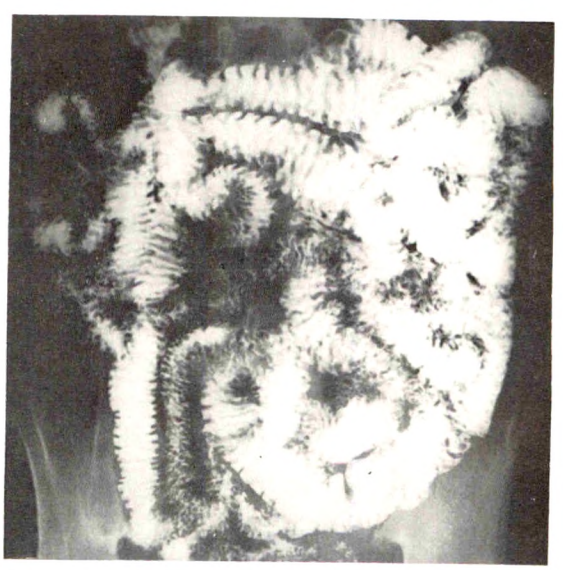


Fig. 1. Case 1. The duodenal, jejunal, and ileal valvulae conniventes are thickened. Separation of bowel loops denotes thickening of bowel wall and possibly mesentery. Small bowel biopsy from proximal jejunum confirmed dense eosinophilic infiltration.

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The stomach appeared normal. Twelve grams of fat was present in a 24 hour stool collection. Esophagogastroscopy revealed patchy distal gastritis and a biopsy from this area showed infiltration of the lamina propria with mature eosinophils. A Crosby capsule small bowel biopsy from the region of the ligament of Treitz showed eosinophilic infiltration of the lamina propria.

On the basis of these studies, it was felt that this patient had thalassemia minor and eosinophilic gastroenteritis. Consequently, he was started on a trial dose of prednisone 15 mg./day. On this dose of corticosteroids his symptoms improved and his peripheral eosinophils decreased from 50 per cent to 23 per cent. Because of the failure of the eosinophils to decrease below 20 per cent, he was treated empirically with tetrachloroethylene; no further reduction in the eosinophils was noted, however. Consequently, the prednisone was restarted in the same dosage and over several weeks the peripheral eosinophils decreased to 5 per cent. Follow-up upper gastrointestinal roentgenograms and small bowel biopsy at this time were entirely normal.

Over the past 4 years, this patient has had to be maintained on corticosteroids. His usual daily maintenance dosage has been 5 mg. of prednisone and on this low dose he has remained asymptomatic with peripheral eosinophils under 10 per cent with no evidence of gastrointestinal bleeding or malabsorption. Discontinuation of steroid therapy has always resulted in recurrence of the symptoms with a concomitant rise in the peripheral eosinophil level.

Case II. A 28 year old Caucasian physician was admitted for evaluation of a 3 month history of diarrhea, abdominal cramps, and a 15 pound weight loss. The abdominal cramps were related to all types of food ingestion and the diarrhea which followed shortly after meals resulted in stool which was watery and brown with considerable mucus. Shortly before admission he noted early satiety and postprandial vomiting. Past medical history was unremarkable except for mild seasonal allergies. The patient specifically denied intolerance to food. He said his father had "hay fever" and his mother "an asthmatic condition." On initial physical examination there was evidence of moderate dehydration and hyperactive bowel sounds; otherwise the remainder of the examination was within normal limits.

Laboratory studies during hospitalization revealed a persistent leukocytosis (15,000–50,000/mm.^{3}) with 50–60 per cent eosinophils. Routine blood chemistry procedures revealed reduced serum proteins and cholesterol. Fecal fat determination and d-xylose absorption were normal. Stools were negative for parasites. Tuberculin skin tests were negative.

Upper gastrointestinal roentgenograms demonstrated irregularity and narrowing of the distal antrum and distortion of the jejunal pattern with effacement of the valvulae, thickened bowel wall and narrowing of the lumen (Fig. 2, *A* and *B*). Results of barium enema examination were normal. Chest roentgenograms revealed minimal pleural effusion and thoracocentesis yielded clear yellow fluid with 4,000 cells/mm.^{3}, 97 per cent of which were eosinophils. Small bowel biopsy showed mucosal edema and a heavy eosinophilic infiltration of the lamina propria.

Two weeks of nasogastric suction and then a diet of clear liquids improved the patient's condition. The peripheral eosinophils decreased to 30 per cent. At the time of his discharge the patient was able to tolerate a regular diet without difficulty. The roentgenographic appearance of the small bowel had returned to normal. A follow-up small bowel biopsy was not performed. Over the next year the patient had a persistently elevated level of peripheral blood eosinophils (10–20 per cent) and experienced intermittent, mild episodes of abdominal cramping and diarrhea associated with a further increase of eosinophils. He treated these episodes by abstaining from food for a day or two and noted improvement within this period; 16 months after onset of symptoms, however, he had a severe flare-up of symptoms with a marked rise of eosinophils. He was hospitalized and given steroids which resulted in almost immediate clinical improvement and a rapid reduction in his peripheral eosinophils. Steroid dosage was tapered then stopped and the patient resumed a normal diet; over the next 5 years he noted only minimal and infrequent symptoms. His peripheral eosinophil count, however, remains elevated.

Case III. A 21 year old Caucasian soldier developed abdominal cramps, nausea, and intermittent diarrhea, while stationed in West

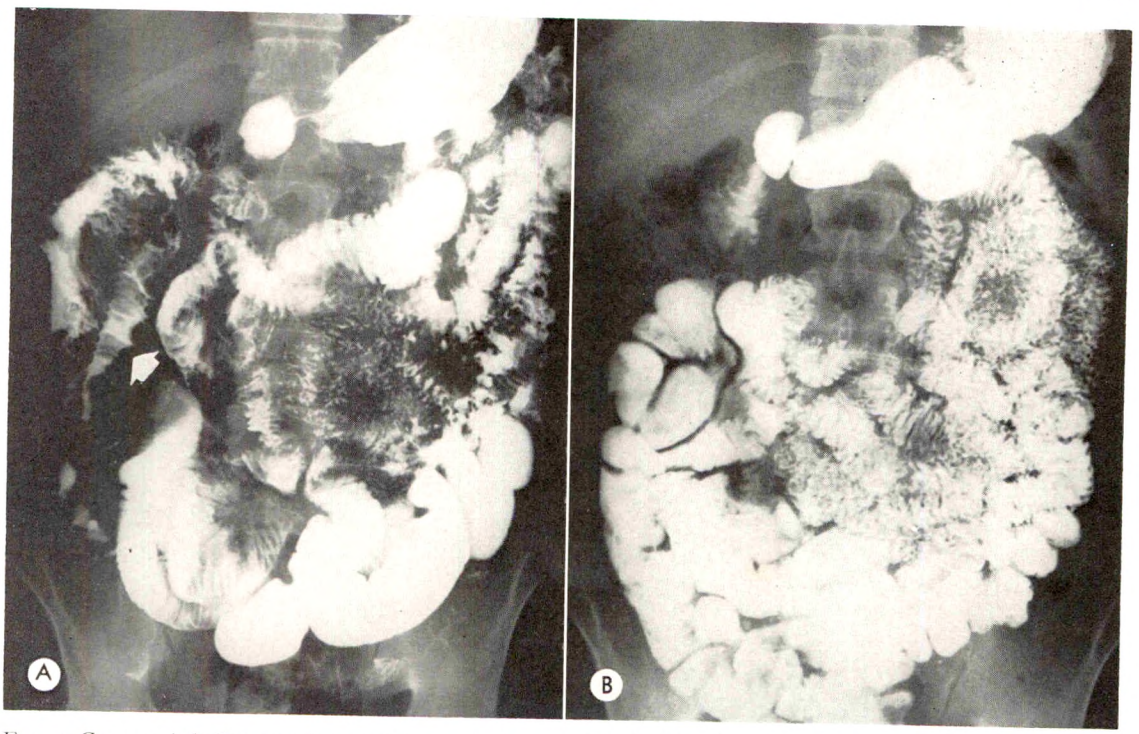


FIG. 2. Case II. (*A*) Concentric antral narrowing is associated with distortion of the mucosal fold pattern of the jejunum. Extensive eosinophilic infiltration may produce narrowing of the intestinal lumen and effacement of mucosal folds, as well as separation of bowel loops (arrow). (*B*) Seven weeks after the abnormal small bowel roentgenograms in *A* were obtained, the small bowel pattern had returned to normal. This patient had not received steroids. The sole method of treatment was bowel rest (nasogastric suction for 2 weeks with concomitant intravenous feeding, then 1 month of liquid diet without milk).

Germany. Since this condition persisted for 1 month, uncontrolled by anticholinergic therapy, he was hospitalized for further evaluation. The year before, while stationed in South Vietnam, he had developed malaria, which was successfully treated. The remainder of his past medical history was noncontributory. He gave no personal or family history of allergy.

Physical examination on admission was unremarkable. A white blood cell count of 15,000 per $mm.^3$ with 60 per cent eosinophils was present. The hematocrit was 37 per cent and blood indices, peripheral smear, serum, and bone marrow iron studies confirmed iron deficiency. Stool guaiacs ranged from 1–3+ positive for occult blood. Intestinal absorption proved to be normal; upper gastrointestinal roentgenograms, however, revealed diffuse mucosal thickening of the small bowel and antral irregularity (Fig. 3, *A–C*). Peroral biopsy of the small bowel from a site near the ligament of Treitz revealed dense eosinophilic infiltration of the lamina propria with normal villous architecture. Multiple stools were negative for parasites. Duodenal aspiration revealed hookworm ova, and the patient was given a 4 day course of thiabendazole resulting in mild symptomatic improvement and a decrease in peripheral eosinophils from 50 per cent to 20 per cent. Within 2 weeks, however, he was again symptomatic and peripheral eosinophils had returned to pretreatment levels. Stools and duodenal drainage this time showed no parasites; the patient was therefore started on a daily dosage of 50 mg. prednisone. Symptomatic improvement was immediate and the peripheral eosinophil count returned to normal. Prednisone dosage was decreased to 20 mg. every other day.

With the decrease in steroid dosage the patient again developed cramps and peripheral eosinophils of 20 per cent. Small bowel roentgenograms at this time showed irregularity of the small bowel pattern similar to that of the initial study; a small bowel biopsy revealed a moderately heavy infiltration of eosinophils. Again multiple stools and duodenal aspiration

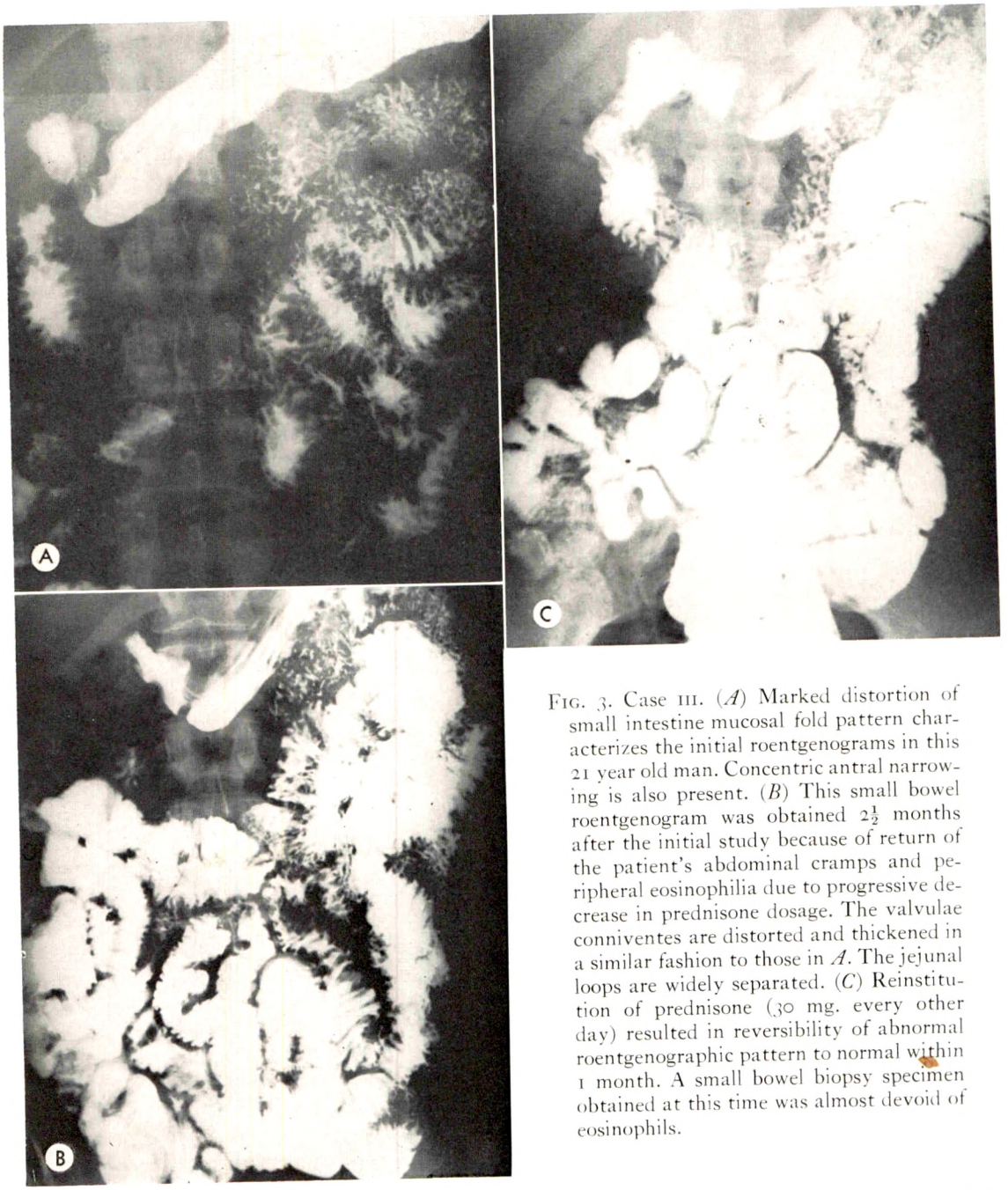


Fig. 3. Case III. (*A*) Marked distortion of small intestine mucosal fold pattern characterizes the initial roentgenograms in this 21 year old man. Concentric antral narrowing is also present. (*B*) This small bowel roentgenogram was obtained $2\frac{1}{2}$ months after the initial study because of return of the patient's abdominal cramps and peripheral eosinophilia due to progressive decrease in prednisone dosage. The valvulae conniventes are distorted and thickened in a similar fashion to those in *A*. The jejunal loops are widely separated. (*C*) Reinstitution of prednisone (30 mg. every other day) resulted in reversibility of abnormal roentgenographic pattern to normal within 1 month. A small bowel biopsy specimen obtained at this time was almost devoid of eosinophils.

revealed no parasites. Empiric retreatment with thiabendazole this time did not improve the abdominal symptoms or decrease the peripheral eosinophils. However, increase of his daily dosage of prednisone to 40 mg. resulted in disappearance of his symptoms and a return of his peripheral eosinophil level to less than 5 per cent. A follow-up biopsy showed the small bowel to be normal. The steroid dosage was slowly tapered to 30 mg., to be administered every other day; on this dose the patient remained asymptomatic. Attempts at decreasing the dosage further resulted in a return of symptoms and a rise in per cent of peripheral eosinophils.

Case IV. A 24 year old Black enlisted man noted the onset of abdominal cramps, early

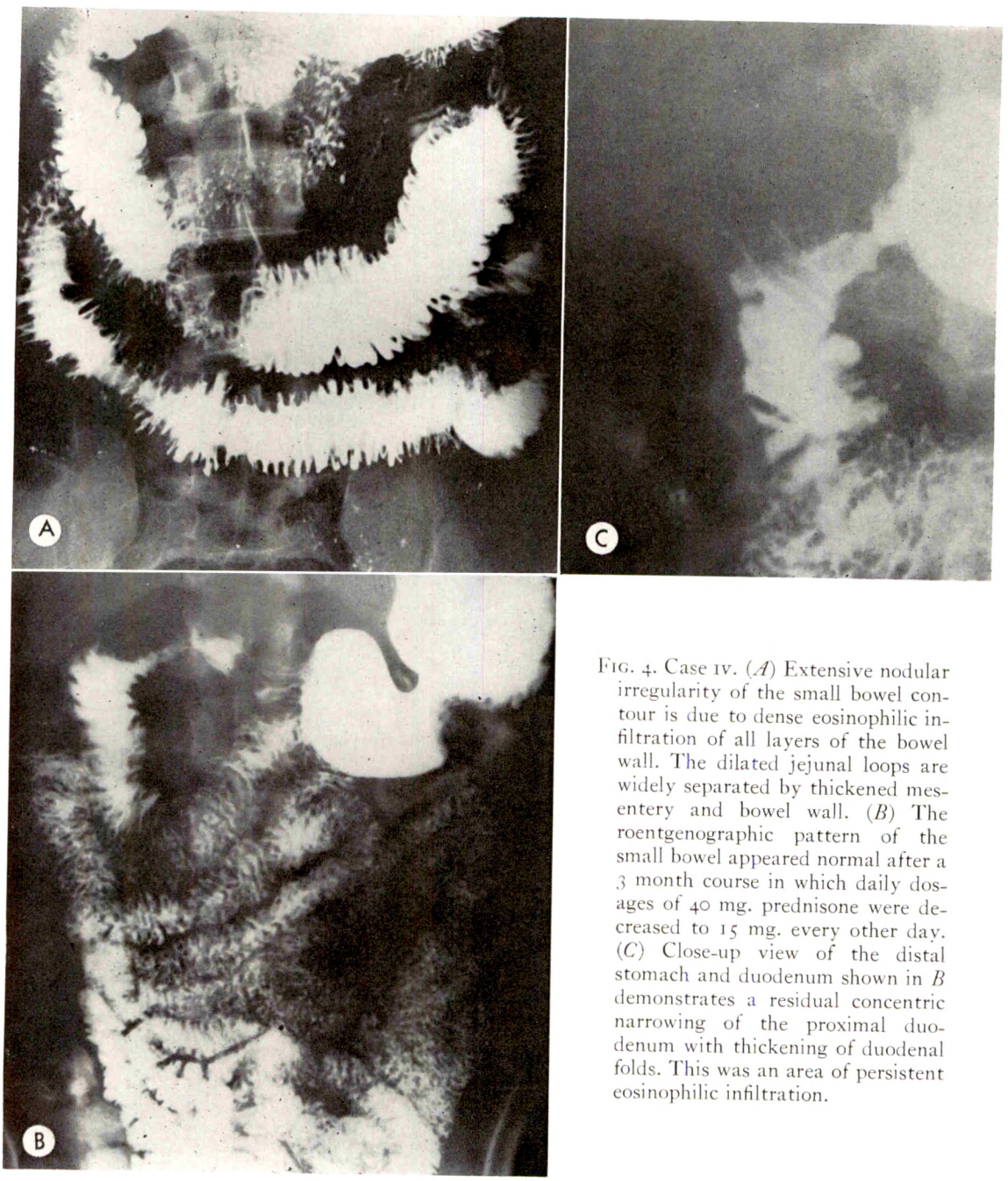


FIG. 4. Case IV. (*A*) Extensive nodular irregularity of the small bowel contour is due to dense eosinophilic infiltration of all layers of the bowel wall. The dilated jejunal loops are widely separated by thickened mesentery and bowel wall. (*B*) The roentgenographic pattern of the small bowel appeared normal after a 3 month course in which daily dosages of 40 mg. prednisone were decreased to 15 mg. every other day. (*C*) Close-up view of the distal stomach and duodenum shown in *B* demonstrates a residual concentric narrowing of the proximal duodenum with thickening of duodenal folds. This was an area of persistent eosinophilic infiltration.

satiety, and recurrent episodes of nausea and vomiting for 1 month. He was treated with anticholinergic drugs for 2 months without relief of his symptoms and during this period lost 30 lb. Past medical history and physical examination were unremarkable. The patient denied any allergies or specific food intolerance; he stated, however, that one of his brothers has "bronchial asthma."

The initial white blood cell count was 25,000/mm.^{3} with 69 per cent eosinophils. Upper gastrointestinal roentgenograms revealed nodular irregularity of the distal stomach and much of the small bowel (Fig. 4, *A–C*). A barium enema procedure and extensive hematologic and malabsorption evaluations disclosed nothing abnormal. Examination of stools and duodenal drainage gave no evidence of ova and parasites.

Histologic examination of the distal stomach and a biopsy of the small bowel revealed heavy eosinophilic infiltration of the lamina propria.

Diagnosis on the basis of these studies was eosinophilic gastroenteritis; the patient was started on a daily dosage of 40 mg. prednisone. Dramatic improvement ensued and the level of peripheral eosinophils decreased to 7 per cent within 1 week. Within 3 weeks, total eosinophil count had returned to normal. Roentgenograms of the upper gastrointestinal tract and histologic examination of the stomach and small bowel yielded no abnormal findings. The prednisone dosage was tapered to 15 mg., to be taken every other day; attempts to decrease the steroid dosage below this level, however, resulted in exacerbation of symptoms and an increase in peripheral eosinophils. At the end of 1 year the patient is asymptomatic on his program of 15 mg. steroid dosage taken alternate days.

ROENTGENOGRAPHIC FINDINGS

The roentgenographic abnormalities of diffuse eosinophilic gastroenteritis are typical of most infiltrative diseases that involve the small bowel. Although the jejunum is most prominently involved,[2,7,11,16] the entire small bowel may be affected,[16] and occasionally the colon.[9,12] All of our patients exhibited extensive duodenal, jejunal, and proximal ileal involvement. Involvement of the small bowel is often associated with gastric eosinophilic infiltration.[2] In 3 of our cases, there was roentgenographic evidence of concomitant gastric involvement. Gastric involvement may be manifested by enlargement of rugal folds,[4] narrowing and rigidity of the antrum, or a bulky irregular intraluminal mass. Although the antrum is the major area affected, the entire stomach may be involved.[4,15] In all 4 of our patients, involvement was limited to the antrum. In Case 1, patchy antral mucosal abnormalities were noted by the endoscopist and later confirmed by biopsy as areas of eosinophilic infiltration, although the antrum appeared normal on upper gastrointestinal roentgenograms.

Thickening and widening of the valvulae conniventes are the most common roentgenographic findings of eosinophilic infiltration of the small bowel (Fig. 1; and 4, *A–C*). Infiltration of the mucosa and lamina propria results in regular nodular contour defects. These thickened valvulae are sharply outlined by barium—unlike the appearance of thickened folds due to edema. More extensive involvement of the bowel wall or transmural involvement results in distorted valvulae with irregular angulation and a saw-toothed contour of the small bowel (Fig. 2*A*; and 3*A*). Infiltration may be so pronounced as to efface completely the valvulae, resulting in narrowing of the bowel lumen (Fig. 2*A*); the bowel at fluoroscopy may then appear rigid. Involvement of the mesentery and omentum may result in separation of intestinal loops. In those patients with steatorrhea from malabsorption, the barium column may be somewhat segmented and flocculated.

DISCUSSION

Eosinophils in the mucosa of the gastrointestinal tract are common. Massive infiltration of the wall of the stomach and small bowel by eosinophils, in association with an increased amount of eosinophils in the peripheral blood are hallmark findings of eosinophilic gastroenteritis. Eosinophilic gastroenteritis has also been called idiopathic eosinophilic infiltration of the gastrointestinal tract, eosinophilic gastroduodenitis, eosinophilic granuloma of the stomach and small intestine, gastric submucosal granuloma with eosinophilic infiltration, gastric lesion of Loeffler's syndrome, and allergic gastroenteropathy.

Because the etiologies of eosinophilic infiltration in the gastrointestinal tract are controversial, some confusion exists as to whether all of these terms should be applied to diffuse full-thickness gastrointestinal infiltration by eosinophils. Review of the literature indicates 3 apparently separate pathologic states. The first is known as allergic gastroenteropathy,[8,16] which occurs mainly in the first and second decades of life. Hypoalbuminemia, hypogammaglobu-

linemia, anemia, blood eosinophilia, and small bowel eosinophilic infiltration limited to the mucosa are all associated with this condition. In patients with allergic gastroenteropathy a definite history is given of allergy, particularly to milk products and meat.[6,8,12,16] Two of our 4 patients (Cases 1 and 11) had only "hay fever" type allergies without apparent food allergy, asthma, or urticaria. The other 2 had no allergic history. All had the same roentgenographic and histologic changes and were over 20 years of age. At the present time, allergic gastroenteropathy is separated from the diffuse eosinophilic gastrointestinal involvement by an established allergic history, almost sole involvement of the mucosa, and the high incidence in childhood. Allergic gastroenteropathy and diffuse eosinophilic gastroenteritis without associated allergy, however, have many features in common. Caution should be exercised in attempting to classify definitely these 2 processes as separate diseases.

In diffuse eosinophilic gastroenteritis, the second pathologic state, eosinophils are prominent in all layers of the bowel wall but mainly the submucosa. About one-half of patients with this disease give a history of childhood allergy.[11] Eosinophilic gastroenteritis is a disease of adults, the youngest reported patient (with 1 exception)[6] was more than 20 years of age.[9] Beside allergy to external agents, a systemic hypersensitivity reaction[3,9,16] and a systemic manifestation of Loeffler's syndrome[15] have been considered causes of diffuse eosinophilic gastroenteritis.

The third disease state is local eosinophilic granuloma of the stomach, which is probably a distinctive entity separate from the diffuse involvement of the gastrointestinal tract. It is best considered as granuloma of the stomach with eosinophils present. Patients may or may not have an allergic history. This disease is localized to the stomach.

In patients with diffuse eosinophilic gastroenteritis either on an allergic basis or without evidence of allergy, the usual gastrointestinal symptoms are cramping abdominal pain, weight loss, and diarrhea (as manifested by our 4 patients). Nausea and vomiting have also been noted, particularly when the stomach as well as the small bowel is involved.

Hypoproteinemia may result in abdominal distention and swelling of ankles.[6,11] Eosinophilic ascites has also been noted in this disease, probably secondary to serosal involvement.[11,16] Eosinophilic pleural effusion was present in 1 of our patients (Case 11), but there was no evidence of ascites. In extensive mucosal invasion, malabsorption results in steatorrhea.[1,11,13] Duration of symptoms varies from a few months[2,14] to several years. One patient (Case 1) had symptoms for 2 years before his condition was diagnosed; the other 3 had symptoms for only a few months.

All patients with diffuse eosinophilic involvement of the gastrointestinal tract with or without allergic histories, have a high percentage of eosinophils in the blood, ranging from 10 to 80 per cent. Anemia may be frequent. In those instances of eosinophilic disease of the intestine with protein-losing enteropathy, serum proteins will be decreased.[10] Immunoglobulin A, immunoglobulin G, and immunoglobulin M have been reported to be normal.[11]

Biopsy of the small bowel in eosinophilic gastroenteritis may reveal a normal-appearing epithelium and preserved villi, with eosinophilic and plasma-cell infiltration of the lamina propria (Fig. 5).[2,11] The nature of these biopsies, however, is such that the extent of the eosinophilic involvement throughout the wall of the intestine cannot be fully appreciated. The depth of which the biopsy specimen is obtained may be important, since some authors have stated that eosinophilic gastroenteritis is best classified by the depth of involvement of the bowel wall.[12,16] The 4 patients in our series all had extensive eosinophilic infiltration throughout the lamina propria of the small bowel. The sites of biopsy extended only slightly into

the submucosa. Since none of the patients had undergone a surgical procedure, we do not know to what depth the bowel wall was involved.

In diffuse eosinophilic gastroenteritis the stomach and small bowel wall are grossly thickened. The appearance is similar to that of lymphoma and regional enteritis.[9,16] Full thickness specimens of the small bowel obtained at the time of surgical operation have shown that the intestinal villi are preserved, but eosinophil and plasma-cell infiltration are distributed diffusely throughout all layers of the bowel wall. Hypertrophy of the muscle layers may add to the bowel thickness.[6] The eosinophilic infiltration in this disease is more dense than in other causes of gastrointestinal eosinophilia, such as Hodgkin's disease, carcinoma, amebiasis, helminthic disease, and regional enteritis.

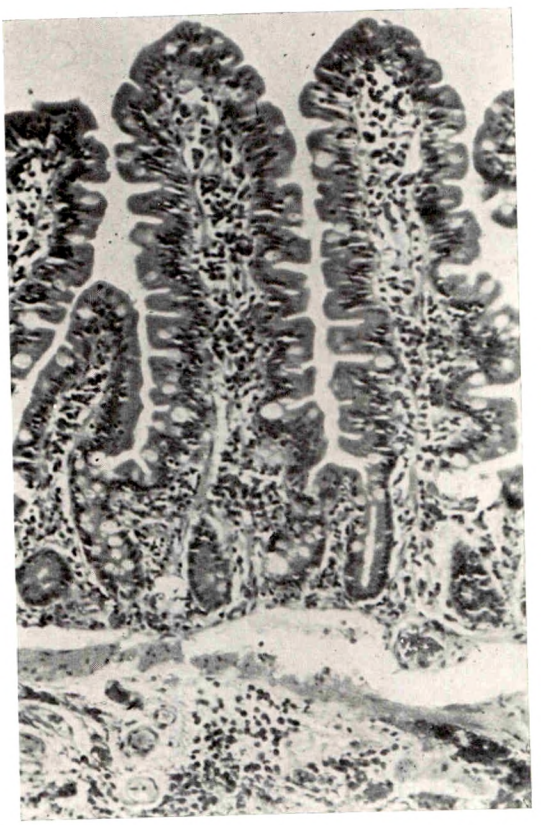


Fig. 5. Microscopic section (×100) obtained by means of small bowel biopsy from one patient (Case IV) demonstrates dense eosinophilic infiltration throughout the lamina propria. The villi, although harboring large numbers of eosinophils, are not thickened or blunted.

Response to Steroid Therapy

After systemic steroid therapy, the roentgenographic evidence of eosinophilic infiltration may disappear,[2,9,12,14] as well as the clinical symptoms and the peripheral eosinophilia. The mucosal pattern has been reported to revert to normal as early as 2 months after the start of steroid therapy and the roentgenographic appearance may remain normal for 5 years.[2] The roentgenographic abnormalities in the small bowel of the patient of Case IV reverted to normal 3 weeks after steroid therapy was initiated. Intestinal motility, frequently altered by the diffuse transmural eosinophilic infiltration as manifested by areas of spasm and dilatation, also returns to normal after steroid therapy.[9] Occasionally, abnormalities may still be seen on roentgenograms even though physical improvement is pronounced.[11] In all of our 4 patients, steroid therapy (varying from 40 mg. prednisone daily to 15 mg. daily) resulted in prompt reversal of clinical symptoms, noted as early as 1 week in 1 instance (Case IV). The eosinophilic infiltration of the small bowel disappeared in 2 of our 3 patients after steroid therapy of 1–3 months; this was confirmed by repeated biopsy. The persistence of eosinophilia in the small bowel in one instance (Case III) even though roentgenographic and clinical findings had reversed, probably reflects that steroid therapy was at a suboptimal level since an increased daily dosage (40 mg. prednisone) led to disappearance of the eosinophilic infiltration. It is noteworthy that, in this patient, abnormalities were not evident roentgenographically—even though steroid therapy was incomplete and eosinophilic infiltration was present. This suggests that the extent of eosinophilic infiltration determines the degree to which the abnormality can be seen on roentgenograms.

Two of our patients have been main-

tained on prednisone (Case I, 5 mg. per day for 4 years, and Case IV, 15 mg. every other day for 1 year) without exacerbation. When steroid therapy was withdrawn from 3 of the patients (Cases I, III, and IV), however, the clinical symptoms and peripheral eosinophilia reappeared. The fourth patient (Case II) has not taken steroids for 5 years and has experienced mild intermittent abdominal cramps and persistent peripheral eosinophilia. Complete clinical, histologic, and roentgenographic remission is apparently steroid-dependent. Although the disease may exacerbate, it is usually not treated surgically. Only when steroids do not effect relief of intestinal obstruction is surgery indicated.

DIFFERENTIAL DIAGNOSIS

Eosinophilic gastroenteritis must be differentiated roentgenographically from other causes of transmural infiltration of the small intestine. Findings in regional enteritis, tuberculous enteritis, Whipple's disease, lymphosarcoma, intramural hemorrhage, periarteritis, and amyloidosis may all resemble those of eosinophilic enteritis. In all these diseases, as well as in eosinophilic infiltration, the stomach and duodenum may be involved along with the small bowel, and all these diseases may result in extensive infiltration with separation of bowel loops. The clinical history of peripheral blood eosinophilia and allergy to certain foods, as well as the recognition of concomitant gastric and small bowel infiltration become exceedingly important for the correct interpretation of roentgenograms. It is not possible to arrive at a specific diagnosis of eosinophilic gastroenteritis solely on the basis of roentgenographic findings; clinical history and laboratory data must also be understood and taken into account.

SUMMARY

Four examples of diffuse eosinophilic gastroenteritis are reported to demonstrate roentgenographically the abnormalities which may be produced by eosinophilic infiltration of the stomach and small bowel.

The nodular contour defects produced by dense eosinophilic infiltration of mucosa and submucosa are often sharply defined.

Involvement of the muscularis, serosa, and mesentery results in separation of the bowel loops. Gastric involvement is often accompanied by small bowel disease.

The roentgenographic features are completely reversible as a result of systemic steroid therapy.

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MALABSORPTION SECONDARY TO MESENTERIC ISCHEMIA*

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THE clinical features of the syndrome of abdominal angina due to vascular insufficiency of the intestines are well recognized. In 1958, Shaw and Maynard[34] first directed attention to the association of malabsorption with intestinal ischemia. Further experience has shown that malabsorption may be the initial or the dominant feature of the clinical presentation. Early recognition of the presence of chronic mesenteric ischemia underlying such cases of malabsorption is imperative in order to avoid catastrophic infarction of the intestine. Surgical advances have now rendered most of these arterial occlusions remediable.

The most definitive diagnostic evaluation is accomplished by abdominal aortography. Certain clinical and roentgenographic features may be helpful in the selection of patients with malabsorption for aortography to delineate the sites of arterial occlusion.

REPORT OF A CASE

This 62 year old white male was admitted with a history of diarrhea and a 70 pound weight loss over the past 15 months. Watery brownish bowel movements alternated with bulky yellowish stools. Some abdominal distention, occasional vomiting and frequent eructation were noted. Generalized abdominal discomfort began 4 months prior to admission, becoming principally epigastric and postprandial, occurring especially 20–30 minutes after meals. This led to sitophobia and the ingestion of frequent small meals. Weight loss had advanced, particularly over the previous 3 months.

Physical examination revealed a cachectic male with angular stomatitis. Blood pressure was 120/80 mm. Hg. No abdominal bruit was present. Femoral pulses were intact but the posterior tibial and dorsalis pedis pulses were not palpable The remainder of the physical examination was within normal limits. Stools were negative for occult blood, ova and parasites. Smear of a random stool revealed the presence of numerous fat globules. White blood cell count was 12,000/ml.^{3}. Blood sugar, blood urea nitrogen, electrolytes, calcium, phosphorus, alkaline phosphatase, amylase, serum albumin and globulin, total and direct bilirubin, serum cholesterol, thymol turbidity, lactic dehydrogenase, and urinalysis were all within normal limits. Electrocardiogram showed changes compatible with an old inferior wall infarct. Chest roentgenogram and a barium enema study were normal.

The clinical impression of malabsorption was confirmed by a radioactive triolein study demonstrating excretion of 50.5 per cent of the administered dose of labeled fat in a pooled 72 hour stool collection (normal: <5 per cent). Less than 6.6 per cent of the administered I^{131} label appeared in the urine within 24 hours.

An upper gastrointestinal and small bowel series, utilizing a colloidal barium suspension, demonstrated a "sprue-like pattern," characterized principally by dilated jejunal loops, segmentation, and a transient ileal intussusception (Fig. 1, *A–C*). No hypersecretion was present.

Further studies of intestinal absorption were initiated and abdominal aortography was considered. On the twelfth hospital day, however, the patient developed severe generalized abdominal pain. The abdominal wall was rigid and for the first time an abdominal bruit was heard. He became hypotensive and despite resuscitative measures expired.

At autopsy, there was infarction of the intestine extending from 1 cm. distal to the ligament of Treitz to the rectosigmoid junction with a 0.5 cm. perforation of the midjejunum secondary to infarct necrosis. In the ileum, there was evidence of marked mucosal atrophy

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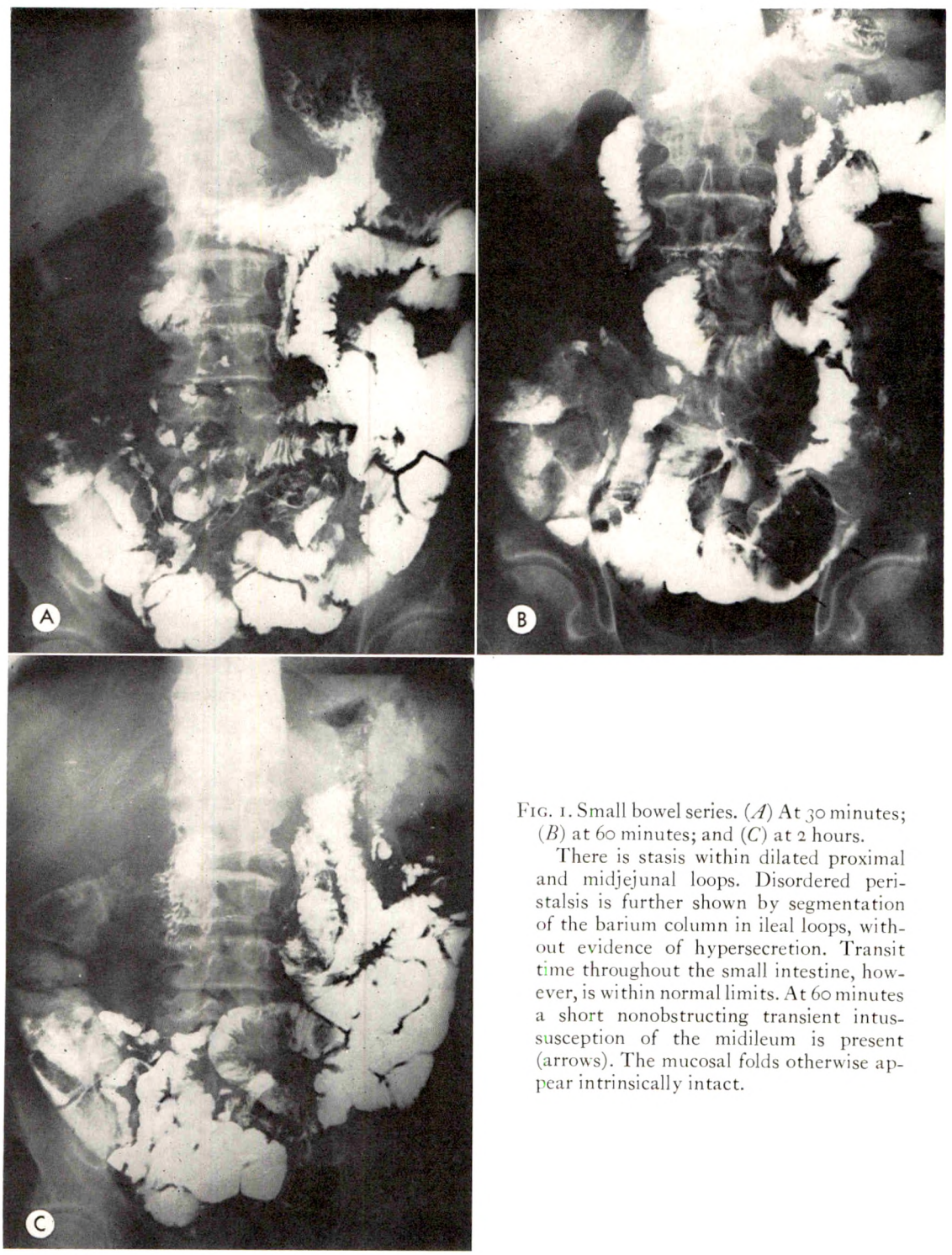


Fig. 1. Small bowel series. (*A*) At 30 minutes; (*B*) at 60 minutes; and (*C*) at 2 hours.

There is stasis within dilated proximal and midjejunal loops. Disordered peristalsis is further shown by segmentation of the barium column in ileal loops, without evidence of hypersecretion. Transit time throughout the small intestine, however, is within normal limits. At 60 minutes a short nonobstructing transient intussusception of the midileum is present (arrows). The mucosal folds otherwise appear intrinsically intact.

and submucosal fibrosis antedating the final episode. The abdominal aorta was involved with moderate atheromatosis with calcification and ulceration of the plaques, occluding the ostium of the inferior mesenteric artery. A common celiacomesenteric trunk was present. A pale yellow to white thrombus of long-standing narrowed its proximal lumen by 50 per cent, extending 1.5 cm. distal to its ostium. Complete occlusion of the remaining lumen had been produced by a bright red and fleshy thrombus. In addition, there was a chronic thrombus in the distal half of the vessel with a possible residual lumen in the area of the occlusion. A 2 cm. prepyloric ulcer of the greater curvature of the stomach had perforated leading to acute peritonitis. Histologically acute centrolobular necrosis of the liver was seen.

The heart showed marked coronary arteriosclerosis.

DISCUSSION

Chronic mesenteric vascular insufficiency has been documented as a cause of the malabsorption syndrome.[8,13,18,21,22,32,34,37,38] Watt *et al.*[37] reported malabsorption in 4 of 5 cases of chronic intestinal ischemia. Morris and DeBakey[22] found frequent abnormal fecal fat excretion in the setting of abdominal angina. The patients reported with ischemic malabsorption have all been shown to have diarrhea, weight loss, and either low serum carotene, abnormal xylose absorption, or increased fecal fat excretion.[8,32,34,38] Correction of the vascular lesions by thromboendarterectomy or bypass graft has been shown to reverse the malabsorption as well as alleviate the pain.[8,34,38] In several instances ischemic malabsorption has been noted following surgical excision of an acute embolic occlusion of a branch of the superior mesenteric artery.[15,34,39] This impairment improves gradually with a complete resolution after several months. Since it is known that villous structure reconstitutes itself rapidly, the explanation for the delayed regression of the malabsorption in this circumstance is not clear. Impaired absorption has also been observed with mesenteric arteriovenous fistulas,[23,24] evidently on the basis of a vascular steal from the intestinal perfusion. In addition to the clinical reports cited, further support for the role of vascular disease comes from studies in which malabsorption has been produced in dogs following partial superior mesenteric artery ligation.[26] These animals develop weight loss and diarrhea with abnormal I^{131} triolein and xylose absorption as well as histologic alterations in the small bowel mucosa. The condition is temporary, however, reverting to normal after several months, presumably as collateral circulation develops.

Abdominal pain has been a concomitant feature in almost all cases of ischemic malabsorption described, but is variable in intensity and character. While it characteristically is postprandial and periumbilical, it may also simulate the pain of pancreatitis with radiation toward the back and relief by a stooped posture. It usually precedes or occurs simultaneously with the malabsorption syndrome, the features of which may dominate the clinical picture until the abdominal angina increases in intensity later in the course. In the case reported, however, diarrhea antedated the onset of abdominal pain, indicating that there is variability in the order of appearance of symptoms.

Certain clinical features in addition to postprandial distress, are of particular significance in distinguishing malabsorption secondary to visceral ischemia: (1) age of onset. In Badenoch's[4] series of 106 patients with idiopathic steatorrhea, all but 14 had the onset of malabsorption in childhood or before the age of 50. In contrast, ischemic malabsorption is seen most commonly after the age of 50; (2) evidence of generalized arteriosclerotic disease; (3) presence of an abdominal bruit.[30]

Not all patients with chronic mesenteric vascular insufficiency and abdominal angina develop malabsorption.[14,19] The weight loss so frequently encountered in this condition may be a consequence of marked reduction of food intake in the avoidance of abdominal pain ("small meal syndrome"), in addition to or instead of frank malabsorption of

nutrients. On the other hand, ischemic malabsorption may not necessarily be accompanied by abdominal pain, as seen in the small vessel disease of periarteritis nodosa.[6]

Malabsorption has been classified into abnormalities of intraluminal digestion, mucosal-cell transport, and lymphatic drainage of the intestine.[35] The exact mechanism for the impaired absorption associated with vascular disease is unknown. Many factors affecting both intraluminal and mucosal phases of digestion are probably involved, including: (1) decreased mucosal cell turnover and villous atrophy with diminished absorptive surface; (2) reduced enzyme production; and (3) altered bowel motility as a result of submucosal fibrosis or autonomic nerve damage with consequent stasis and bacterial overgrowth. Complicating factors must also be considered such as stricture formation with the so-called blind-loop syndrome, decrease in pancreatic enzyme production from coincident hypoperfusion of the pancreas, and localized damage to the terminal ileum with impaired bile salt and vitamin B_{12} absorption.

A spectrum of laboratory tests is performed in the routine evaluation of malabsorption, including blood cell count and smear, prothrombin levels, serum calcium, albumin, carotene and vitamin A. Each of these, however, is nonspecific and unreliable as a screening test. The documentation of the presence of a malabsorption syndrome is generally predicated on the finding of an abnormality in the I^{131} triolein absorption, 72 hour stool fat, d-xylose excretion, and/or radioactive vitamin B_{12} absorption (Schilling) tests. The triolein test has been shown to be a useful screening test for steatorrhea with few false positives.[27] Fecal fat determination is the most sensitive and quantitative assessment of fat malabsorption.[28] Both tests, however, supply no etiologic information nor do they distinguish between the 3 main groups of malabsorptive disorders. The determination of xylose and vitamin B_{12}, while dependent on jejunal and terminal ileal mucosal-cell transport, respectively, may be similarly misleading since each may also be metabolized by overgrowth of luminal bacteria in a variety of conditions.[11] Thus, in mesenteric vascular insufficiency, routine laboratory evaluation may document the presence of malabsorption, but gives no clue to its ischemic origin.

Jejunal biopsy for the direct examination of the histopathology is also nonspecific in most instances. The changes in the small bowel mucosal histology classically associated with idiopathic sprue, consisting of total or subtotal villous atrophy and chronic inflammatory cell infiltration in the submucosa,[7] are by no means pathognomonic. Indeed these findings are a feature of many specific conditions,[36] including gluten-responsive sprue, tropical sprue, lymphoma, bacterial overgrowth, dermatopathic enteropathies, and radiation enteritis. Similar histologic changes have been noted in ischemic malabsorption. Watt *et al.*[37] described a patient with subtotal villous atrophy and plasma cell infiltration in the lamina propria which regressed entirely following revascularization. Carron and Douglas[6] have shown atrophic mucosal changes in patients with steatorrhea secondary to vascular insufficiency of the small intestine produced by periarteritis nodosa. Mucosal atrophy has also been produced following experimental partial ligation of the superior mesenteric artery in dogs.[26] In addition, the presence of submucosal fibrosis associated with malabsorption[13] probably indicates, as in our case, mesenteric vascular insufficiency of some duration. It is thus apparent that small bowel mucosal biopsy cannot distinguish between idiopathic sprue and ischemic malabsorption, with the exception of a truly flat mucosal lesion (total villous atrophy) which is much more consistent with sprue.

Small bowel barium studies are utilized in the routine evaluation of malabsorption, but are generally most specific in delineating localized lesions such as di-

verticula, strictures, enteric fistulas, and conditions such as regional ileitis, Whipple's disease, and intestinal lymphangiectasia.

The early reports of roentgenologic abnormalities in the small bowel in idiopathic steatorrhea emphasized a characteristic "deficiency pattern" based on flocculation and segmentation of a simple barium sulphate suspension. Frazier *et al.*[12] documented that such changes are related to the quality of the intestinal contents and not a reflection of morphologic changes or disordered motor function. It was shown that segmentation and flocculation could be induced in normal subjects by the presence of excess fatty acids, hypertonic solutions and mucus in the small bowel. In the presence of these changes in the contrast material, the caliber and mucosal pattern of the small bowel loops could not be accurately evaluated. It was not until the development of colloidal barium preparations, now in general use, that clumping and precipitation of the barium could be avoided[2] and the primary characteristic features recognized. These include dilatation of the small bowel, particularly in the mid- and distal jejunum, and evidence of increased intraluminal fluid as manifested by progressive dilution of the barium column within the ileum.[17] The features of dilatation and hypersecretion are virtually always present in the active phase of the disease.[17] Transit time through the small bowel may be normal or delayed. The mucosal folds may appear normal, thickened or thinned, depending on the degree of dilatation and the amount of secretion. Short transient nonobstructive intussusceptions may occasionally occur.[31] The exact cause of the small bowel dilatation is not clear but, since there appears to be some correlation between the degree of jejunal dilatation and the severity of the steatorrhea, it may be secondary to the increased bulk of intestinal contents.[16] Diminished serum potassium may also play a role in inducing paralytic ileus in malabsorption.

Descriptions of the roentgenologic abnormalities in the small bowel in reported cases of malabsorption secondary to visceral ischemia are scanty or imprecise. They include: "some tendency to delay in emptying of the proximal jejunum"[21]; "decreased peristalsis with an irregular mucosal pattern"[15]; "a malabsorption flocculation pattern"[37]; "puddling of barium in the small intestine"[8]; and "segmentation of the barium column occurring more proximally than in the usual case of malabsorption."[33] On occasion, a normal small bowel series has been reported,[34] but of interest is the description of a "small bowel deficiency pattern" in a case of abdominal angina without documented malabsorption.[5]

Our case and others previously illustrated do show several roentgenologic features distinct from the classical findings in idiopathic sprue which might be helpful in the differentiation of ischemic malabsorption. The degree of dilatation is relatively mild and tends to be confined to the proximal jejunal loops in contrast to the mid- and distal jejunum in sprue. While over-all transit time may be within normal limits, definite stasis is present within the dilated jejunum. No hypersecretion is seen and segmentation of the barium column, while present to a mild degree, is not a conspicuous feature. Disordered peristalsis may, as in idiopathic sprue, be further reflected by short transient intussusceptions. It would thus appear that the roentgenologic findings in the small bowel may be more directly a consequence of the ischemia than of the resultant malabsorption. These roentgenologic features may be helpful in selecting patients with malabsorption for aortography.

Severe mesenteric vascular involvement occurs almost exclusively with advanced aortic atherosclerosis,[29] with the most extensive and common stenosis extending from the orifice over the proximal 1.5–2.0 cm.[9,10,29] Lateral aortography thus allows definitive delineation of involvement of the orifices and main stems of the celiac and superior mesenteric arteries. This information of the extent and distribution of the

arteriosclerotic occlusions is of crucial importance in the decision and choice of a revascularization procedure.

Because of the rich anastomoses, it has been generally accepted that at least 2 of the 3 visceral arteries must be involved to result in ischemic symptoms. The most constantly involved vessel has been the main stem of the superior mesenteric artery. It is apparent that factors other than the extent and degree of arteriosclerotic occlusion of the mesenteric vessels play a role in the production of ischemic malabsorption. Indeed, Reiner *et al.*[29] have emphasized the pitfalls in transposing their postmortem observations directly to a clinical setting. Paramount among these are: (1) acuteness or chronicity of occlusion, and the development of collateral circulation. Complete arteriosclerotic stenosis of all 3 mesenteric vessels without ischemic signs or symptoms has been described,[20] with intestinal blood supply maintained *via* rich collateral branches; (2) degree of occlusion of the distal vessels. Occlusive lesions of the small mesenteric arteries have been shown in several cases of intestinal ischemia.[1,3] These include: intimal fibrous hyperplasia, medial thinning, elastosis and hypertrophy, and periarterial fibrosis; and (3) other conditions leading to a low perfusion state.[25,32]

CONCLUSIONS AND SUMMARY

Malabsorption may be caused by chronic mesenteric ischemia.

It may be the initial or the dominant feature of the clinical presentation, with the characteristic postprandial pain of "abdominal angina" not appearing until later in the course. In contrast to other malabsorptive disorders, this condition is distinguished by an onset after the age of 50 years in a patient with evidence of generalized arteriosclerotic disease. An abdominal bruit is also commonly present.

The mechanism of impaired absorption is complex and probably related to abnormalities of intraluminal and mucosal phases of digestion. Both absorption tests and jejunal mucosal histology are useful in documenting the presence of a malabsorptive disorder, but often do not distinguish either its mechanism or underlying etiology.

A small bowel series demonstrating the features of mild dilatation of *proximal* jejunal loops with stasis, in the absence of hypersecretion and significant segmentation, is helpful in the differentiation of ischemic malabsorption from idiopathic sprue. These roentgenologic features may be of further assistance in selecting patients with malabsorption for aortography to define the presence and extent of potentially remediable vascular occlusions.

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VOL. 119, NO. 2

GARDNER'S SYNDROME*

A MODEL FOR CORRELATIVE RADIOLOGY

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IN 1721 Menzel first described adenomatosis of the colon. Virchow further considered multiple colon polyposis in 1863. Cripps first noted the heritable quality of colon polyposis and named it familial adenomatosis. Fitzgerald[5] in 1943 reported a case of a 39 year old Italian woman with multiple rudimentary teeth and odontomas: frontal, sphenoidal, mandibular and long bone osteomas all combined with rectal bleeding due to colon adenomatous polyps. No family members were evaluated in this report.

In 1951 Gardner[7] evaluated 5 generations of a family with familial adenomatosis, particularly as this was related to the development of carcinoma. He noted that this family also had evidence of hard tumors of the head and face, and soft tumors of subcutaneous tissues. He was unable to further study the associated tumors in this first report.

Gardner and Plenk[8] further studied the family with polyposis and obtained skull and face roentgenograms of 6 patients with polyposis. Each of these patients had osteomas of the mandible, maxilla, frontal or other cranial bones. Cutaneous and subcutaneous lesions in this family were reported by Gardner and Richards[9] in 1953 as epidermoid cysts, fibromas and ill-defined masses of fibrous connective tissue. These investigators concluded that the combination of familial intestinal polyposis, osteomas and fibrous or epithelial tumors represented a hereditary pattern transmitted as a mendelian dominant trait and postulated a single pleiotropic gene or two closely linked genes to account for their data.

Since these reports, familial intestinal polyposis combined with long and flat bone osteomas and a variety of cutaneous and subcutaneous tumors has become known as Gardner's syndrome. Familial polyposis may exist without other specific features and must be considered as purely entodermal abnormality. Gardner's syndrome is a lesion of all 3 germinal layers. Several excellent reviews of the general features of Gardner's syndrome appeared in the literature.[3,13,15]

The following cases of Gardner's syndrome are reported to illustrate the unique features of the patients and show how the radiologist may serve to correlate the features of Gardner's syndrome for the various clinicians.

REPORT OF CASES

CASE 1. C.M., a 17 year old black male, had been treated for multiple compound odontomas and impacted teeth for 5 years prior to admission. He presented with a mandibular mass.

Family History. The patient's mother had intestinal polyps on a previous barium enema examination done for rectal bleeding. The maternal grandfather died with an intraabdominal malignancy, presumably carcinoma of the colon, 20 years ago. Two siblings aged 13 and 14 were alive and well.

Roentgenographic Findings. Paranasal sinus and mandible roentgenograms were first obtained. These illustrated multiple odontomas, unerupted teeth, the mandibular osteoma and osteomas of the ethmoid sinuses. Such findings suggested Gardner's syndrome and further roentgenographic study was obtained. Skull roentgenograms showed a mottled osteoblastic nodularity of the calvarium. A long bone survey demonstrated osteomas of the humerus, femurs and ribs. Conventional barium enema examination and air contrast study revealed numerous polyps. The roentgenographic features are illustrated in Figures 1, *A–D*, and 2, *A* and *B*.

Several areas of epithelial cysts were found

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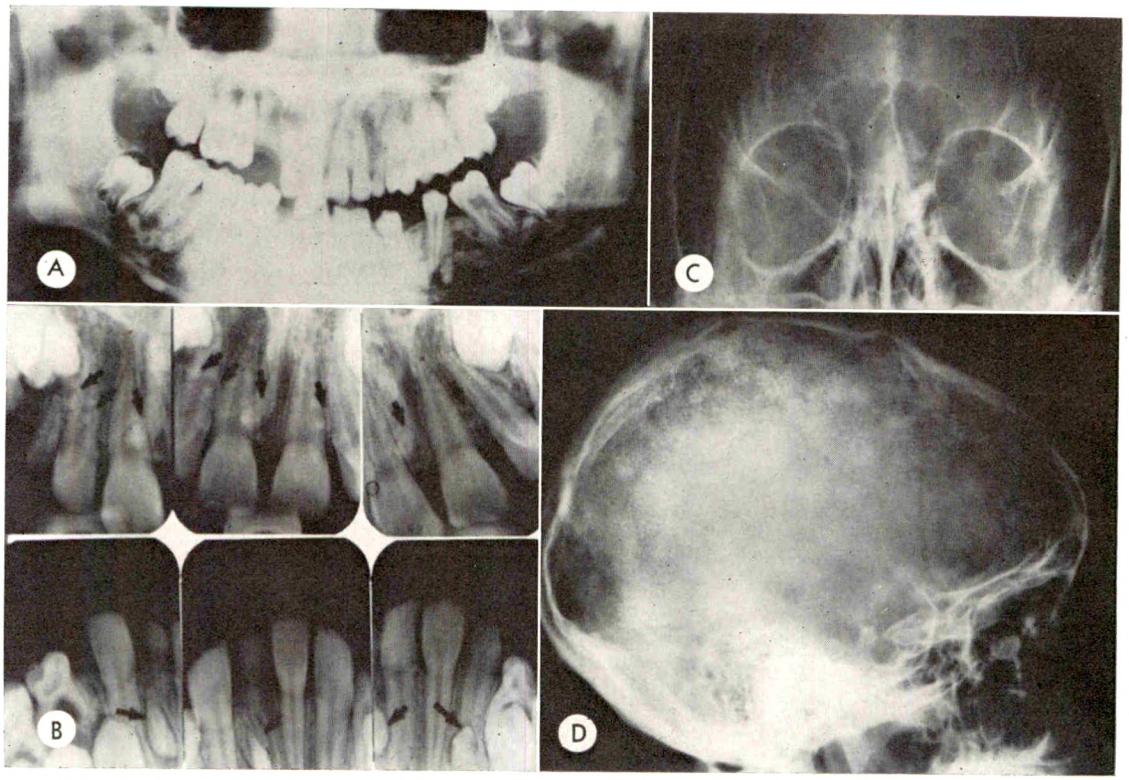


FIG. 1. Case 1. (*A*) Panoramic view of the mandible showing an osteoma along the right horizontal mandibular ramus. Odontomas can be seen near the roots of the mandibular molars bilaterally. (*B*) Apical views of the incisors. Odontomas are indicated by arrows. (*C*) Caldwell view of skull showing osteomas in the left frontal and left ethmoidal sinuses. (*D*) Lateral view of the skull. The calvarium has a "nodular" appearance.

on the patient's back and arms. Sigmoidoscopy also revealed the polyps and biopsy showed adenomatous change. Total colectomy with ileorectal anastomosis was performed. A total of 20 adenomatous polyps was found in the surgical specimen.

Comment. This case represents the third reported instance of Gardner's syndrome among blacks. Only 2 other occurrences among blacks have been reported by Weary *et al.*[15] when they reviewed 118 cases found in the literature.

The patient had a combination of intestinal polyposis, osteomas and dental abnormalities with cutaneous tumors. This is the most common combination in Gardner's syndrome. The radiologist served to alert the oral surgeon and gastroenterologist to the presence of Gardner's syndrome.

CASE II. C.L., a 15 year old white girl, was referred to this hospital complaining of a mandibular mass which was first noted at age 9. It had been asymptomatic until 15 months before admission when the onset of sharp pains in the lesion began. A past history of black, tarry stools on one occasion prior to admission was elicited.

Family History. The patient's mother had rectal polyposis at age 23. Several "small, benign tumors" were removed from her foot at the same time. She underwent a total colectomy at age 33 following the death of a sister due to colon carcinoma. The patient's maternal grandmother had died of a "liver cancer" (possible carcinoma of the colon with metastasis?).

Roentgenographic Findings. A 1.5 cm. mass was present at the left mandibular angle. A similar, but smaller, mass was found in the middle of the right vertical mandibular ramus. No cutaneous lesions were present. Roentgenographic studies demonstrated the presence of mandibular osteomas. A large osteoma was also present in the left ethmoid sinus. A long bone survey was normal. Air contrast barium enema examination revealed several sigmoid polyps. These changes are shown in Figure 3, *A–C*.

At sigmoidoscopy a 3 mm. polyp with multi-

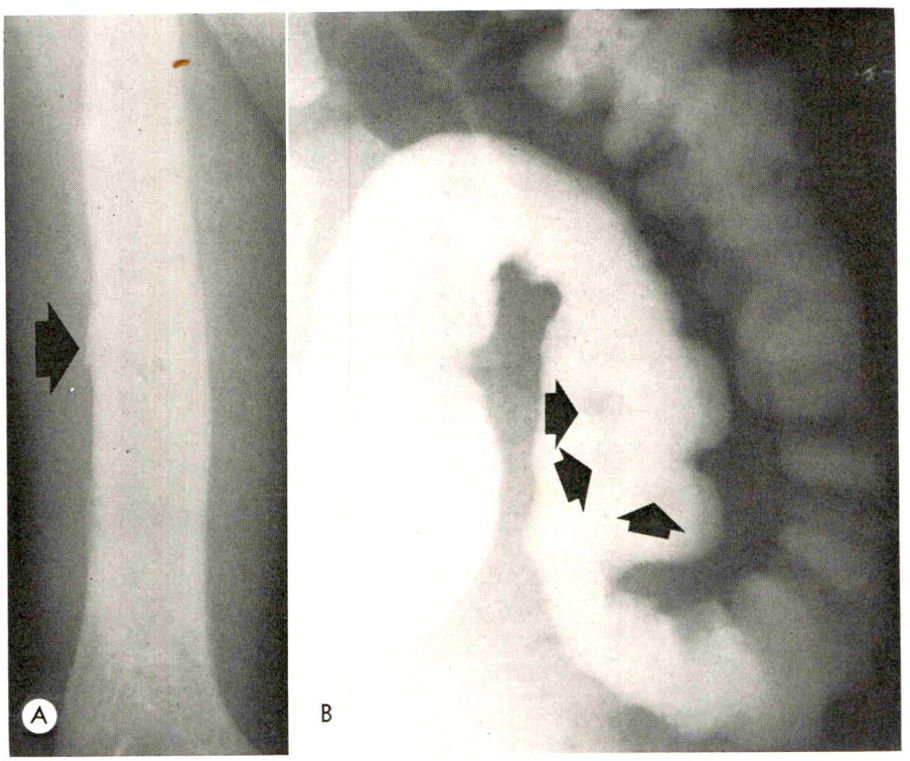


Fig. 2. Case 1. (*A*) Anteroposterior view of the right humerus with a marginal osteoma in the lateral diaphysis (arrow). (*B*) Spot roentgenogram of the sigmoid colon showing several polyps in the proximal portion (arrows).

focal smaller areas of adenomatous change were found and confirmed by biopsy. The mandibular angle mass was surgically removed. A subtotal colectomy is planned.

Comment. Fuhrmann *et al.*[6] postulate the possibility of several forms of Gardner's syndrome including: an osteocutaneous type; a cutaneointestinal type; and an osteointestinal type. They were not able to find any examples of the osteointestinal type. This patient seems to qualify as an example of osteointestinal form of Gardner's syndrome. She may at a later age develop cutaneous or subcutaneous lesions, but at this time is free of such lesions.

Since she is living in a foster home we are unable to study any siblings. Her mother seems to have had the osteocutaneous type of Gardner's syndrome.

DISCUSSION

The radiologist is in a unique position to recognize the osseous changes of Gardner's syndrome and then perform the colon survey to uncover polyposis.

OSTEOMAS

Osteomas, particularly those in sinuses and mandible, should strongly suggest the possibility of accompanying polyposis. Eckel and Palm[4] reviewed the paranasal sinus roentgenograms of 16,000 patients and found 74 osteomas. Only 8 osteomas were associated with facial deformity or produced symptoms. No specific effort was made to relate colon polyposis to the sinus osteomas in this group.

Weary *et al.*[15] report that 50 per cent of patients with Gardner's syndrome had osteomas: 48.2 per cent were located in the skull and sinuses; about 40 per cent in the frontal sinuses; 25 per cent in the ethmoid sinuses; 10 per cent in the maxillary sinuses; 5 per cent in the nasal cavity; and 1.5 per cent in the sphenoid sinuses.

About 20 per cent of Gardner's syndrome patients have mandible osteomas. Many of these produce jaw masses that are

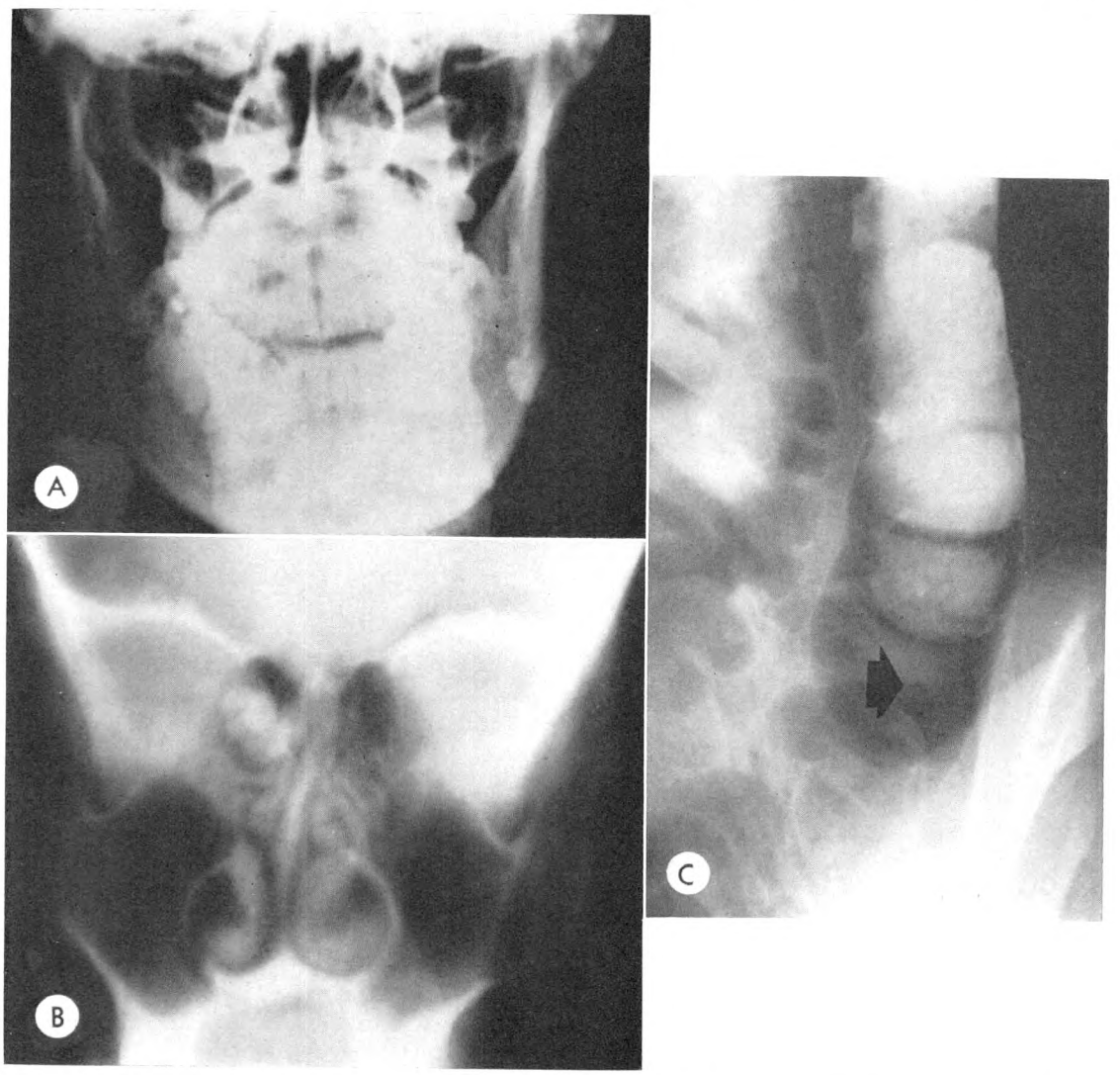


FIG. 3. Case II. (*A*) Anteroposterior view of the mandible. An osteoma is present at the left mandibular angle. (*B*) Hypocycloidal tomogram showing an osteoma in the right ethmoidal sinus. (*C*) Air contrast study showing polyp in the distal descending colon (arrow).

palpable. Several authors report recurrence after excision of the mandibular osteoma.

Osteomas of the ribs and long bones may be found. Some are well-defined masses while others are only ill-defined local areas of cortical thickening.

Colon examination, especially air contrast enema study and sigmoidoscopy, should be performed on patients with skull, sinus, mandible, rib and long bone osteomas. The likelihood of intestinal polyposis would increase in those with multiple osteomas.

DENTAL ABNORMALITIES

Cystic odontomas, composite odontomas, supranumerary and multiple unerupted teeth, follicular odontomas and dentigerous cysts should all suggest the possibility of Gardner's syndrome. Search for osteomas should be made. If osteomas coexist with the above dental abnormalities, the colon should be evaluated, although we feel that

odontomas should be sufficient indication for colon examination.

SOFT TISSUE TUMORS

Radiologists may occasionally see a cutaneous or subcutaneous mass altering the margin of chest, neck or abdomen. In general, the dermatologist, general surgeon or internist would more likely be asked to evaluate the epidermoid cysts, fibromas or desmoid tumors accompanying Gardner's syndrome.

Mesenteric fibromatous masses may occur after resection of the colon for polyposis.[2] Desmoid tumors have also been found in the surgical scar of patients with Gardner's syndrome.

POLYPOSIS

Peutz-Jeghers syndrome is characterized by the presence of hamartomas of colon mucosa, while familial polyposis produces adenomatous proliferation. The relationship between familial polyposis and carcinoma is well established. Mayo *et al.*[12] reported that those with familial polyposis will either be dead with carcinoma of the colon or have an advanced neoplasm by the age of 50. Bochetts *et al.*[1] related that polyps usually appear by age 20, but have been discovered as early as 2 to 4 months or as late as age 70. They further stated that malignancy has its onset some 15 years after the onset of polyposis, so that the average patient is between 35–40 years when the neoplastic features of familial polyposis begin.

It seems reasonable to assume, therefore, that different aspects of Gardner's syndrome arise at various time periods during the patient's life. Duncan *et al.*[3] found in their study that 80 per cent (66 of 83 patients) had 1 manifestation of Gardner's syndrome before age 30; 88 per cent (73 of 83 patients) had 2 manifestations before age 40; and concluded that patients over age 40 had a good chance of being disease free.

On the basis of this information, one would suspect that the onset of polyps, osseous changes and soft tissue changes would have dissimilar appearance time in various individuals. This may explain why Fuhrmann *et al.*[6] were able to find 5 individuals in generations of a family with osseous and cutaneous lesions of Gardner's syndrome, but without polyposis. Dissimilar development of Gardner's syndrome is further demonstrated by a pair of identical twins studied by Gorlin.[10] One patient at 28 years of age had osseous and soft tissues tumors, polyposis and adenocarcinoma, while the other twin had only mandibular osteomas.

Shiffman[13] related a study of familial polyposis in which 30 per cent of 90 cases would qualify as Gardner's syndrome. It seems likely that many cases initially found to represent familial polyposis are not further studied in regard to the possibility of osseous or soft tissue abnormalities.

CONCLUSION

We feel that there are several unanswered questions resulting from the material presented which makes Gardner's syndrome a model for further correlative study. The relationship between certain osseous, dental, soft tissue abnormalities and adenomatous polyposis seems certain. The relationship between familial polyposis and carcinoma of the colon is equally certain.

Perhaps, there is an equally frequent association, individually, between osteomas, the above described dental abnormalities, the soft tissue tumors and colon polyposis. Also, this may represent a large enough group of individuals and families to constitute a significant group of colon cancer suspects.

Therefore, we recommend the following procedure: patients with a single osteoma of skull, mandible, sinus, ribs or long bones should have an air contrast enema examination and sigmoidoscopy. Patients with multiple unerupted teeth, odontomas, or dentigerous cysts should also have a colon examination. Similarly, a group of patients with epidermoid cysts, subcutaneous fibro-

mas or desmoid tumors should have a colon examination.

In order to obtain significant numbers of patients, this undertaking would probably have to be a multiple-institution cooperative program. Such a program would serve to evaluate the possibility of correlative criteria which might be useful in finding patients with familial polyposis and possible colon neoplasms.

Furthermore, it would be interesting to study the skull, sinuses, mandible, ribs, long bones, skin and subcutaneous tissue of a patient group with known colon adenocarcinomas. Perhaps some relationship between ectodermal, mesodermal and endodermal abnormalities might also be uncovered in this group.

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TWO CASES OF FAMILIAL POLYPOSIS SIMULATING LYMPHOID HYPERPLASIA*

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THE differentiation between lymphoid hyperplasia of the colon and familial polyposis is extremely important, as the significance of these conditions and the management of patients differ entirely. Lymphoid hyperplasia is generally considered to be a benign condition, which probably represents the normal response of lymphoid tissue to a variety of stimuli;[6] therefore, no specific treatment is needed. Familial polyposis of the colon is a premalignant condition and requires a proctocolectomy.

Lymphoid hyperplasia has been characterized roentgenographically by innumerable small, uniform, polypoid lesions involving all or part of the colon. A fleck of barium is seen in the center of the polyp, representing umbilication at the apex of the lymphoid nodule. This finding has been stated to be unique for lymphoid hyperplasia. It has also been reported that such umbilication has not been observed in multiple polyposis of the colon in children and that its presence permits a specific roentgenographic diagnosis.[6]

The purpose of this communication is to present 2 cases of multiple polyposis of the colon, occurring in the same family, possessing roentgenographic features that have only been described in cases of lymphoid hyperplasia of the colon. The significance of these cases in terms of the differential diagnosis of multiple, small polypoid lesions of the colon and their management will be discussed.

REPORT OF CASES

CASE I. J.D., a 10 year old white female with a family history of multiple members developing carcinoma of the colon. At the age of 8 years, she developed diarrhea mixed with blood. A barium enema examination on February 9, 1971, revealed multiple, small, nodular, polypoid lesions throughout the colon with a central fleck of barium seen in practically every area. Compare findings with a proven case of lymphoid hyperplasia (Fig. 1, *A–C*; and 2, *A–C*). Proctoscopy revealed numerous small polyps, and a proctocolectomy was performed. The pathologic diagnosis was polyposis of the colon, revealing a multitude of tiny polyps, and no evidence of lymphoid hyperplasia. The largest polyps were 0.3 cm. and most were 0.1 to 2 cm. (Fig. 3).

CASE II. L.D., a 16 year old sister of the first case, had a history of weight loss, fatigue and diarrhea with some bleeding. The patient was proctoscoped and had a barium enema examination in 1966, at which time multiple, small, polypoid lesions were discovered (Fig. 1, *A–C*; and 2, *A–C*). On November 19, 1971, a proctocolectomy was performed and the pathologic diagnosis was polyposis of the colon. The largest polyps were 0.6 cm. and most were 0.3 cm.

DISCUSSION

Considerable information concerning concepts with regard to etiology, pathogenesis and significance of lymphoid hyperplasia has appeared in the literature.[1,2,6,7,9,10] A discussion of the roentgen findings in familial polyposis with emphasis on differential diagnosis has also been reported.[8] Lymphoid hyperplasia is a benign lesion which does not undergo malignant changes, and it is extremely important that an awareness of this entity exist so as not to confuse this condition with multiple polyposis of the colon. The lesions of lymphoid hyperplasia may regress without treatment in some cases. However, total colectomies

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Fig. 1. (*A*) Case I. Multiple nodular defects are demonstrated with a central fleck of barium seen throughout the colon. (*B*) Case II. The nodular defects in the colon are revealed. (*C*) Compare *A* and *B* with this proven case of lymphoid hyperplasia of the colon.

have been performed on at least 2 patients with lymphoid hyperplasia who were erroneously diagnosed as having multiple polyposis of the colon.[3,4] In other instances, less extensive bowel resections have been performed in patients with lymphoid hyperplasia who were misdiagnosed preoperatively.[5,11]

We feel that as a result of the roentgenographic findings in our cases of multiple

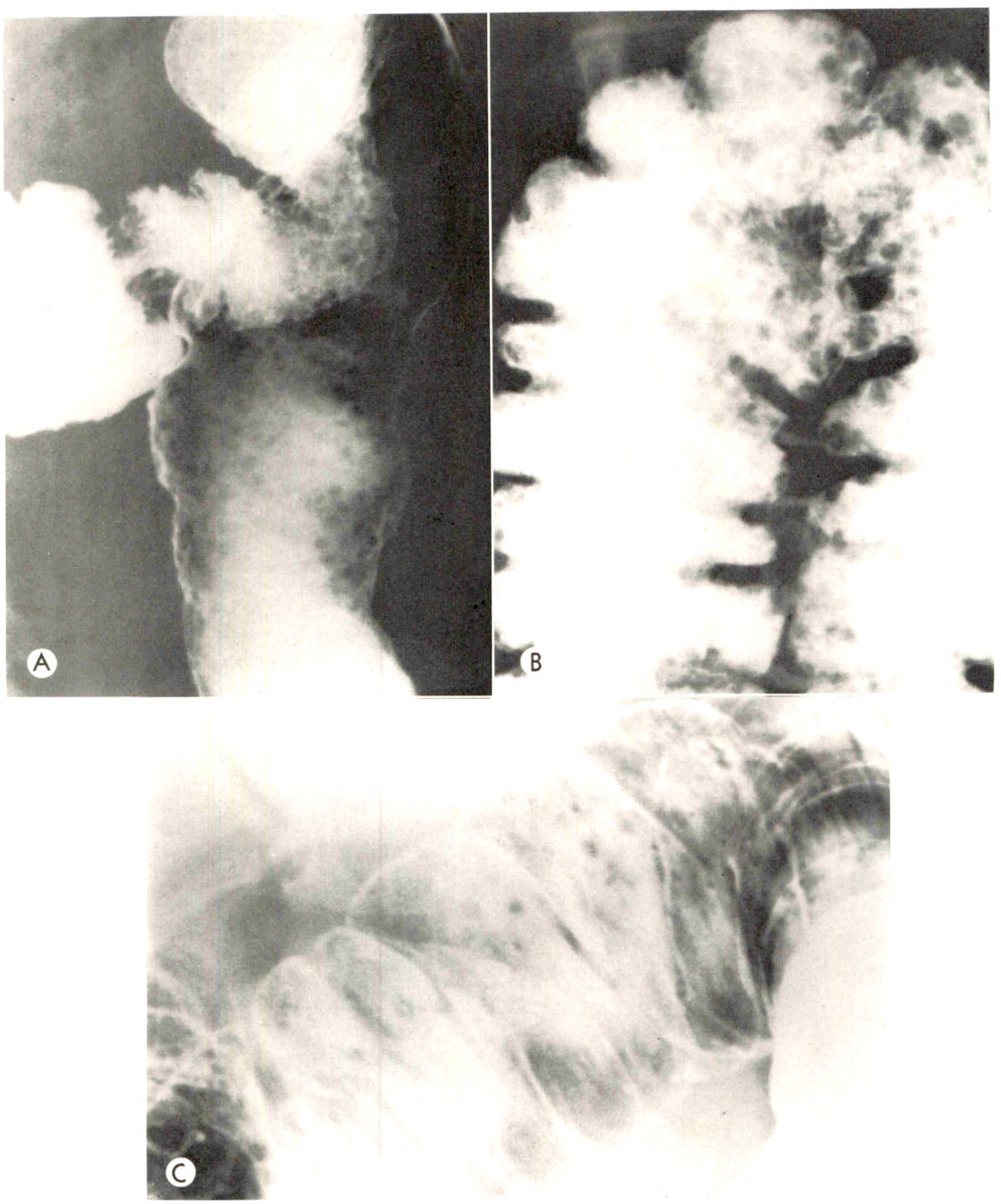


FIG. 2. (*A*) Figure 1*A* magnified to show the nodular lesions with the central fleck of barium. (*B*) Figure 1*B* also magnified to show individual lesions more clearly. (*C*) Close-up view of lymphoid hyperplasia. Compare Case I and Case II of familial polyposis of the colon.

polyposis with small nodular lesions and central umbilication, it is important to be aware of the converse, and not diagnose lymphoid hyperplasia instead of a much more serious disease process.

CONCLUSIONS

1. The differentiation of lymphoid hyperplasia of the colon and multiple polyposis is extremely important, as the signifi-

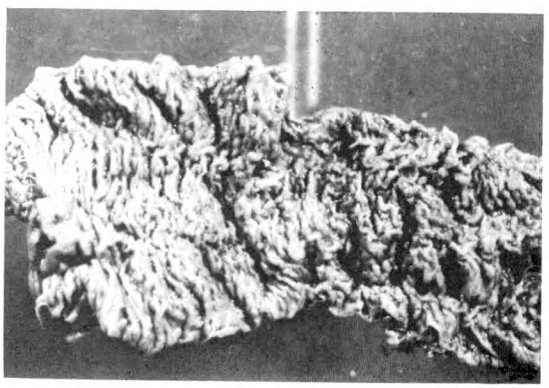


Fig. 3. The colon specimen of Case 1. Note the very small uniform polypoid lesion measuring 0.1 to 0.3 cm.

cance and management of these conditions are entirely opposed.

2. The findings of small, uniform, umbilicated polypoid lesions involving all or part of the colon are characteristic in patients with lymphoid hyperplasia but not pathognomonic.

3. Two cases of familial polyposis are presented with roentgenographic findings previously only described in patients with lymphoid hyperplasia.

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ULCERATIVE AND GRANULOMATOUS COLITIS*

VARIATIONS IN OBSERVER INTERPRETATIONS AND IN ROENTGENOGRAPHIC APPEARANCE AS RELATED TO TIME

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THE overlap in clinical and roentgenographic findings of ulcerative and granulomatous colitis has been recently emphasized by Glotzer *et al.*,[2] Margulis *et al.*,[4] and Schachter *et al.*[5] As the 2 diseases progress, changes in their roentgenographic pattern may also contribute to the diagnostic confusion. We decided, therefore, to determine if radiologists who specialize in gastrointestinal disease could correctly differentiate these 2 forms of colitis when the set of films from each of several roentgenographic examinations of patients with pathologically proved granulomatous colitis and ulcerative colitis were viewed and presented as separate cases. With 3 radiologists participating, we wanted to study inter-observer variation[1] and, by having each radiologist reread several of the same sets of films at a later session, intra-observer variation was to be ascertained. Finally, when results were recorded and the films were again analyzed, we wished to determine which roentgenographic findings most commonly led to correct or to incorrect diagnosis.

MATERIAL AND METHOD

From the series of colitis cases previously reported by Margulis *et al.*,[4] we selected 10 cases of pathologically proved granulomatous colitis in which the patients had had a total of 32 roentgenographic examinations. The basis of selection was that each patient had at least 1 examination in which roentgenographic interpretation was difficult. The 32 sets of roentgenograms were first separated and any identifying data masked. Each set was then put in a separate jacket to be presented as a single unknown case of colitis. The "cases" were next studied by 3 gastrointestinal radiologists, who were to determine if they represented granulomatous colitis or ulcerative colitis.

Approximately 3 months after the analysis of the first series of cases of granulomatous colitis, a second series was assembled. Eleven cases of pathologically proved ulcerative colitis in which the patients had had several barium enema examinations were selected. The criterion again was that at least 1 of the examinations of each patient yielded an atypical roentgenographic pattern. This group of 11 cases also comprised a total of 32 roentgenographic examinations. Each set of films from these examinations also had identifying data masked and was then packaged as a separate case. The 32 envelopes of ulcerative colitis films were next randomly interspersed with 18 of the sets of granulomatous colitis films used in the initial series. This second series thus consisted of films of 50 "unknown" cases of colitis—18 of which had actually already been analyzed by the 3 radiologists. This second series of roentgenograms was given to the 3 radiologists to be interpreted as either granulomatous colitis or ulcerative colitis. After the second series of 50 sets of roentgenograms had been read, 2 of the investigators (JAN, ARM) reviewed each analysis made by the 3 radiologists to determine which findings led to correct and which to incorrect diagnosis.

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TABLE I

INTERPRETATIONS BY 3 GASTROINTESTINAL RADIOLOGISTS OF 32 ROENTGENOGRAPHIC EXAMINATIONS MADE AT VARIOUS STAGES OF PATHOLOGICALLY PROVED GRANULOMATOUS COLITIS

Radiologist	Ulcerative Colitis	Granulomatous Colitis
A	14	18
B	23	9
C	7	25

TABLE II

INTER-OBSERVER AGREEMENT ON INTERPRETATIONS OF 32 ROENTGENOGRAPHIC EXAMINATIONS FIRST SERIES (GRANULOMATOUS COLITIS)

Radiologist	Agreement (per cent)
A and B	72
B and C	50
A and C	66

It is important to note that the 3 radiologists used techniques of interpretation different from one another. One relied on his experience and general expertise. Another radiologist analyzed each case with a check list of differential findings considered typical of granulomatous *versus* ulcerative colitis.[2–4] The last radiologist analyzed the series with residents in Radiology, as a teaching exercise.

The time span covered by the several roentgenographic examinations given each patient varied from 1 month to 8 years and 3 months; the average period was 36 months.

TABLE III

SECOND SERIES

SUMMARY OF INTERPRETATIONS OF 50 ROENTGENOGRAPHIC EXAMINATIONS: 32 PROVED ULCERATIVE COLITIS AND 18 PROVED GRANULOMATOUS COLITIS

Interpretation	Observer		
	A	B	C
Ulcerative colitis called granulomatous colitis	11	8	8
Granulomatous colitis called ulcerative colitis	8	10	9
Granulomatous colitis correctly diagnosed	10	8	9
Ulcerative colitis correctly diagnosed	21	24	24

Unanimous Interpretations

Interpretation	Cases
Ulcerative colitis called granulomatous colitis	3
Granulomatous colitis called ulcerative colitis	7
Granulomatous colitis correctly diagnosed	6
Ulcerative colitis correctly diagnosed	16
Total	32

Mixed Interpretations

Reading	Cases
2 correct and 1 incorrect (Of the 12 errors, 8 occurred when ulcerative colitis was called granulomatous colitis.)	12
1 correct and 2 incorrect (Of the 12 errors, 10 occurred when ulcerative colitis was called granulomatous colitis.)	6
Total	18

TABLE IV

INTER-OBSERVER AGREEMENT ON INTERPRETATIONS OF 50 ROENTGENOGRAPHIC EXAMINATIONS SECOND SERIES (32 ULCERATIVE COLITIS, 18 GRANULOMATOUS COLITIS)

Radiologist	GC Correct	UC Correct	GC called UC	UC called GC	Agreement
A and B	7/18 (39%)	19/32 (59%)	8/18 (44%)	4/32 (13%)	76%
B and C	6/18 (33%)	19/32 (59%)	8/18 (44%)	3/32 (9%)	72%
A and C	8/18 (44%)	18/32 (56%)	6/18 (33%)	5/32 (16%)	74%

GC = granulomatous colitis; UC = ulcerative colitis.

TABLE V

INTRA-OBSERVER VARIATION IN INTERPRETATION OF 18 ROENTGENOGRAPHIC EXAMINATIONS OF GRANULOMATOUS COLITIS ANALYZED A SECOND TIME, 3 MONTHS AFTER INITIAL READING

Radiologist	Diagnosis changed	Diagnosis correct on 1st reading	Diagnosis correct on 2nd reading
A	3/18 (16%)	11/18 (61%)	10/18 (56%)
B	2/18 (11%)	7/18 (39%)	7/18 (39%)
C	8/18 (44%)	15/18 (83%)	9/18 (50%)

RESULTS

FIRST SERIES

The roentgenographic interpretations of the initial series of 32 sets of films of proved granulomatous colitis are presented in Table I. Table II is a tabulation of inter-observer agreement for this series. There was unanimous correct diagnosis of granulomatous colitis in 9 instances and unanimous incorrect diagnosis of ulcerative colitis in 5.

SECOND SERIES

Table III shows the individual readings from the second series of 50 roentgenographic examinations and tabulates the

TABLE VI

FREQUENCY OF "TYPICAL" FINDINGS* IN CASES INTERPRETED UNANIMOUSLY (CORRECTLY OR INCORRECTLY) FOR GRANULOMATOUS COLITIS OR ULCERATIVE COLITIS FIRST AND SECOND SERIES

	Unanimous Reading of GC (Total of 13)		Unanimous Reading of UC (Total of 22)	
	GC correct (10)	GC called UC (3)	UC correct (16)	UC called GC (6)
Area of involvement				
Total colon	1/10	0/3	6/16	2/6
Terminal ileum	7/10	0/3	3/16	0/6
Cecum	8/10	0/3	7/16	2/6
Ascending colon	4/10	0/3	7/16	2/6
Transverse colon	7/10	0/3	8/16	6/6
Descending colon	8/10	1/3	12/16	6/6
Sigmoid	6/10	2/3	11/16	6/6
Rectum	2/10	2/3	11/16	6/6
Concentric involvement	7/10	2/3	11/16	6/6
Eccentric involvement	3/10	1/3	2/16	1/6
Ulcer type				
Shallow	8/10	0/3	12/16	4/6
Deep	5/10	0/3	4/16	5/6
Collar button	1/10	1/3	3/16	3/6
Pseudopolyps	1/10	0/3	5/16	1/6
Contracted colon	1/10	0/3	3/16	0/6
Megacolon	0/10	0/3	0/16	0/6
Gaping ileocecal valve	0/10	0/3	3/16	0/6
Focal involvement	0/10	0/3	0/16	0/6
Cobblestone mucosa	2/10	1/3	0/16	1/6
Fistula	2/10	1/3	0/16	0/6
Abscess	1/10	0/3	0/16	0/6

* Adapted from Table 1 of Margulis *et al.*, 1971.[4]
GC = granulomatous colitis; UC = ulcerative colitis.



Fig. 1. This smooth, shortened colon with a patulous ileocecal valve led to an interpretation of ulcerative colitis by all 3 radiologists. Histologic study showed the lesion to be ulcerative colitis.

number of unanimously correct and incorrect diagnoses. Table IV gives a summary of inter-observer agreement on the second series. It is apparent from this table that granulomatous colitis was agreed upon correctly less than half the time. Table V shows the intra-observer variation in interpretations of the 18 examinations of granulomatous colitis which were reread after a 3 month interval as part of the second series.

Each of the 50 examinations was next assessed for the presence or absence of the accepted roentgenographic findings in the 2 diseases,[4] which are listed in Table VI. Cases that had been interpreted unanimously, either correctly or incorrectly, by the 3 radiologists were then analyzed for each of these findings (Table VI). It was found that unanimous correct readings of granulomatous colitis were most often influenced by findings of terminal ileum disease, right-sided colonic disease, and deep ulcers. When it occurred, a demonstrated fistula led to correct diagnosis of granulo-



Fig. 2. (*A*) Barium enema examination demonstrates diffuse colonic disease with deep ulcers and an abnormal terminal ileum. This case was correctly diagnosed as granulomatous colitis by all 3 radiologists. (*B*) Roentgenogram of the same patient made 5 years after *A* shows a smooth colon with decreased haustration. This roentgenogram of a case of histologically proved granulomatous colitis was interpreted by all 3 radiologists to be ulcerative colitis.

matous colitis, but in 1 case of ulcerative colitis a fistula was misleading. Unanimous incorrect interpretations of ulcerative colitis as granulomatous colitis in 3 cases were difficult to account for. Criteria for unanimous correct and incorrect readings of ulcerative colitis were most often total or left-sided involvement, rectal disease, shallow ulcers, concentric involvement, and, occasionally, a totally contracted colon (Fig. 1).

ROENTGENOGRAPHIC CHANGES

After the films from the 64 examinations were reassembled into the original files of 21 cases, we reviewed each original case, in total, to determine if change in the roentgenographic pattern was evident as the disease progressed. No clear evidence of evolution of 1 roentgenographic pattern into an entirely different one was found. Over a time period extending from 1 month to 3 years, 3 of the 10 cases of granulomatous colitis with typical roentgenographic findings showed development of an ulcerative colitis pattern on the roentgenograms (Fig. 2, *A* and *B*), but these changes did not persist. In only 2 of the 11 cases of ulcerative colitis was there any opinion that the roentgenographic pattern had changes that represented granulomatous colitis (Fig. 3). This was noted by 2 of the 3 radiologists, but never was such a conclusion unanimous.



Fig. 3. Two of the 3 radiologists interpreted this case as granulomatous colitis. The pathological specimen proved it to be ulcerative colitis. Spasm and poor filling in the hepatic flexure probably contributed to the roentgenographic misinterpretation.

SUMMARY

This analysis of 21 cases of ulcerative and granulomatous colitis once again points out the overlapping patterns of the 2 pathologic processes. Data indicated that ulcerative colitis was diagnosed correctly more often than granulomatous colitis at this institution. The reliability of the various "classical findings" used as criteria at this institution may be questioned, since many such findings are apparently responsible for incorrect as well as correct diagnoses.

Inter-observer variation may well represent individual bias in this study; it was wide enough however to pinpoint the equivocal nature of some of the findings. The intra-observer variation (when the first reading of 18 sets of roentgenograms was compared with a second reading made 3 months later) was also substantial enough (11 to 44 per cent) to raise doubts about the possibility of achieving correct diagnosis strictly from barium enema examination. The findings in this study stress once again the necessity for basing diagnosis of a case of colitis on all its current and previous clinical roentgenographic and pathological data.[5] Even when all of this information is available, instances may occasionally arise in which definite diagnosis is in doubt or in which a previous diagnosis must be re-evaluated. Although most cases of ulcerative colitis and granulomatous colitis retain their roentgenographic characteristics throughout the course of the disease, there are sometimes situations in which one roentgenographic pattern evolves to another over an unpredictable period of time.

A study similar to this but using histologic material and highly trained patholo-

gists should be interesting and would contribute further to the understanding of the set of conditions surrounding granulomatous and ulcerative colitis.

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ROENTGENOLOGY OF INFECTIOUS COLITIS*

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INFECTIOUS colitis, a relatively common disease, has increased steadily over the past few years.[2,7,10] This increased incidence is related to the mass production and transport of meat and food products, with consequent susceptibility to infection because of exposure and inadequate refrigeration as well as poor hygienic conditions. In addition, many household pets are known to harbor and excrete *Salmonella*.[2] Since patients with infectious colitis present with diarrhea, some may undergo barium enema studies of the gastrointestinal tract. This is generally performed to substantiate an initial clinical impression of the commonly occurring colitides, especially ulcerative or granulomatous colitis.

We have, in this manner, had the opportunity to study the alterations of the colon on barium enema examination in 7 cases of colitis due either to *Shigella* or *Salmonella* organisms. A review of the literature has revealed no detailed description of the roentgenologic findings in this disorder.[8] Our observations in 7 cases uncovered changes that were not specific in themselves, but in many aspects resembled those encountered in ulcerative and granulomatous disease of the colon. Some differences were, however, noted which helped to distinguish these specific colitides from the latter nonspecific varieties. The roentgenologic features in our cases and their differential diagnosis form the basis of this report.

PATHOLOGY

The pathologic changes found in *Salmonella* and *Shigella* infections vary in location. The colon is predominantly involved in infections caused by *Shigella* organisms, whereas *Salmonella* mainly involves the terminal ileum. With severe infection, however, diffuse involvement of both large and small bowel occurs.[1]

The basic pathologic feature encountered in both lesions is mucosal necrosis followed by ulceration. These features have been readily identified in both the rectum and sigmoid by sigmoidoscopy.[11] The entire bowel wall becomes involved and edematous. In salmonellosis the primary infection spreads quickly *via* the blood stream as well as the lymphatic system. Lymphoid hyperplasia, therefore, forms an important part of the disease process. Consequently, there is enlargement of the Peyer's patches within the small bowel as well as the draining mesenteric lymph nodes. *Shigella* bacilli rarely invade the blood stream, although their toxins may act peripherally on the nervous system or the large joints.

CLINICAL AND ROENTGEN OBSERVATIONS

Shigella was the causative organism in 4 of our 7 patients and *Salmonella* in 3. All were adults who ranged in age from 21 to 56 years. The clinical history was usually that of an acute episode of diarrhea, abdominal cramps and vomiting, and the offending food substance was, on occasion, readily identified by the patient. In one instance, however, a patient (Case VI, Table 1) gave a history of recurrent diarrhea which failed to respond to conservative treatment following a summer vacation in Europe.

Two patients (Cases IV and VII, Table 1) had small intestinal contrast studies in addition to barium enema examination. One patient, infected with *Salmonella*, manifested diffuse changes which involved the duodenum and the entire small bowel.

* Presented at the Seventy-third Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 3–6, 1972.
From the Departments of Radiology, State University of New York-Downstate Medical Center, Brooklyn, New York;† and The New York Hospital-Cornell University Medical Center, New York, New York.‡

These appeared dilated, atonic and edematous (Fig. 1, *A* and *B*). Ulcerations were also present and the mucosa manifested a nodular appearance. These latter features are those usually caused by swollen edematous mucosa, but it is quite possible that enlarged lymphoid patches may have contributed as well. In the second patient who had a small bowel study, the examination was within normal limits.

The extent and degree of colonic involvement was variable. In 2 patients (Cases I and III, Table I) the colon was diffusely involved. In the one patient who manifested the markedly abnormal small bowel changes described above, the colon was atonic with loss of the normal haustral pattern (Fig. 2). There was excess fluid present in the bowel which diluted the barium solution. Our second patient with total colonic involvement demonstrated a dilated edematous colon. The changes were especially well seen in the cecum and ascending colon where the coarsened, thickened edematous folds were responsible for the corrugated appearance as well as the irregular margins of the bowel wall (Fig. 3). Superficial segmental ulcerations were present also in this patient.

The remaining 5 patients had segmental colonic involvement only. This pattern, however, differed both in location and

TABLE I

SUMMARY OF CASES

Case	Age and Sex	Organism	Small Intestine Involvement	Colon Involvement		Follow-Up
				Diffuse	Segmental	
I. W.V. (Fig. 4; 6, *A–C*)	30 M	*Shigella*	No study	—	Rectum, sigmoid and descending colon; collar button ulcerations	Normal
II. K.S.	22 M	*Shigella*	No study	—	Superficial ulcerations in rectum and sigmoid colon	Died
III. S.K. (Fig. 3)	56 F	*Salmonella*	No study	Edema; superficial ulceration in distal colon	—	Normal
IV. M.H. (Fig. 1, *A* and *B*; 2)	52 F	*Salmonella*	Ulceration, edema and dilatation of duodenum, jejunum and ileum; dilution of barium	Diffuse dilatation	—	Died
V. D.H. (Fig. 5, *A* and *B*)	33 F	*Shigella*	No study	—	Superficial and deep ulcerations involving rectum and descending colon	Normal
VI. E.K.	21 F	*Shigella*	No study	—	Edema of sigmoid colon	Normal
VII. M.O'M.	44 F	*Salmonella*	Normal	—	Ulceration in the descending colon	Normal

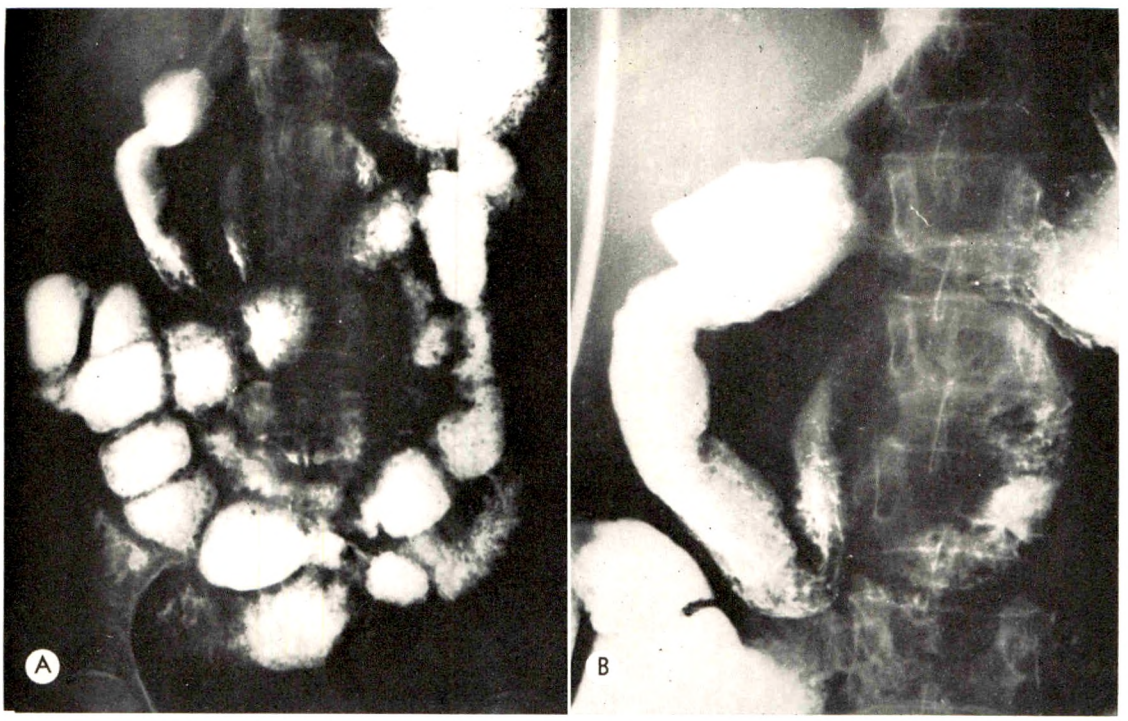
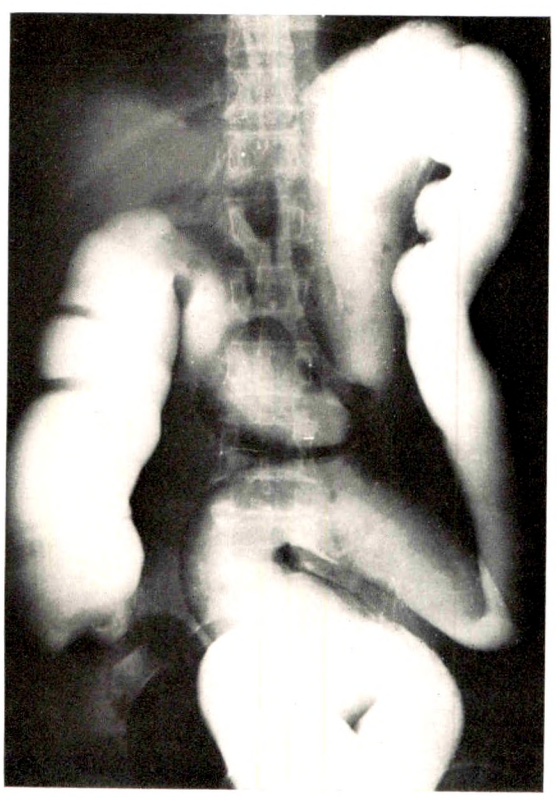


Fig. 1. Case IV. (*A*) Upper gastrointestinal and small bowel roentgenogram. Salmonellosis. The small bowel study shows edematous, dilated bowel. There is loss of the normal mucosal pattern and nodular filling defects are present. (*B*) Localized view of the duodenum. There is evidence of edema, ulceration, and nodular mucosa involving the duodenum and jejunum.



roentgenologic appearance. The proctocolitis which was present on occasion and extended in continuity to involve the sigmoid and descending colon was responsible for edema and ulceration as well as skip lesions which were variable in appearance. At times, the ulcers were superficial and diffuse, much like those seen in classical ulcerative colitis (Fig. 4), but on other occasions, the ulcers were deeper and asymmetric, resembling those visualized in Crohn's disease of the colon (Fig. 5, *A* and *B*).

The rectum was not always involved roentgenologically and skip areas of colonic involvement alternating with areas of normal bowel was a feature that was also

Fig. 2. Case IV. Barium enema roentgenogram. Diffuse colonic involvement with loss of the haustral pattern. Superficial ulcerations are present in the sigmoid.

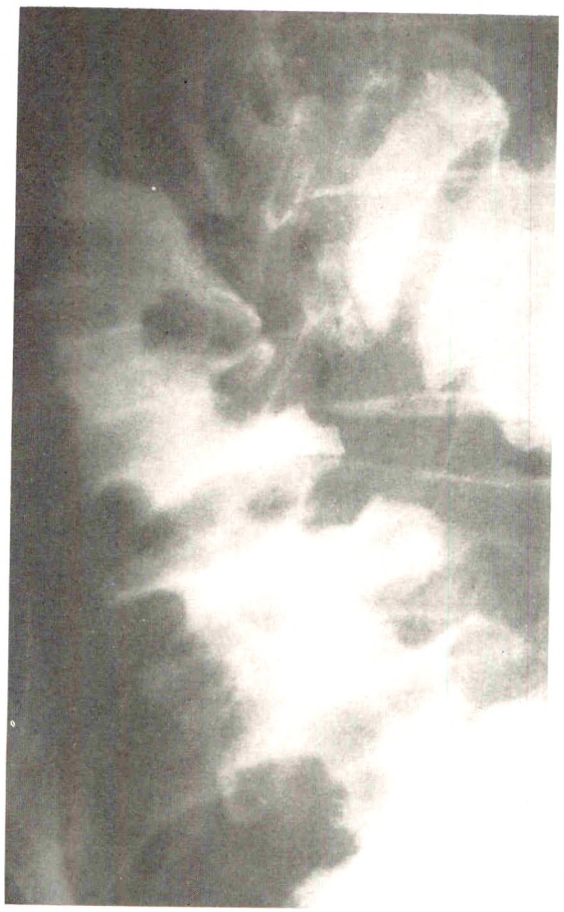


FIG. 3. Case III. Barium enema roentgenogram. Salmonellosis. Localized view of the hepatic flexure demonstrates the marked edema of the bowel.



FIG. 4. Case I. Barium enema roentgenogram. Shigellosis. Ulceration and edema of the rectum.

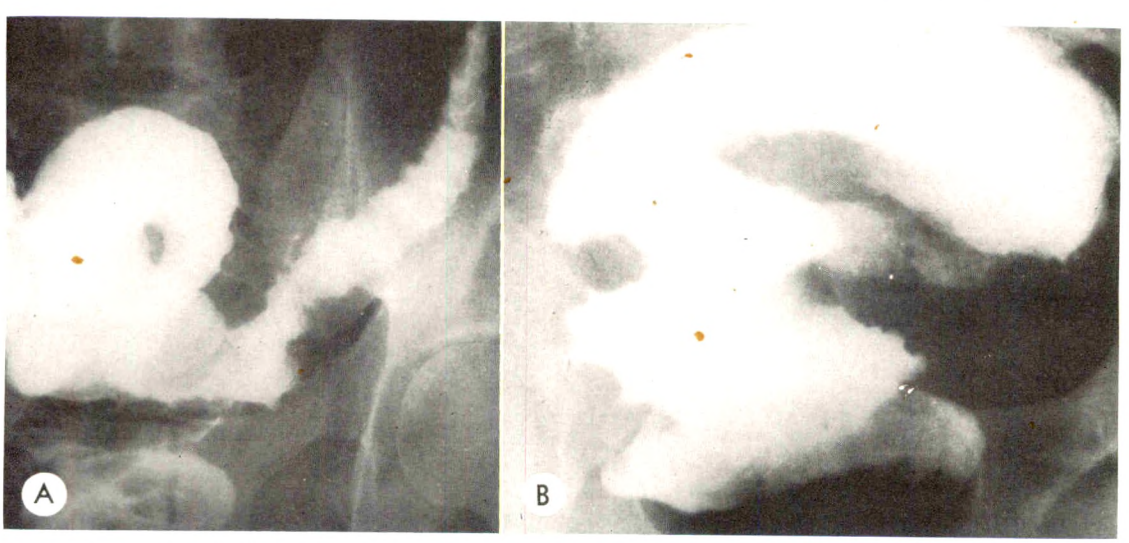


FIG. 5. Case V. (*A*) Barium enema roentgenogram. Shigellosis. Severe ulceration and edema of the rectum, sigmoid and descending colon. (*B*) Localized view of the rectum shows the deep ulcerations.

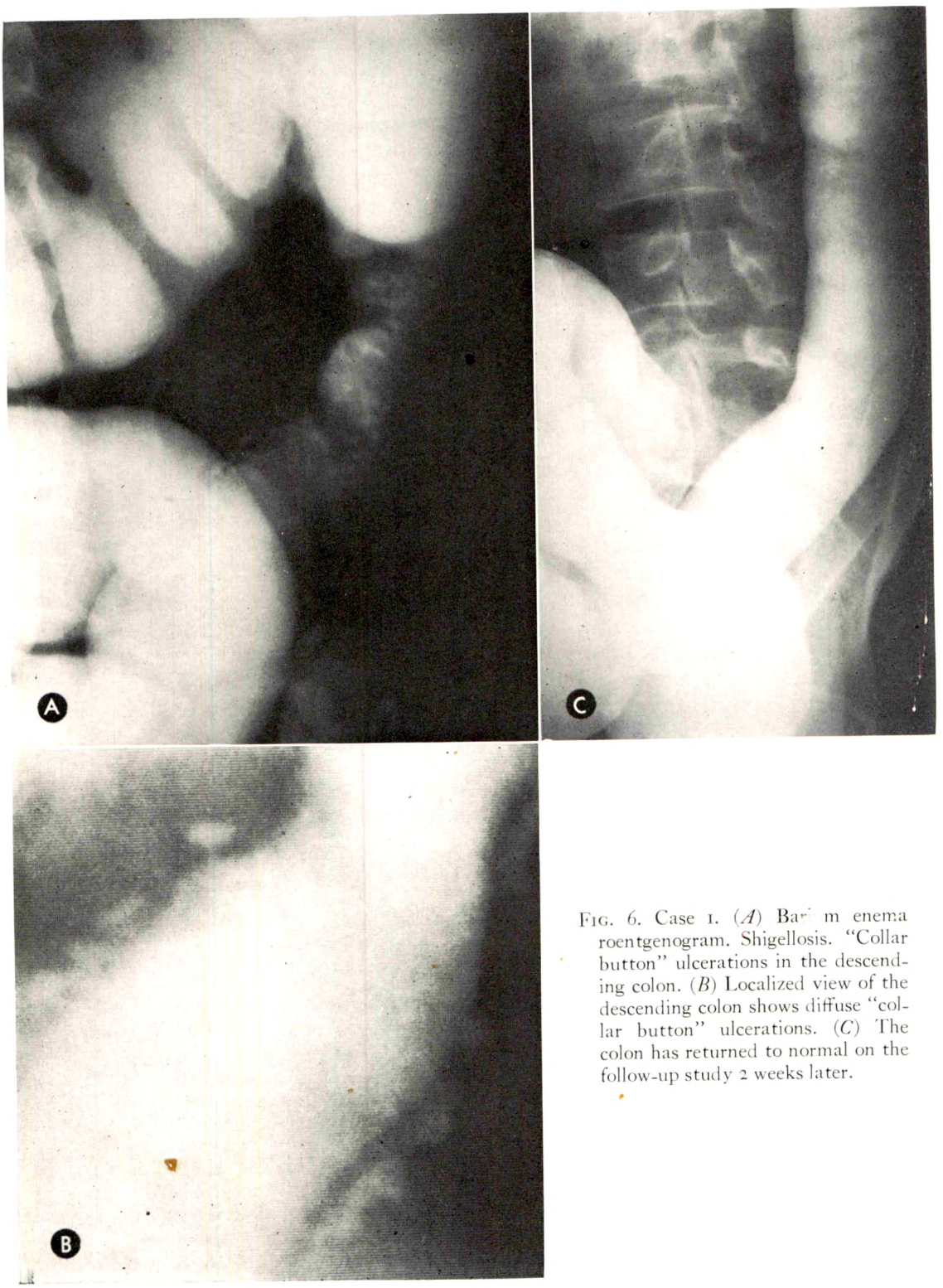


Fig. 6. Case 1. (*A*) Barium enema roentgenogram. Shigellosis. "Collar button" ulcerations in the descending colon. (*B*) Localized view of the descending colon shows diffuse "collar button" ulcerations. (*C*) The colon has returned to normal on the follow-up study 2 weeks later.

encountered. The descending colon was a frequent site of segmental colitis. Spasm, edema and ulceration were the main roentgenologic features in the involved area. The mucosa between the diffuse ulcerations was, at times, prominent and coarsened due to swelling and edema.

Deep ulcerations with a "collar button" appearance where encountered in 1 patient (Fig. 6, *A* and *B*). (Similar "collar button ulcers" have been shown to us in yet another patient with shigellosis not included in this present series.) This form of ulceration is most commonly seen in ulcerative colitis and has been encountered in granulomatous colitis and ischemic colitis.

DIFFERENTIAL DIAGNOSIS

The appearances of the colon in the 2 patients who exhibited diffuse involvement showed a striking resemblance to ulcerative and Crohn's colitis. The atonic colon with loss of the normal haustral pattern, in one instance, suggested an ulcerative colitis in the subacute or chronic rather than the active phase. Diffuse edema with superficial ulcerations has also been seen in uremic colitis,[1,9] and edema of the colon, both localized and diffuse, has been found in Henoch-Schönlein purpura.[8]

When the disease is segmental in location, differentiation from Crohn's disease may be difficult on roentgenologic grounds alone. The early features of granulomatous colitis are characterized by nodular swelling of the mucosa and asymmetric ulcerations. At this stage, Crohn's disease is not associated with an acute clinical picture and bloody diarrhea. The frequent occurrence of right-sided Crohn's disease is not a helpful differentiating point, as segmental colitis due to granulomatous disease occurs frequently enough on the left side as well.

In this series, all barium studies performed in patients who recovered following successful medical therapy demonstrated a colon which returned to complete normality (Fig. 6*C*). It is conceivable that a stricture may ensue when severe involvement of the bowel wall with deep ulcerations and necrosis occurs.[4,5]

Another infectious process which may cause localized or diffuse ulceration is amebic colitis[3] and should also be considered in the differential diagnosis of the roentgenologic lesions. Isolation of the organism responsible for the infection may be the only means available to establish the correct diagnosis.

SUMMARY

The appearances of the colon on barium enema examination in 7 patients with infectious colitis due to *Salmonella* and *Shigella* are described.

The roentgenologic alterations of the upper gastrointestinal tract and small bowel were studied in 2 of the 7 patients.

Diffuse involvement of the colon was seen in 2 patients and segmental disease with variable rectal and colonic involvement was present in the remaining 5.

In general, the features were those of ulcerations, spasm and edema.

The changes were found to be reversible in all patients who responded to therapy. These latter features of an acute colitis which may revert to normal should differentiate infectious colitis from both Crohn's colitis and ulcerative colitis.

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REMOTE CONTROL TECHNIQUE IN DOUBLE CONTRAST STUDY OF THE COLON*

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SINCE February 1966, at Notre Dame Hospital in Montreal, we have been using routinely Welin's double contrast technique in the examination of the colon. Since June 1969, all these barium enema examinations are done by remote control. So far, we have performed 20,000 examinations and present here a detailed description of that technique.

Theoretically, it is possible to diagnose minute lesions of the colon, when free from its solid and liquid contents and lined with a fine layer of uniformly distributed opaque medium over a mucosa adequately spread in a colon distended with gas. Colic preparation then implies 2 essential phases: empty the gut; and dry the mucosa. This is done in 2 stages: remote preparation; and immediate preparation.

REMOTE PREPARATION

It might seem impossible to completely evacuate the colon, but by a strict observance of the following rules, it can be achieved in a high proportion of cases.

1. *Two days prior to examination.* The patient is given a low residue diet, excluding milk, vegetable and animal fibers. The following are allowed: boiled or fried eggs, butter, rice, noodles, ground or "baby type" meats, filtered fresh fruit or vegetable juices, consommes, jellies, Jell-O, soft drinks, tea and coffee without milk. The patient must be convinced, along with the referring physician, of the importance of that diet, for the result of the examination depends upon it.

2. *The day prior to examination.* It is always imperative to administer an effective laxative and, so far, we have been favoring castor oil, at a dosage of up to 2 ounces given plain, with juices or soft drinks. The emulsified form* is also as effective after dilution with an equal amount of water. In patients suffering from chronic nonspecific colitis, a smaller dosage (1 to 1½ ounce) is given.

3. *The night prior to examination.* The patient is given a first cleansing enema. While administering 1,500 to 2,000 ml. of tap water, the patient rotates on himself.

IMMEDIATE PREPARATION

The day of the examination, when admitted to the department, the patient is given the medication and a last cleansing enema. We routinely give atropine and Valium *per os*. In exceptional cases, Probanthine or Demerol are given intramuscularly.

1. *Atropine.* A 1 mg. dosage tablet reduces mucus secretion, dries up the mucosa and counteracts the vagolytic effect produced on certain patients by intestinal distention. The enema is then better tolerated. In children decreasing dosages are given according to age:

Age	*Atropine Dosage*
3– 6 years	0.25 milligram
6– 9 years	0.50 milligram
9–12 years	0.75 milligram
12 years and up	1.00 milligram

2. *Valium.* For 2 years now, we have also been giving the patient 5 mg. of Valium *per os* as a premedication; we believe that it reduces the patient's anxiety.

A 50 mg. dosage of Demerol is occasionally administered intramuscularly to very anxious people, whereas, when the colon appears too spastic, 30 mg. of intramuscular Probanthine may permit the examination in 20 minutes.

* Unisoil. Unik Medical Lab., Inc., Montreal.

* From the Department of Radiology, Notre Dame Hospital, Montreal, Quebec, Canada.

3. *Cleansing enema.* About half an hour after the premedication has been given, the last cleansing enema, about 1,500 ml., is administered by a trained person in a small room adjoining the roentgenographic room, so that we can control the preparation and reduce the accidents to a minimum. If, for example, the patient cannot hold the enema, we will resort to an inflated retaining balloon during the examination (Fig. 3).

The enema contains in 2 liters, 0.02 mg. of the contact laxative, oxyphenisatin,† and 2.5 gm. of tannic acid.

We also use the above mentioned preparation when searching for the cause of rectorrhagia in children but not in the investigation of their constipation. We think that, while receiving the enema, the patient, if possible, should first turn on his left side, then prone, then on his right side and finally on his back. The enema should be completely evacuated and the examination should not be undertaken for at least half an hour. Children are given varying amounts according to age.

† Lavema. Winthrop Laboratories, Aurora, Ontario, Canada.

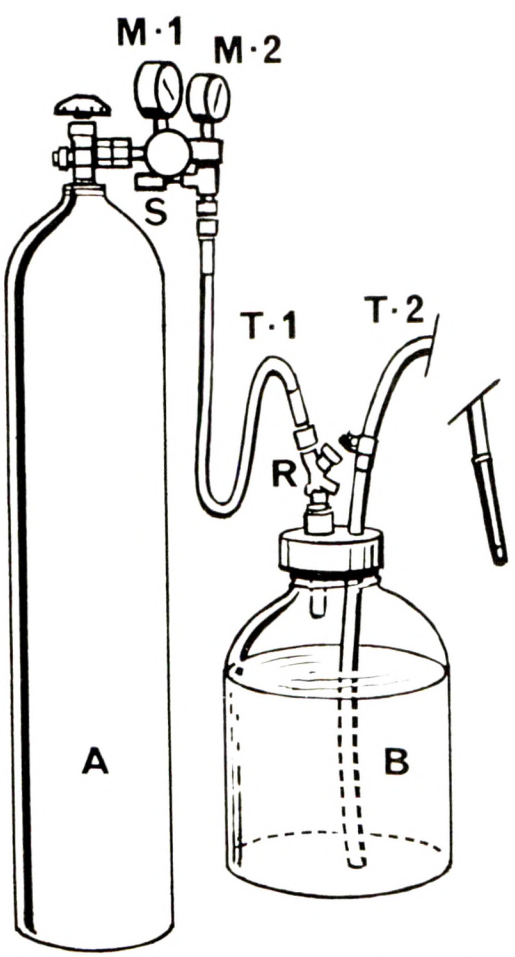


Fig. 1. Drawing of the barium injector.

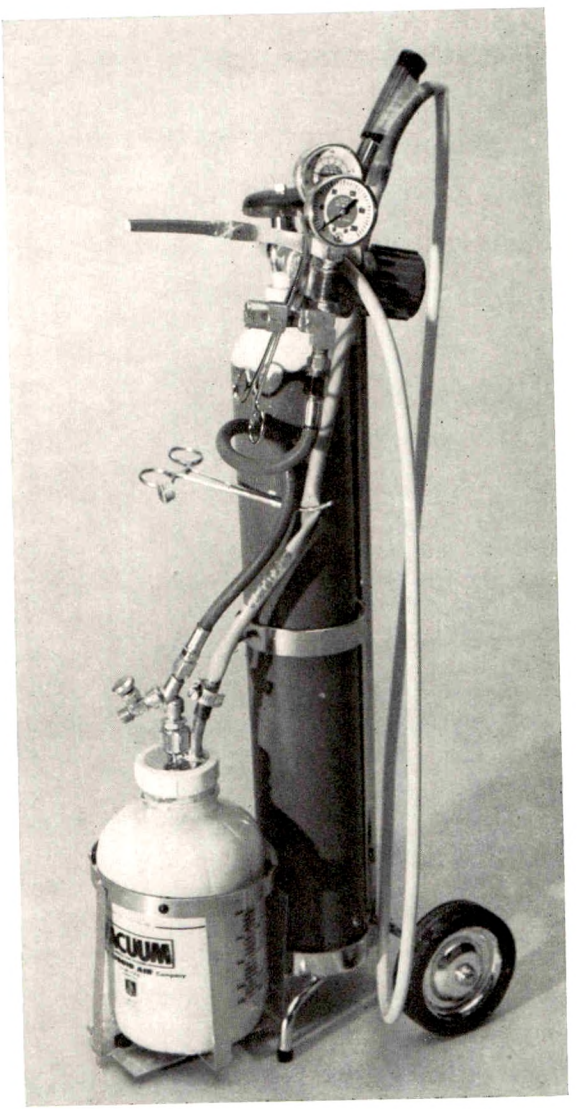


Fig. 2. Mobile apparatus of the barium injector.

BARIUM

For the last 4 years, we have been using a colloidal suspension of barium sulfate containing also sodium citrate, carboxy-

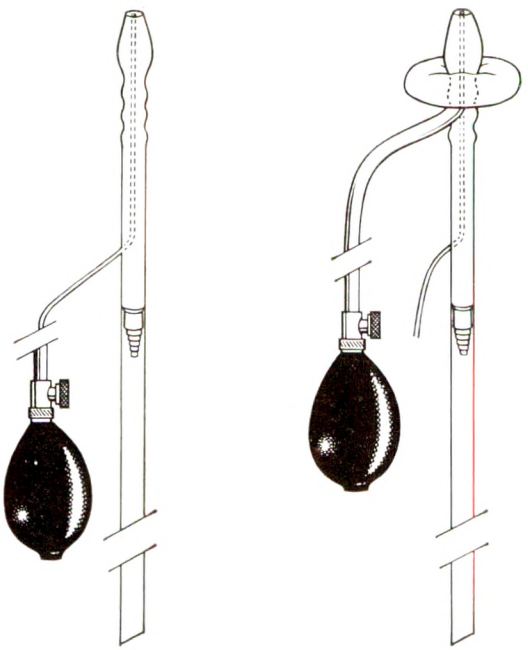


FIG. 3. Miller air tips, with and without cuff, adapted to a 50 cm. tube (internal diameter—1 cm.).

methyl-cellulose and polysiloxane, an anti-foam agent.* It has a viscosity of about 6,000 Ctps and a density of 1.80–1.85. It is a very viscous barium which adheres to the mucosa and produces very little bubbling. Before pouring it into the injector system, we add 1.25 gm. of tannic acid and a sachet of Lavema (oxyphenisatin, 0.02 mg.) for each liter of solution. This preparation is still the simplest and most effective we have experienced so far.

EQUIPMENT

A. ROENTGENOGRAPHIC

In over 3 years, we have performed more than 10,000 examinations on remote control roentgenographic tables,† which permit various positions including a Trendelenburg at 90°. We believe that a 40° to 50° Trendelenburg is necessary for an easy and satisfactory examination in adults. This is of great help in an adequate distribution of barium and air with the use of only a minimal amount of barium, about 250 cc., spreading by gravity through the loops and angles.

The focus-film distance is 100 cm., the least high kilovoltage is 140 kv., with 1,000 ma. generators. The focal spot may vary from 0.6 to 1.2 mm. We have been generally satisfied with short exposures.

Another characteristic of the tube, indispensable for rapidity of examination and efficacy of fluoroscopy, is the degree of angulation possible with regard to the level of the table: a maximum possible cephalic or caudal angulation of 30°, without which the fluoroscopic examination of

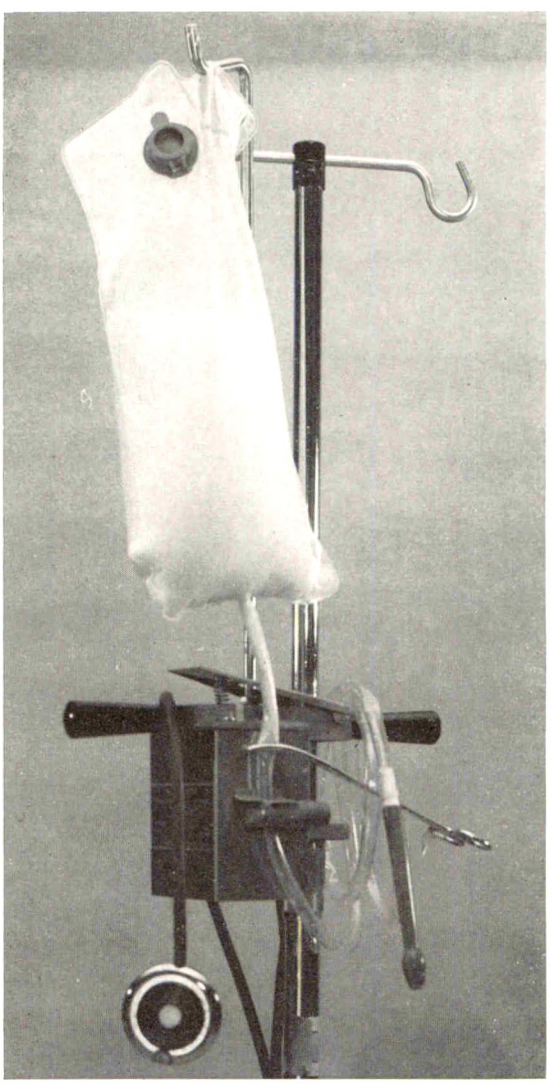


FIG. 4. E-Z-EM. Remote control interrupter.

* Gel-Unix.S., 60% W/V, Barium suspension. Unik Medical Lab., Inc., Montreal.

† Siregraph. Siemens of Canada Ltd. Montreal.

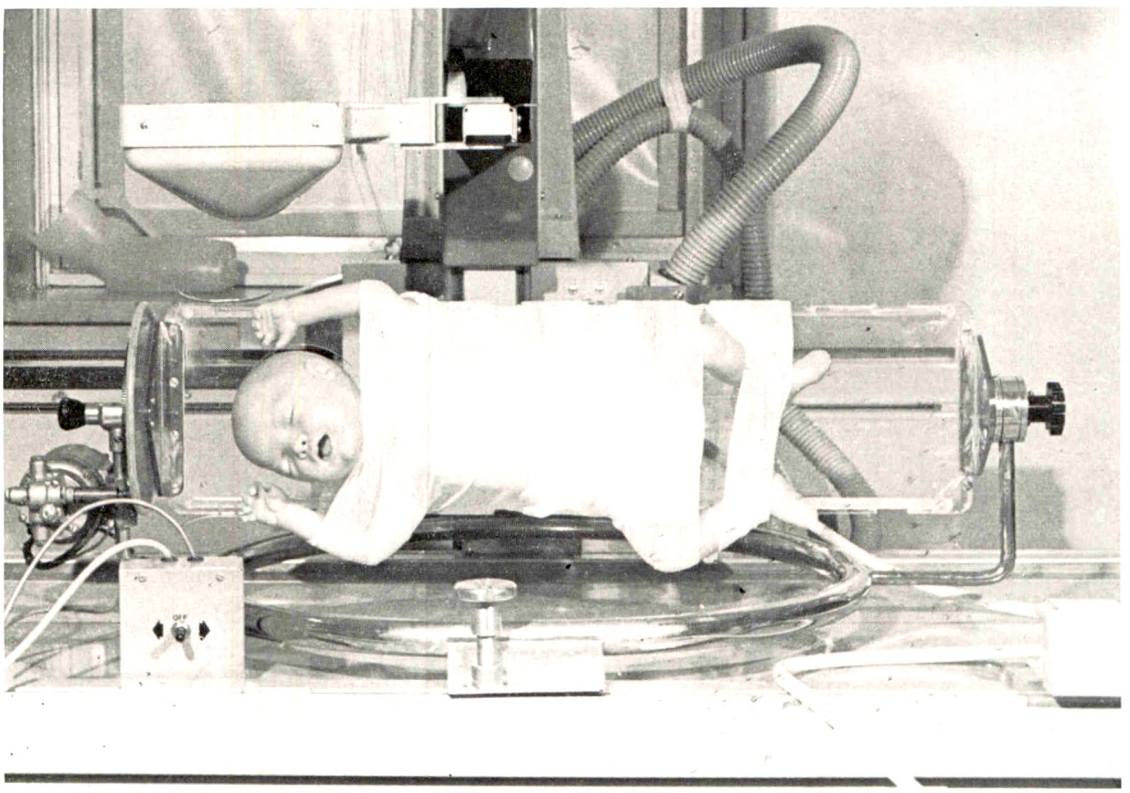


Fig. 5. Motorized and remote control Ayme's wheel.

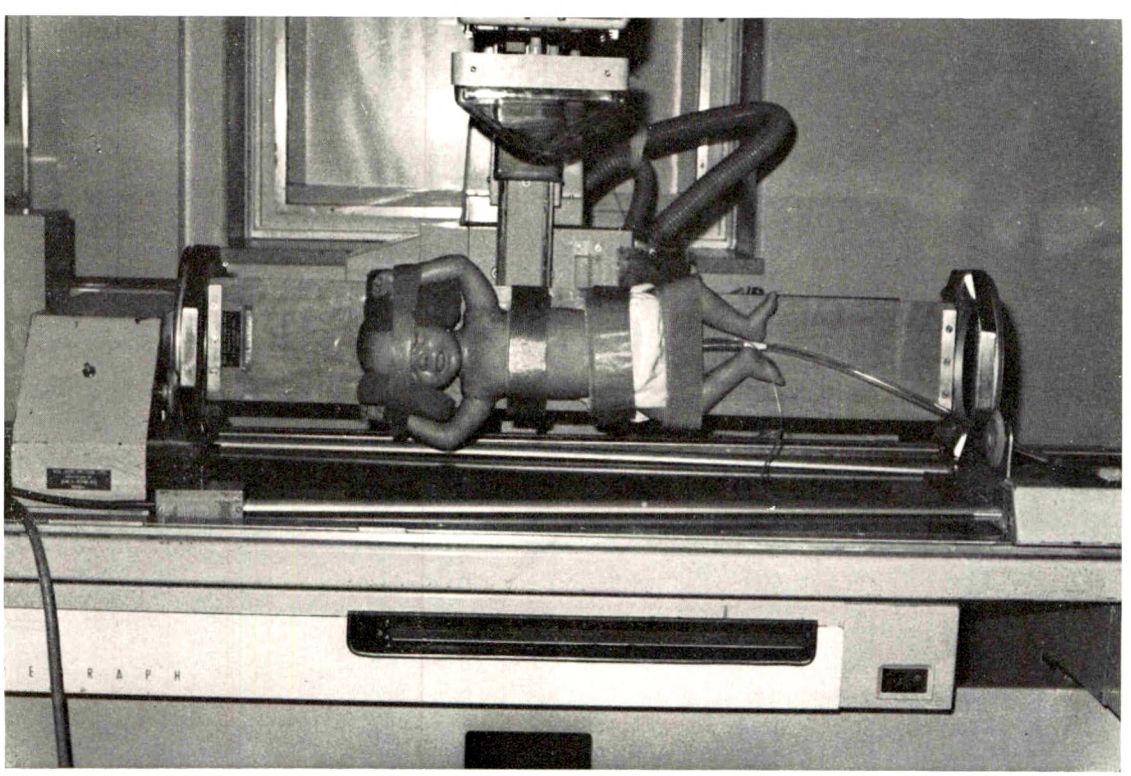


Fig. 6. Remote control Vezina's octagon.

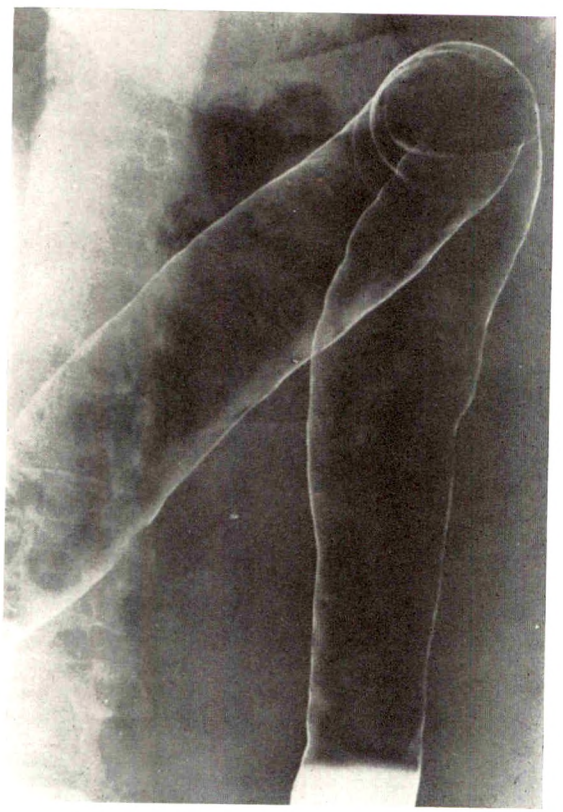


Fig. 7. Splenic flexure in erect position.

the rectosigmoid colon is difficult and unreliable. Angulation of the tube allows a clear visualization of these segments of colon which are usually out of reach.

Both examination rooms are equipped with overhead tubes, permitting exposure of the 2 most important roentgenograms with this technique—in right and left lateral decubitus positions with horizontal beam—which will be discussed later.

B. Barium and Air Injection Equipment

First, we used the conventional method utilizing a bag hanging above the table. The injection was so slow, we had to conceive a more convenient system which is illustrated in Figures 1 and 2.

A compressed air cylinder (A) is connected to a 4 liter container (B) filled with barium, by way of a tube (T_1), whose opening is placed above the level of the barium. A second tube (T_2), whose tip stands at the bottom of the container (B), carries the barium to the patient. Two manometers are part of the system: the first (M_1) indicates the amount of compressed air left in the cylinder; and the second (M_2) the pressure in pounds per square inch which will be exerted upon the surface of the barium. The pressure is about 10 pounds per square inch. There is also a safety valve (S) which opens if the pressure exceeds 12 pounds. Additional barium should be added to the container after 2 or 3 examinations, so as to maintain the level above the tip of the tube (T_2), in order to avoid accidents.

The apparatus also includes a set of tubes and solid forceps to pinch off the tubes. We use the Miller air tip with a 1 cm. internal diameter (Fig. 3). It is provided with a small polythene tube penetrating into the lumen of the cannula and through which air is insufflated. A 50 cm. long tube with a 1 cm. lumen is connected to the cannula. It is most important to use this diameter to obtain a complete evacuation of the rectum when needed.

The adaptation of the Miller air tip with cuff (Fig. 3) assures complete closure of the rectum. Finally, the insufflation tube is manipulated from the control desk through another tube connected to the insufflation pump (Fig. 3). A stop valve can be fixed to the tube preventing a backflow and an 18 gauge needle can be used as an adapter

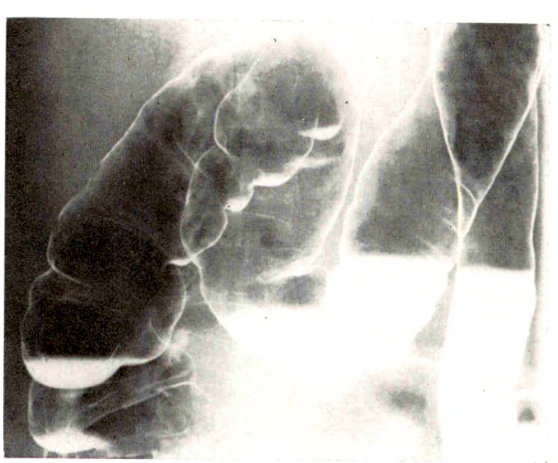


Fig. 8. Hepatic flexure in erect position.

fixed to the small insufflation tube of the Miller cannula (Fig. 3).

In about 2 per cent of our patients, the conventional barium enema study is the sole feasible technique; the same equipment should be used, especially as regards angulation of the roentgen ray tube. In these cases we use the electrical switch* fixed on the tube running from the barium bag to the patient. This is a spring forceps connected to an electromagnet. It is supplied with a luminous guide indicating the barium flow (Fig. 4).

This system allows control of colic filling from the control desk, while keeping the personnel safe from radiations.

C. CONTENTION EQUIPMENT

Our tables are not equipped with cradles, which are not necessary for routine examination in adults, but they might be of great help, especially in bed-ridden patients.

When examining infants or young children a contention equipment with remote control such as motorized Ayme's wheel (Fig. 5) or Vezina's octagon (Fig. 6) seems essential. We have somewhat modified the Ayme's wheel, so that it could be fixed to the table allowing a better movement utilization. Moreover, Vezina's octagon* has another advantage: the young patients may be prepared and prefixed to the octagon outside the examination room, thus saving valuable time.

* E-Z-EM. Stop valve: E-Z-EM, Port Washington, New York.

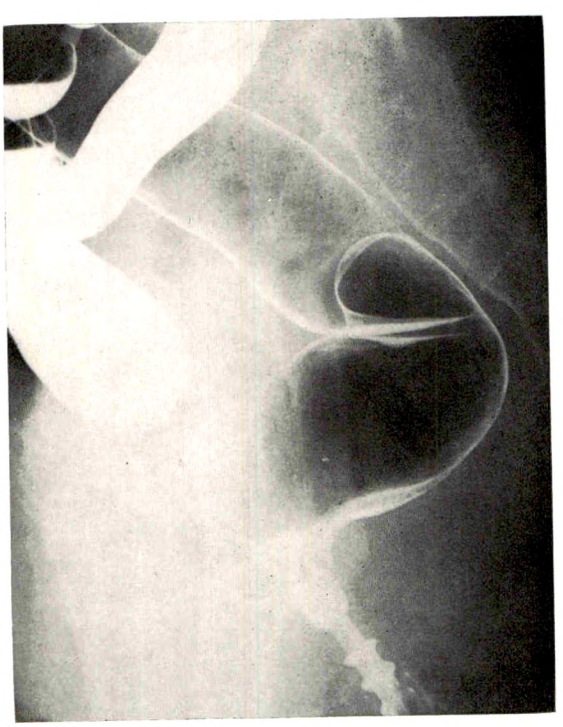


Fig. 9. Lateral view of the rectum in erect position.

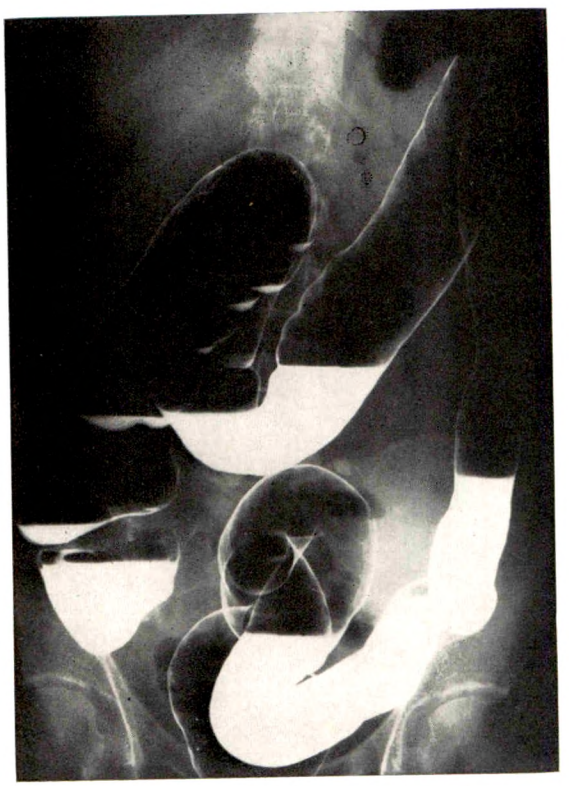


Fig. 10. Anteroposterior view of the abdomen in erect position.

THE EXAMINATION

The patient who adhered to the prescribed diet, received the medication and the 2 cleansing enemas is finally ready for examination about half an hour after the last enema. He is first instructed to lie on his left side. A well trained technical person can proceed with the introduction of the cannula, but sometimes the radiologist himself would have to do it. The cannula

* Octagon Board. Reno Micro Precision Ltd., 8422 10th Ave., St. Michel, Montreal 455, Quebec.

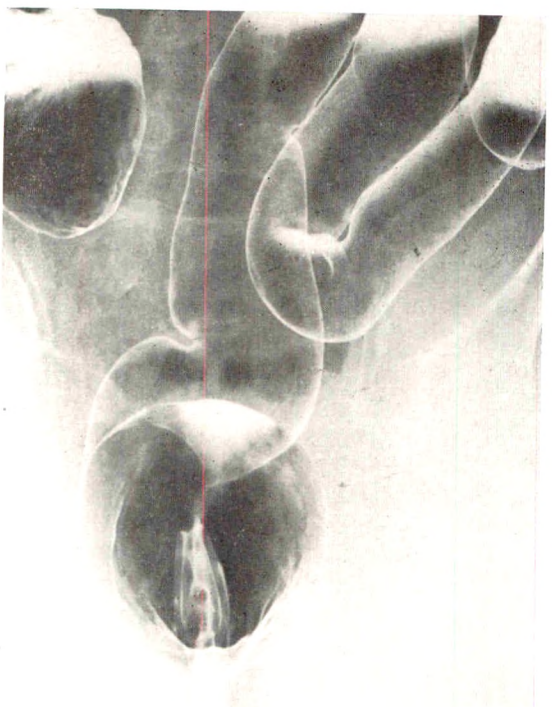


FIG. 11. Sigmoid view in angle prone projection (caudad 30°).

must be firmly fixed by diachylons, so that it will stay in place during the entire examination. If a balloon is used, its position and inflation are checked under fluoroscopy; generally speaking, its diameter on the screen should not exceed 7 cm. and it should be kept in the lowest part of the rectum in contact with the internal anal sphincter.

The cannula and its 50 cm. long tube are adapted to the injector and the smaller tube is also connected to the insufflation pump. The patient is then turned prone and the radiologist takes over. We routinely evaluate the symptomatology by a brief questionnaire and we encourage the patient to hold his barium enema and to breathe deeply if filling causes colicky pains.

After a quick fluoroscopic survey the radiologist tilts the table to the Trendelenburg position and the injector is opened.

Under a pressure of 10 pounds/square inch, the barium slowly flows into the rectum and sigmoid colon. By utilizing first the cephalad and then the caudad angulation of the roentgen ray tube, almost any loop of the rectosigmoid colon can be unfolded.

When the barium reaches the level of the left iliac crest, the tubes are shut off and the injector is disconnected. After a while, experience will teach how much barium should be used, taking into account some factors such as the diameter and the length of the rectosigmoid colon and the presence of chronic constipation.

The insufflation tube is then opened and the colon is dilated with air. The lumen of the tube being small, the insufflation is more gradual and smoother and the barium pushing ahead goes through the splenic flexure, while the patient is in a prone position and the table more or less in a Trendelenburg tilt.

When a sufficient amount of barium has penetrated the transverse colon, the table is tilted back to the horizontal position. The patient is then turned to his right side, thus allowing passage of barium through



FIG. 12. Lateral view of the rectum in left decubitus position.

the hepatic flexure. The patient turns supine and insufflation is continued: the barium flows into the ascending colon and often down to the cecum. The latter is definitely filled when the table is tilted upright. Then the insufflation is completed, and spot filming is done under fluoroscopic control. If the colon is longer than usual or if feces and liquid were retained in the colon, multiple rotations and different angulations of the table are used to get an adequate distribution of barium and air with satisfactory coating of the mucosa, the feces being dispersed in the barium.

We have selected a uniform size of 25 ×30 cm. for these spot film roentgenograms which are obtained in the following order (No. 1, 2, 3, and 4 are exposed upright):

1. *Splenic flexure.* Slight rotation of the patient to the right (Fig. 7).

2. *Hepatic flexure.* The patient is slightly rotated to his left side (Fig. 8).

3. *Lateral view of the rectum.* The left side of the patient is close to the table, the rectal tube is opened into a container placed behind the patient's feet and complete evacuation of the rectum is checked fluoroscopically. Small insufflations and modifications of the position of the cannula usually favor a more satisfactory evacuation and it is better if we also ask the patient to contract his abdominal wall. When the latter is completed, the drainage tube is closed and the rectum is again inflated before the film can be exposed (Fig. 9).

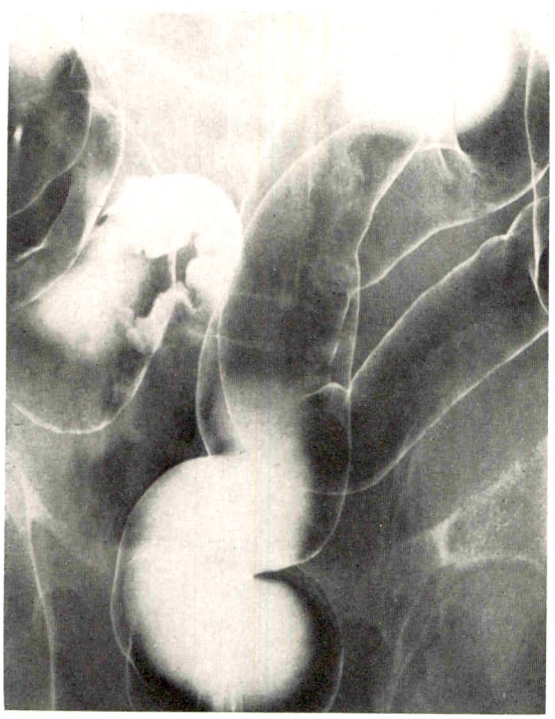


Fig. 13. Second view of the sigmoid in angle supine projection (cephalic 30°).

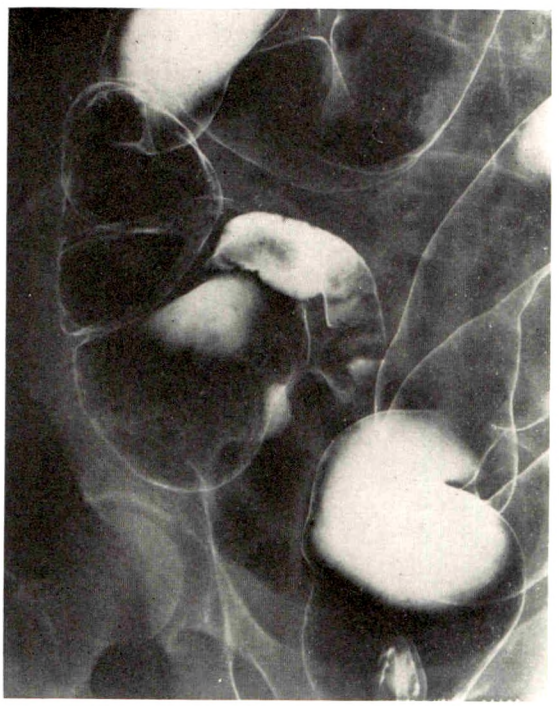


Fig. 14. Cecum.

4. *Anteroposterior view of the abdomen on a 35×43 cm. film.* The patient is turned with his back on the table and the film is exposed after checking the distention of the colon centering the beam over the interspace between L_4-L_5 (Fig. 10).

5. *Sigmoid colon, the patient lying prone on the table in Trendelenburg and 30° caudad angulation of the tube.* The patient is again turned on his left side, while the table is tilted down to horizontal position. The patient then turns prone and the table is put to a 55 to 40° Trendelenburg, thus permitting the barium to flow from the sigmoid into the descending colon. When

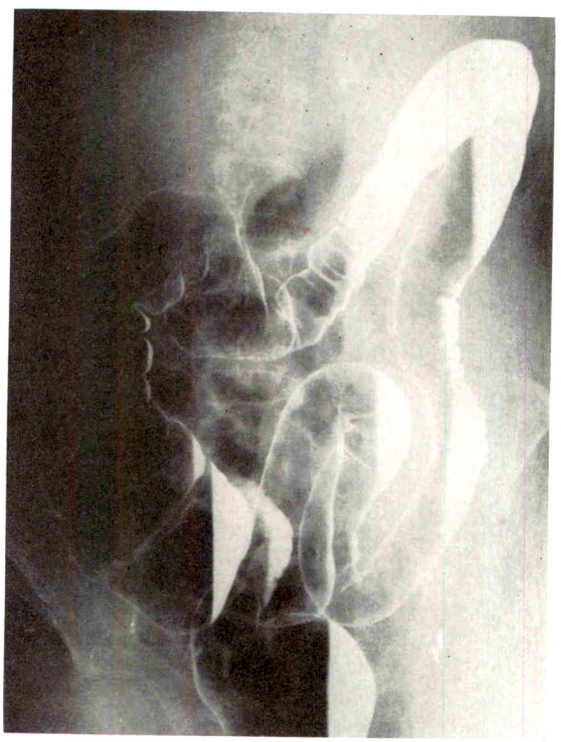


Fig. 15. Abdomen in left lateral decubitus position with horizontal beam.

insufflation of the sigmoid appears satisfactory the film is exposed, with the beam angulated 30° caudally (Fig. 11).

6. *Left lateral decubitus of the rectum.* The table is tilted back to the horizontal position with the patient turned again on his left side. A second lateral roentgenogram of the rectosigmoid colon is taken, the optimal angulation of the tube being selected so as to avoid superimpositions: the sigmoid colon must be empty of barium and well distended (Fig. 12).

7. *Sigmoid colon, the patient being in the supine position and the tube angulated 30° cephalad.* The patient lying supine, the sigmoid colon is located and the roentgenogram is taken using a 30° cephalad angulation of the beam (Fig. 13).

8. *Cecum.* The patient is usually slightly rotated to the right. The table is tilted to the Trendelenburg position so that the cecal barium can flow into the ascending colon. The insufflation is completed and the film is exposed under a slight compression (Fig. 14).

Many of the preceding roentgenograms are obtained under a proper compression. From then on the participation of the radiologist is no longer needed, the next roentgenograms being taken by the technicians once the cannula has been removed, provided the inflated balloon is not needed for retention of the enema.

The next 6 roentgenograms are made on 35×43 cm. films, the centering point being the upper border of the iliac crests.

9. *Right anterior oblique (about 25°) with the patient in the supine position.*

10. *Left anterior oblique (about 25°) with the patient in the supine position.*

11. *Right posterior oblique (about 25°) with the patient in the prone position.*

12. *Left posterior oblique (about 25°) with the patient in the prone position.*

13. *Left lateral decubitus with horizontal beam* (Fig. 15).

14. *Right lateral decubitus with horizontal beam* (Fig. 16).

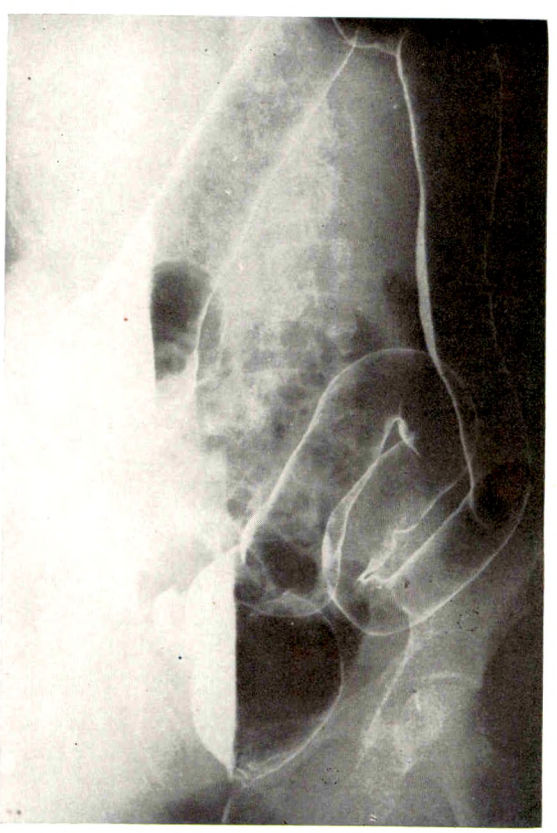


Fig. 16. Abdomen in right lateral decubitus position with horizontal beam.

The latter 2 roentgenograms should be taken with special care. The patient is first asked to turn on his left side and then to sit. A 100×50 cm.×10 cm. foam pad is placed under the patient from the hip to the axilla, the patient lying down again on his left side. A 35×43 cm. grid cassette fixed to a holder is placed vertically on the patient's abdomen centering over the upper border of the iliac crest or a little lower if the patient is taller so that the entire rectum will show on the film. The roentgen ray tube is placed at about 150 cm. from the film holder. We always use a small focal spot (0.6 mm.), a kilovoltage of 120 to 140 and the shortest exposure time possible. A second grid cassette is necessary for the last roentgenogram, after the patient has rotated 180° on his pad with the film close to the patient's back.

We do not as a rule take a postevacuation roentgenogram because, according to our experience, it is not contributory. However, in cases of suspected chronic colitis, an early evacuation roentgenogram is required. We also use postevacuation fluoroscopy, with spot roentgenograms if necessary, when the clinical history suggests the presence of acute abdomen, fistula or appendicitis.

CONCLUSION

The complete examination usually requires 20 minutes and a maximum fluoroscopic time of 3 minutes. Considering the results, 4.5 per cent of cancers and 13 per cent of polyps found with less than 0.5 per cent of error, we believe that this method should be used as a routine procedure or at least as a complement to examination after a conventional barium enema study has failed to corroborate the clinical impression.

The most important factors for the success of this technique are: an adequate preparation insuring a clean and dry mucosa; a suitable barium adhering uniformly to the mucosa; and finally a well trained staff in a well organized department.

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Vol. 119, No. 2

THE VALUE OF THE POSTEVACUATION VIEW OF THE COLON IN THE DETECTION OF PELVIC PATHOLOGY*

A REPORT OF 9 ILLUSTRATIVE CASES

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THE value of routine roentgenologic procedures such as plain abdominal roentgenography, intravenous urography, and the barium enema examination of the gastrointestinal tract has been well established in the diagnosis of pelvic pathology.[1] The complementary use of the postero-anterior, anteroposterior, lateral, oblique, angled, and Chassard-Lapine views of the barium filled colon, and specifically of the sigmoid, has been stressed.[4,5] The examination of the colon by means of a barium enema assumes an added importance when the pelvic tumor is not sufficiently large to be palpable on abdominal examination, or the patient is obese, and particularly when a pelvic examination is not performed due to the absence of a history indicating pelvic pathology.

Extracolonic pathologic processes arising in the pelvis may produce pressure effects on the outline of the colon because of their close proximity or secondary involvement.[7] Pelvic masses, when sufficiently large, displace, straighten or flatten the rectum and sigmoid.[1] When they become larger and extend into the abdomen, they may also displace the intra-abdominal segments of the colon[1] or flatten the superior contour of the distal sigmoid in the frontal projection.[2] In the lateral view of the pelvis, the sigmoid may be compressed between the mass and the sacrum and may also fail to move anteriorly following evacuation of the colon.[6]

We have noticed that the routine postero-anterior view of the colon following evacuation may be the only one to reveal the evidence of an extracolonic lesion and, particularly, of a pelvic lesion. The colon exhibits various degrees of mobility. During filling, the distention and uncoiling produced by the hydrostatic pressure move the proximal sigmoid away from the limited confines of the pelvis, and therefore away from the pelvic mass into the abdomen, thus reducing or eliminating contact. At this stage of the examination the pressure effect on the colon is therefore not present. Following evacuation, the empty sigmoid resumes its position within the pelvis and drapes the mass, exhibiting the characteristic concave extrinsic pressure effect and stretching of the intact folds of the mucous membrane in a longitudinal direction. In the relatively fixed distal sigmoid and rectum, the effect, during the filling stage of the barium enema examination, is similar to that of a tire tube inflated with air. The area of contact between the mass and the colon decreases as the latter is distended. The opposite effect occurs, obviously, following evacuation and deflation. The same effect is noticed in other, normally mobile portions of the colon, when fixed by adhesions.

REPORT OF CASES

CASE 1. A 58 year old obese female presented with a history of backache and constipation. No mass was palpable on abdominal examination.

Displacement of the distal sigmoid to the right and elevation of its proximal segment was demonstrated only in the postevacuation roentgenogram (Fig. 1, *A–C*).

Pelvic examination revealed a left adnexal mass.

Operation confirmed the presence of a 13

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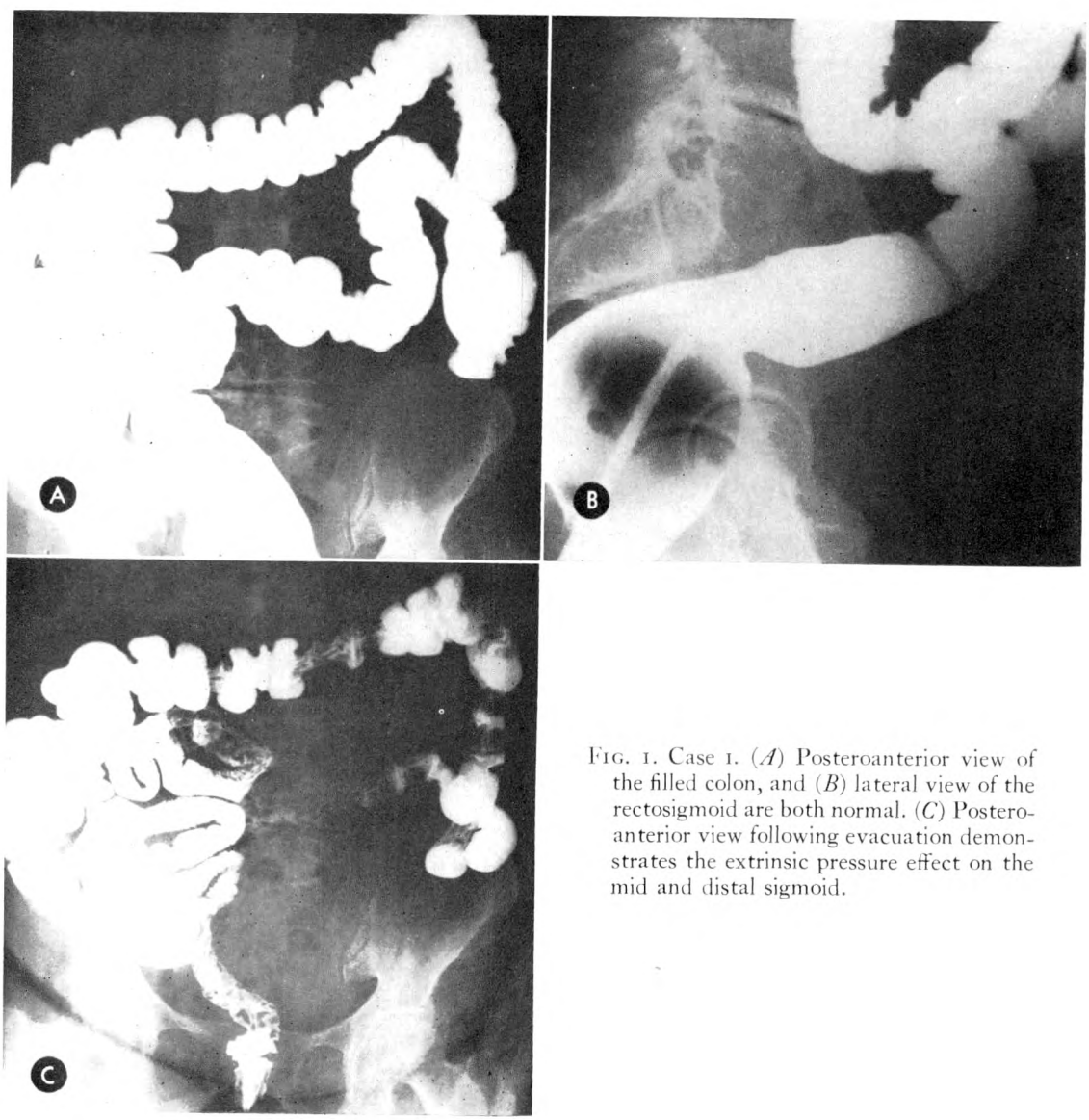


FIG. 1. Case 1. (*A*) Posteroanterior view of the filled colon, and (*B*) lateral view of the rectosigmoid are both normal. (*C*) Posteroanterior view following evacuation demonstrates the extrinsic pressure effect on the mid and distal sigmoid.

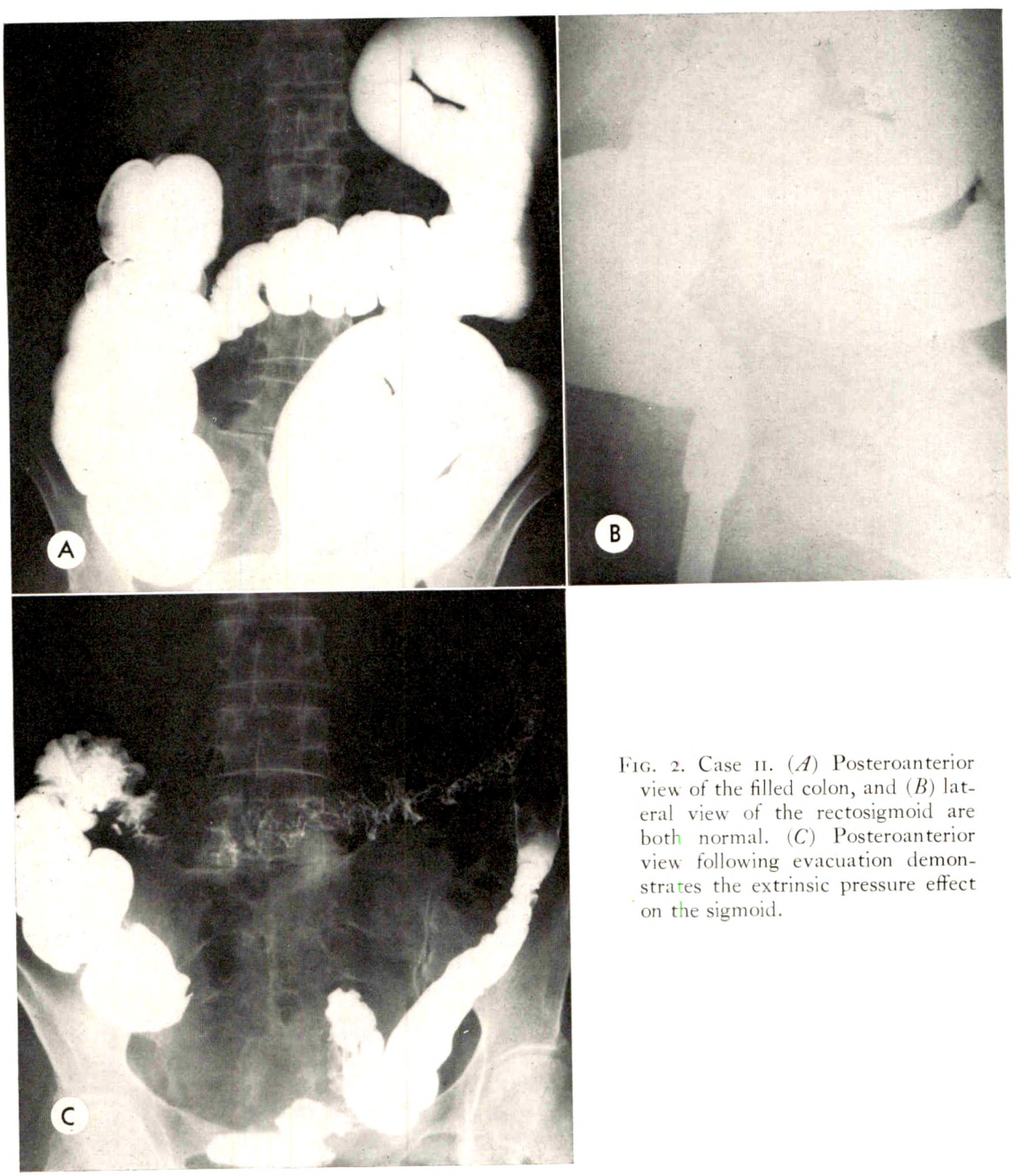


Fig. 2. Case 11. (*A*) Posteroanterior view of the filled colon, and (*B*) lateral view of the rectosigmoid are both normal. (*C*) Posteroanterior view following evacuation demonstrates the extrinsic pressure effect on the sigmoid.

×13 cm. multilobular cystic mass involving the left broad ligament.

Histologic examination revealed an intraligamentary leiomyoma with myxoid and hyaline degeneration.

Case II. A 35 year old female presented with a history of backache and intermenstrual spotting. A fibroid tumor had been removed 10 years previously. Pelvic examination revealed a 6×7 cm. mass attached to the uterus.

Displacement of the sigmoid to the left and inferiorly was demonstrated only on the postevacuation roentgenogram (Fig. 2, *A–C*).

Operation confirmed the presence of a fibroid tumor of the uterus.

Case III. A 55 year old female presented with a past history of hysterectomy and bilateral salpingo-oophorectomy. Recently she had felt a left lower quadrant mass. Physical examination revealed an ill-defined fullness in the left lower abdomen.

An extrinsic pressure effect over the inferior aspect of the proximal sigmoid colon with intact but stretched mucosal pattern was demonstrated only on the postevacuation roentgenogram (Fig. 3, *A–C*).

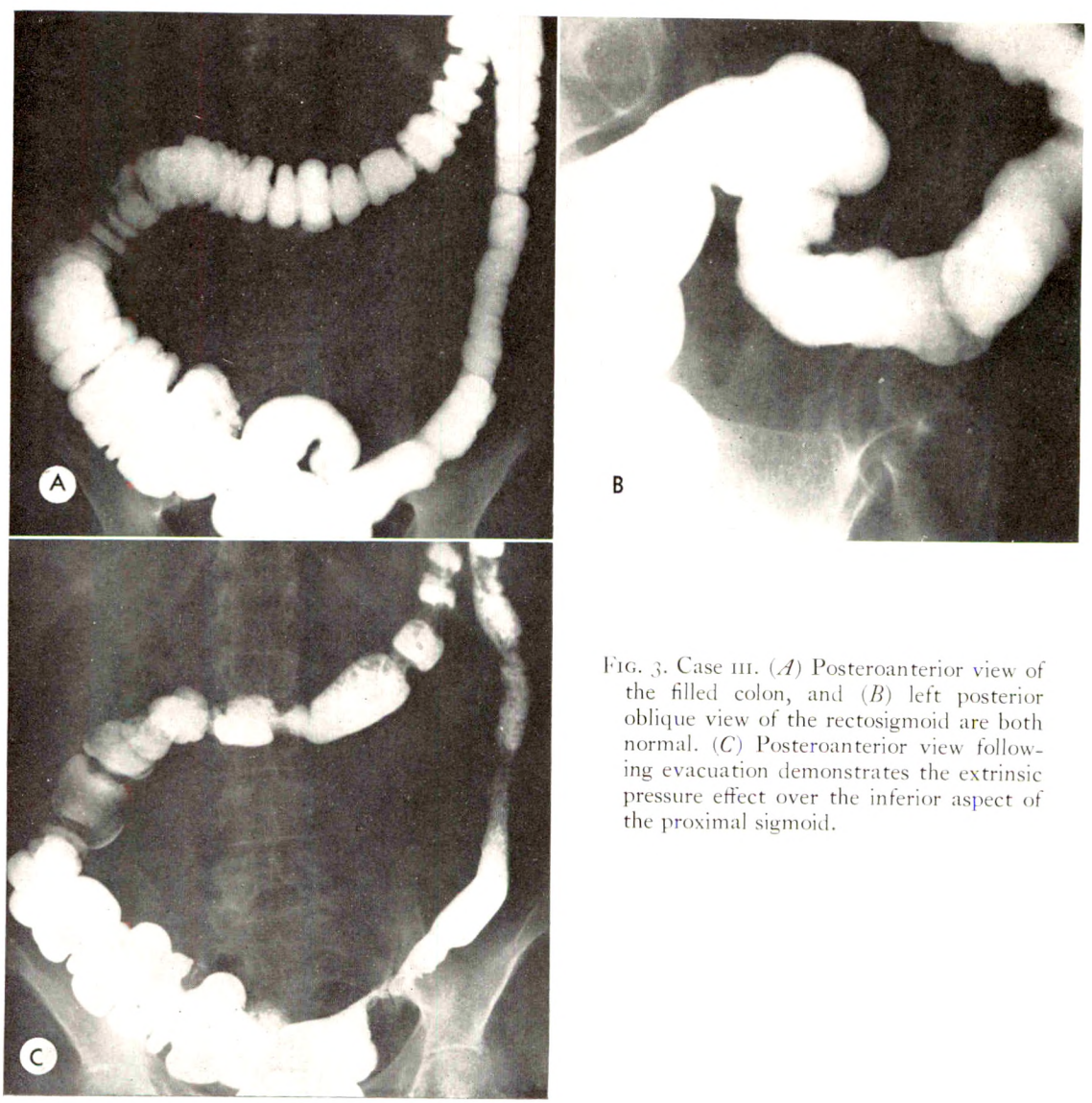


Fig. 3. Case III. (*A*) Posteroanterior view of the filled colon, and (*B*) left posterior oblique view of the rectosigmoid are both normal. (*C*) Posteroanterior view following evacuation demonstrates the extrinsic pressure effect over the inferior aspect of the proximal sigmoid.

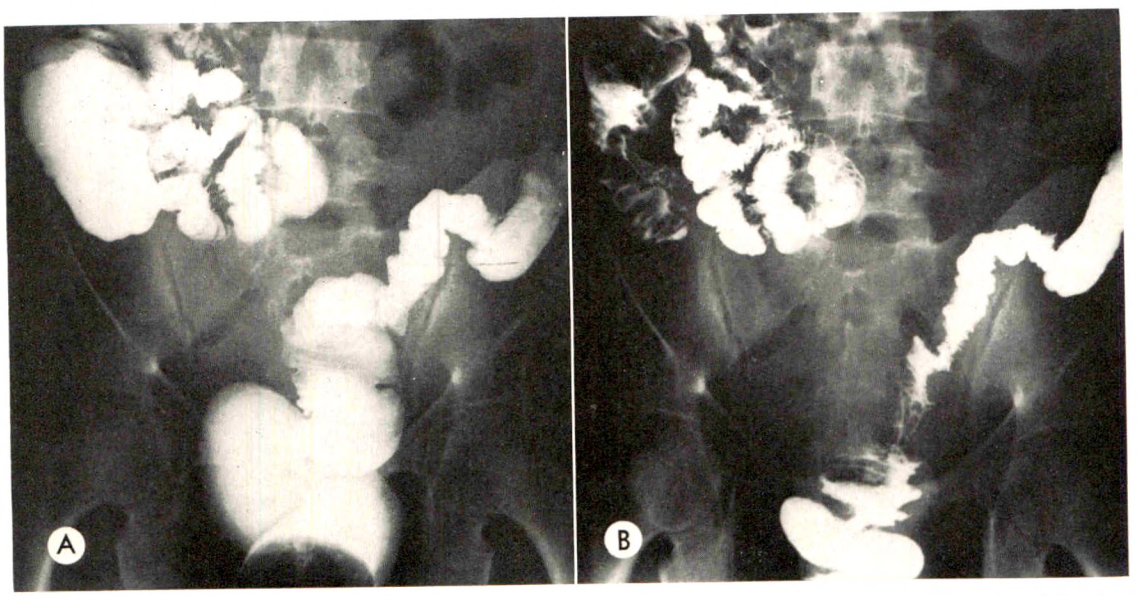


Fig. 4. Case iv. (*A*) Posteroanterior view of the filled sigmoid is normal. (*B*) Posteroanterior view following evacuation demonstrates the extrinsic pressure effect over the sigmoid.

At operation, a mass, formed by adherent loops of small intestine and omentum, was found. There were no intraluminal or intramural abnormalities of the proximal sigmoid colon.

Case iv. A 50 year old male presented with a history of gradual abdominal distention. Physical examination revealed marked ascites, but no palpable mass. There was no peripheral lymph node enlargement.

Depression, displacement to the left, and stretching of the mucosal folds of the proximal sigmoid colon were demonstrated only on the postevacuation roentgenogram (Fig. 4, *A* and *B*).

At operation, in addition to the ascites, matted masses were found throughout the abdomen.

A biopsy revealed lymphoma.

Case v. A 55 year old male presented with a history of weight loss and lower abdominal discomfort. On rectal examination a hard mass was felt on the right side, extending anteriorly. Proctosigmoidoscopy revealed an intact mucosa.

Extrinsic pressure effect on the right lateral wall of the rectum was best demonstrated on the postevacuation roentgenogram (Fig. 5, *A–C*).

Biopsy revealed metastatic carcinoma, primary site undetermined.

Case vi. A 40 year old female presented with a history of low abdominal pain. Physical examination revealed no palpable abdominal mass.

The lateral view demonstrated only minimal posterior displacement of the anterior wall of the sigmoid at the pelvic inlet (Fig. 6*B*). The postevacuation posteroanterior roentgenogram of the colon demonstrated flattening of the sigmoid and left lateral displacement of an ileal loop (Fig. 6, *A–C*).

Pelvic examination revealed a mass attached to the left side of the uterus.

At operation a 10×9×7 cm. fibroid of the left wall of the uterus was found.

Case vii. A 46 year old obese female presented with a history of intermenstrual bleeding. Physical examination revealed no palpable abdominal mass. Pelvic examination revealed a large irregular midpelvic mass.

Displacement of the sigmoid to the left with longitudinal stretching of the mucosa was seen only on the postevacuation roentgenogram of the colon (Fig. 7, *A–C*).

At operation a 15×15 cm. fibroid was found on the posterior wall of the uterus.

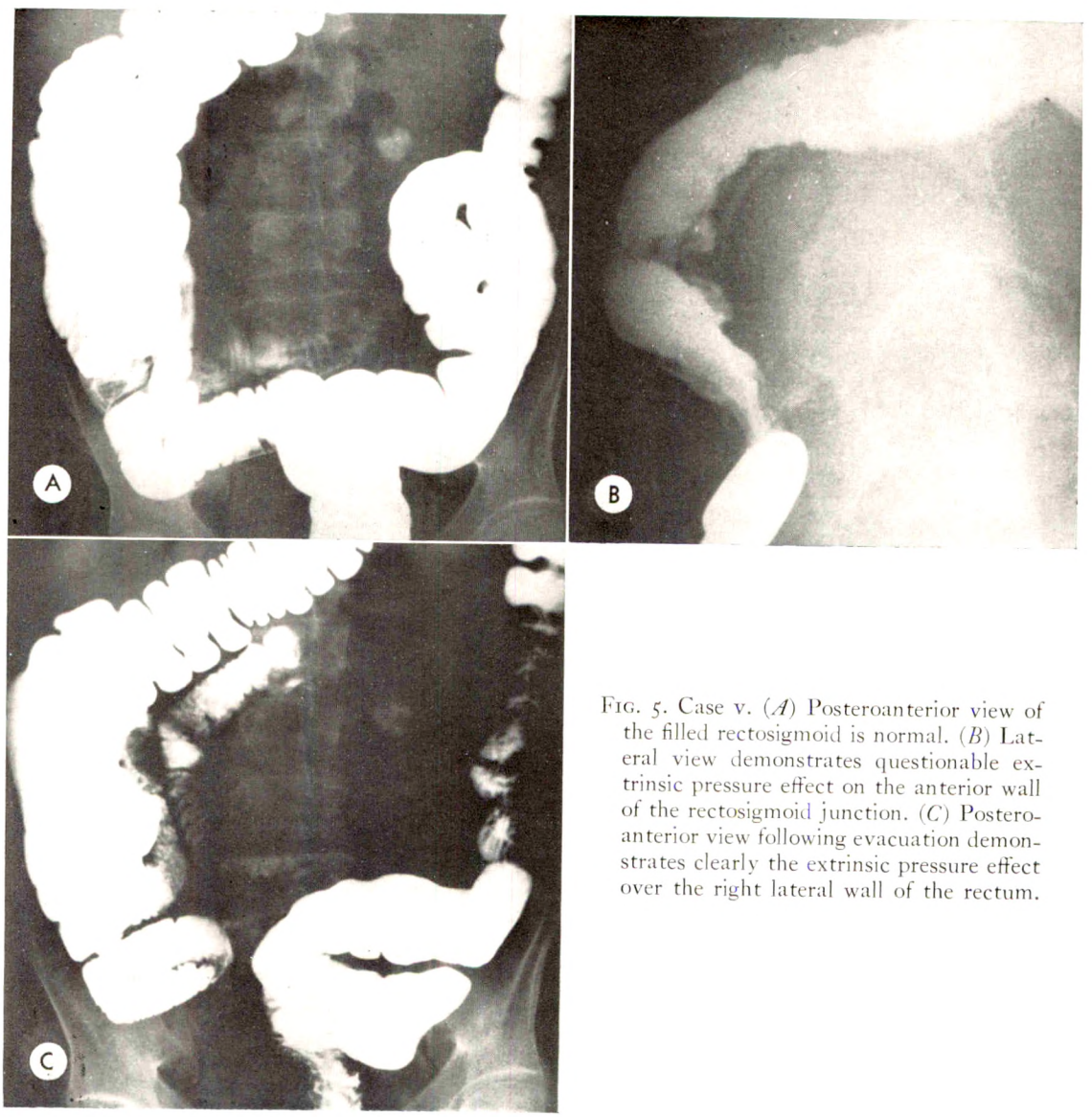


FIG. 5. Case v. (*A*) Posteroanterior view of the filled rectosigmoid is normal. (*B*) Lateral view demonstrates questionable extrinsic pressure effect on the anterior wall of the rectosigmoid junction. (*C*) Posteroanterior view following evacuation demonstrates clearly the extrinsic pressure effect over the right lateral wall of the rectum.

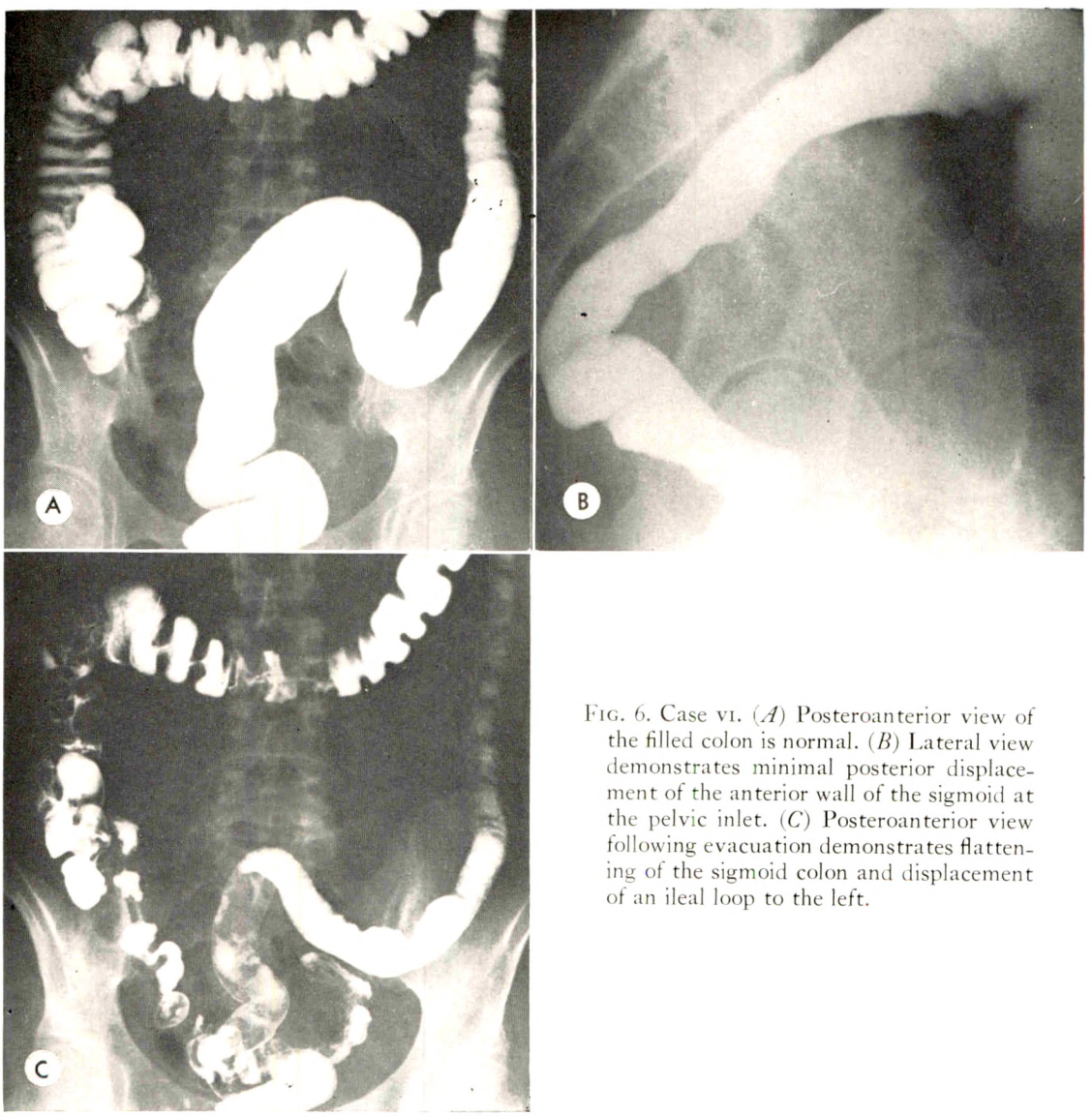


Fig. 6. Case VI. (*A*) Posteroanterior view of the filled colon is normal. (*B*) Lateral view demonstrates minimal posterior displacement of the anterior wall of the sigmoid at the pelvic inlet. (*C*) Posteroanterior view following evacuation demonstrates flattening of the sigmoid colon and displacement of an ileal loop to the left.

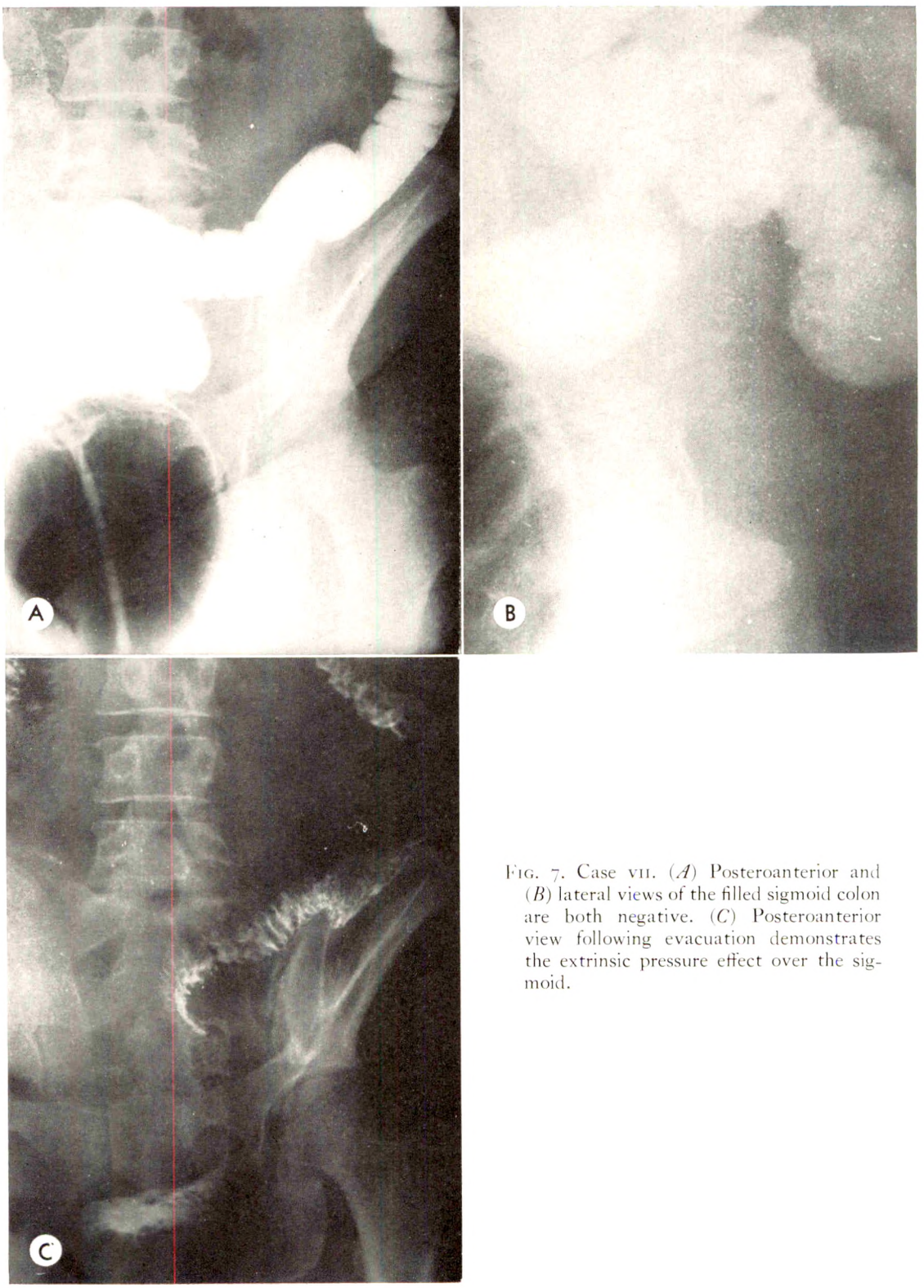


Fig. 7. Case VII. (*A*) Posteroanterior and (*B*) lateral views of the filled sigmoid colon are both negative. (*C*) Posteroanterior view following evacuation demonstrates the extrinsic pressure effect over the sigmoid.

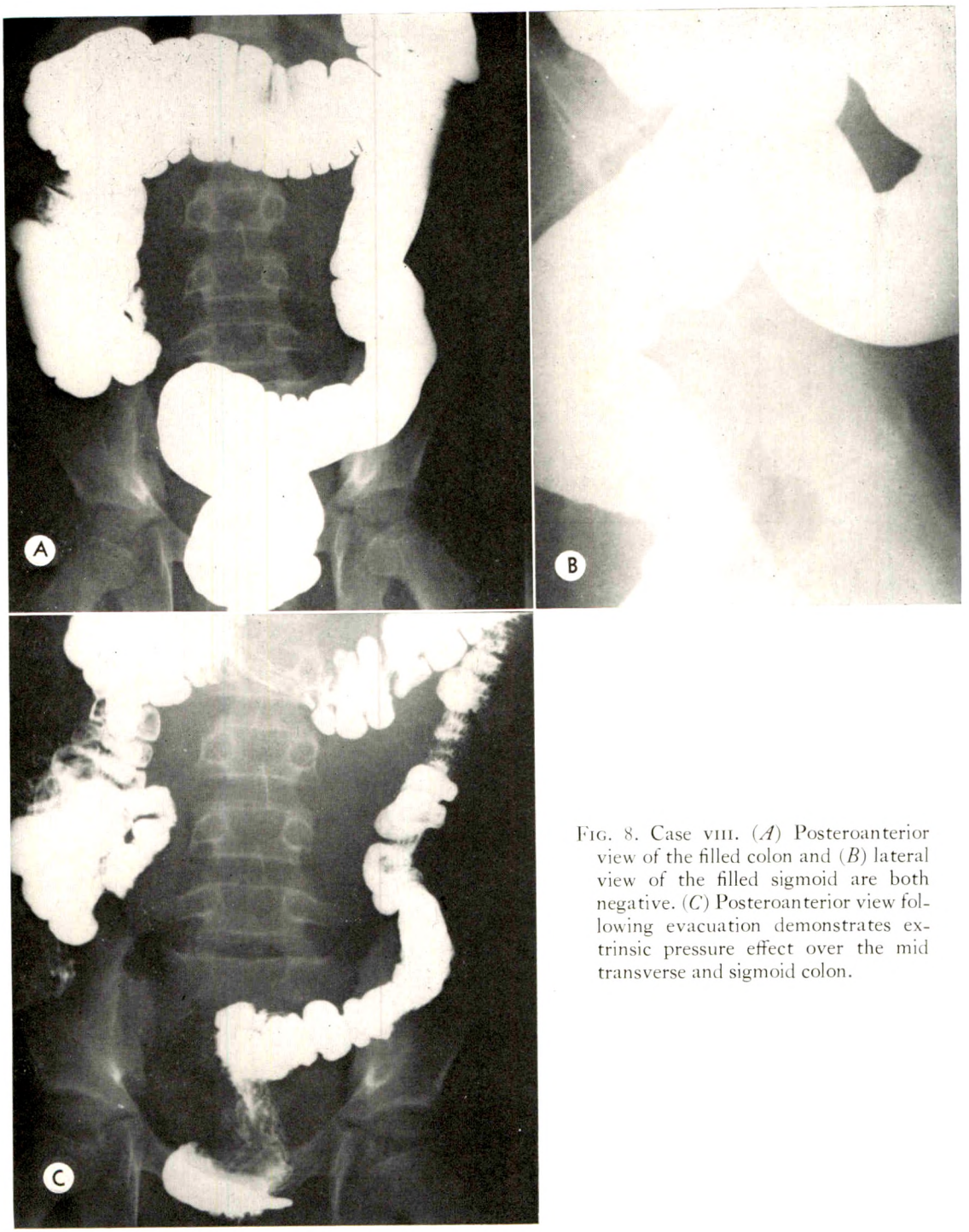


Fig. 8. Case VIII. (*A*) Posteroanterior view of the filled colon and (*B*) lateral view of the filled sigmoid are both negative. (*C*) Posteroanterior view following evacuation demonstrates extrinsic pressure effect over the mid transverse and sigmoid colon.

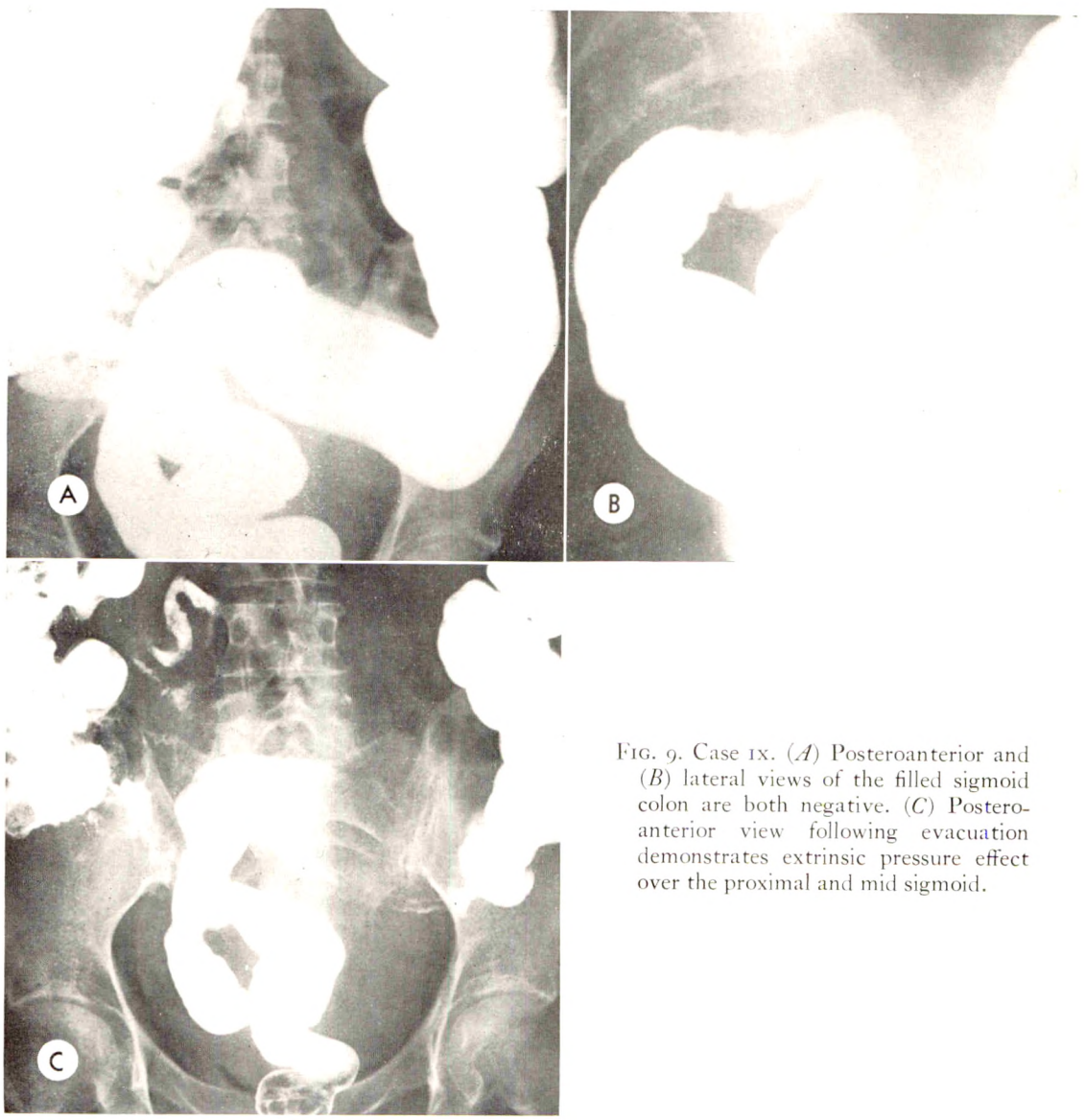


Fig. 9. Case ix. (*A*) Posteroanterior and (*B*) lateral views of the filled sigmoid colon are both negative. (*C*) Posteroanterior view following evacuation demonstrates extrinsic pressure effect over the proximal and mid sigmoid.

Case viii. A 16 year old male presented with a history of fever, weight loss, pallor and abdominal distention. Abdominal examination revealed marked ascites and a tender mass at the level of the umbilicus.

Extrinsic pressure effect at the mid transverse colon and displacement of the distal sigmoid to the left was seen only on the post-evacuation roentgenogram (Fig. 8, *A–C*).

At operation matted enlarged mesenteric lymph nodes were found.

A biopsy revealed lymphoma.

Case ix. A 50 year old obese female presented with a history of low abdominal pain. No mass was palpable on abdominal examination.

Inferior displacement of the proximal sigmoid and probable right lateral displacement of the mid sigmoid were seen only on the post-evacuation roentgenogram (Fig. 9, *A–C*).

Pelvic examination revealed a large left pelvic mass.

At operation a large adenocarcinoma of the left ovary was found, with no invasion of the colon.

SUMMARY

The barium enema examination is a valuable routine roentgenologic procedure

in the detection of extracolonic, intra-abdominal, and, particularly, intrapelvic pathology.

Nine cases are presented to illustrate that the routine postevacuation postero-anterior roentgenogram may be the only view to indicate the presence of an extra-colonic lesion.

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LYMPHANGIOMA*

A RARE PELVIC MASS LESION

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LYMPHANGIOMA is one of the rare entities presenting as a pelvic mass. Occasionally it causes displacement of adjacent organs. The following case report describes such a clinical situation.

REPORT OF A CASE

A 66 year old housewife was admitted to the hospital for investigation of the source of hematest-positive stools discovered at the time of her annual examination. She had been taking aspirin tablets frequently at night for the relief of suboccipital throbbing headaches. Fifteen months earlier her physician had discovered a hemoglobin of 9 grams and ordered Trinsicon, to which she attributed dark colored stools. She was free of abdominal pain, nausea, vomiting or diarrhea. Upon admission the hemoglobin was found to be 11.1 grams per cent, hematocrit 34 per cent, reticulocyte count 3 per cent, normal white blood cell count, and a sedimentation rate of 42 per hour.

Sigmoidoscopy revealed an edematous rectal mucosa; narrowing prevented examination beyond the rectosigmoid junction.

Barium enema examination revealed the proximal sigmoid colon to be elevated by a smooth, extrinsic soft tissue mass which occupied the left pelvis (Fig. 1). No intrinsic lesion of bowel was identified, and the mucous membrane appeared intact. An upper gastrointestinal series was normal. An intravenous urogram revealed medial displacement of the lower left ureter and a soft tissue left pelvic mass above the bladder (Fig. 2). A pelvic arteriogram disclosed a nonspecific type of hypervascularity localized to a small area of the left pelvis associated with displacement of the pelvic arterial branches in the same area (Fig. 3).

Under anesthesia, examination revealed a normal-sized uterus in third degree retroverted position and adnexal thickening of an ill-defined nature. Laparotomy showed dilated lymphatic channels on the posterior aspect of the uterus, on the left broad ligament and hypogastric artery, on the surface of the ovaries, and retroperitoneal to the proximal sigmoid colon. The remaining abdominal organs were palpated and failed to reveal abnormalities. Biopsies taken of posterior uterus, left and right ovaries, and a left peri-aortic lymph node demonstrated the lymphatics to be markedly dilated in the uterine myometrium and the peri-aortic node, and the ovaries to be normal (Fig. 4, *A* and *B*).



FIG. 1. Barium enema roentgenogram. The sigmoid colon is elevated by a smooth extrinsic mass in the left pelvis.

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Postoperative Course. Five months following this exploratory operation, the patient considered her general condition improved over the preoperative state, although pelvic examination revealed no changes since the initial evaluation. She has remained asymptomatic.

DISCUSSION

Case reports of this entity are relatively rare, but the lesion can occur intraabdominally[6] and present as a mass in the pelvis,[5] ileum,[7] mesentery, retroperitoneum, greater omentum, or spleen.[3,4] It has been described in the duodenum presenting as an intraluminal or intramural filling defect.[2] These tumors are sometimes discovered at surgery or autopsy, as they usually are benign and often asymptomatic for years. Patients may become symptomatic, if the lesion enlarges and causes compression and/or displacement of adjacent organs.

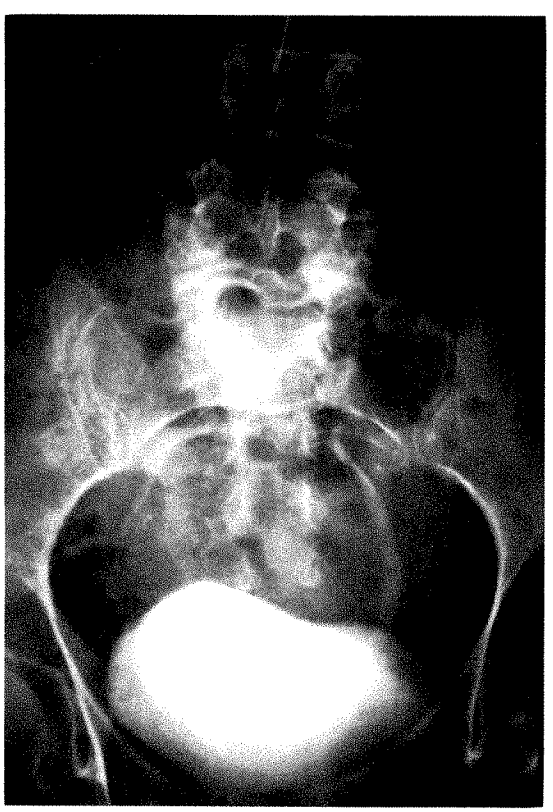


FIG. 2. Intravenous urogram. The left lower ureter is displaced medially and slightly dilated. A soft tissue pelvic mass deforms the left superior portion of the bladder.



FIG. 3. Pelvic arteriogram (late arterial phase). There is slight displacement and luminal irregularity of a major hypogastric branch supplying the left pelvis (straight arrow). Numerous small irregular vessels can be identified in the area of the soft tissue mass (curved arrow). The vascular pattern is distinctly abnormal when compared to the right side.

Lymphangioma is a lesion which has been classified microscopically into 3 basic types.

1. Lymphangioma simplex (capillary lymphangioma): A circumscribed swelling with an ill-defined margin, composed of dilated lymph vessels with a richly cellular connective tissue stroma.

2. Lymphangioma cavernosum: A common type composed of dilated lymphatic sinuses, usually filled with lymph, but sometimes empty and, in rare instances, filled with blood.

3. Lymphangioma cysticum: A thin-walled cystic structure lined by endothelium. It may be unilocular, but often contains subsidiary cysts of varying sizes and

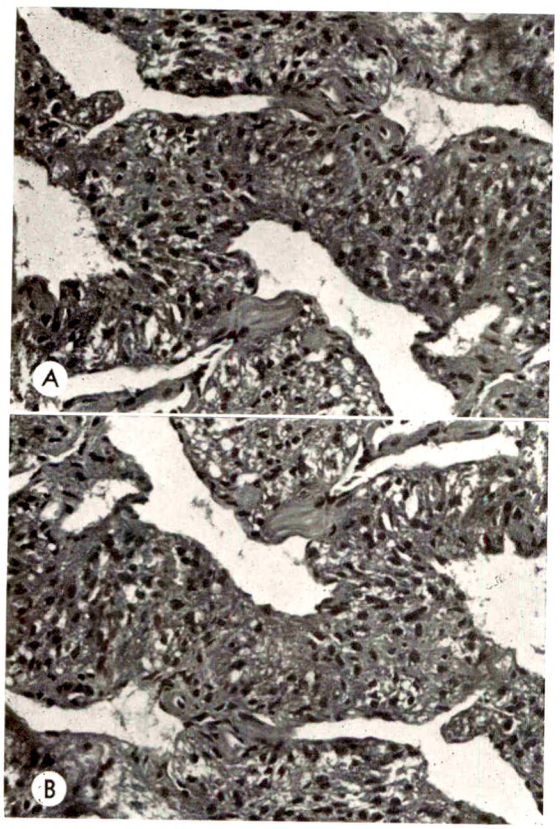


Fig. 4. (*A* and *B*) Histologic sections. Numerous dilated lymphatic channels are interspersed between normal uterine tissue (250×).

shapes which may intercommunicate or be separated by fibrous septa.[5]

Usually lymphangioma is a more or less circumscribed tumor regardless of its basic histologic type, but in this instance it could only be categorized as a nondescript mass by clinical evaluation, and the operative findings revealed a rather diffuse accumulation of dilated lymphatic tissue throughout the pelvis, not lending itself to further histologic categorization.

The positive stool hematest which led to this patient's study was never explained satisfactorily by subsequent findings, except for known aspirin ingestion. Any benign or malignant pelvic mass could have produced the findings of sigmoid displacement noted on the abnormal barium enema study.

It was difficult to explain our angiographic observations in this particular instance. Arteriograms were evidently not obtained on any of the previously reported cases and therefore we did not know what kind of vascular pattern to expect. Abrams' discussion of benign splenic tumors including lymphangiomas notes that they are usually round or oval, displace surrounding arterial branches and result in an avascular parenchymal phase.[1] The mass of lymphangiomatous tissue was apparently too diffuse to cause major vascular displacement, although a smaller pelvic arterial branch was somewhat distorted. We also noted an unusual area of localized hypervascularity in the left pelvis which one would not expect unless part of the diffuse mass contained blood filled sinuses. No biopsy was taken from this site, but tumor or inflammation of the adjacent ovary was ruled out by normal histologic sections.

One must conclude that there are no characteristic roentgenographic findings of lymphangioma regardless of its location. However, nonspecific abnormalities may be noted on barium studies, pyelograms, arteriograms, or a combination of the 3 which can be helpful to the surgeon if symptoms indicate that a laparotomy is necessary.

SUMMARY

A case of pelvic lymphangioma in a 66 year old female is reported.

The pelvic mass was surgically explored upon demonstration by sigmoid displacement on barium enema examination, by intravenous urography and abnormal pelvic arteriography.

This entity must be added to the list of differential diagnostic possibilities when evaluating the patient with obscure pelvic mass.

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HOLLOW VISCUS INJURY IN BLUNT ABDOMINAL TRAUMA*

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THE most common cause of hollow viscus injury in patients with nonpenetrating abdominal trauma is a steering wheel injury.[5] In recent years the increase in automobile accidents has led to an increase in this type of injury. Earlier reports have emphasized the paucity of significant early symptoms, the subtlety of the roentgenographic abnormalities, and the high morbidity and mortality associated with delayed operation.[1,6,10]

MATERIAL

(Table I)

At Wayne County General Hospital 145 patients with blunt abdominal trauma were operated upon from 1969 to 1971. In 28 of these patients (19 per cent) hollow viscus injury was found at operation. Thirteen had perforations of the gastrointestinal tract and 15 had intramural hematomas.

DIAGNOSTIC ACCURACY IN THE ASSESSMENT OF THESE PATIENTS' ABDOMINAL ROENTGENOGRAMS

(Table II)

The correct diagnosis was established prior to operation in only 3 of the 13 patients with perforation. In 4 patients (all with duodenal blow-out) the diagnosis was missed initially but could be made in retrospect. Three patients in whom a perforated viscus was found at surgery had normal abdominal roentgenograms, even in retrospect. One patient had no abdominal roentgenograms. In the final 2 patients the studies were of such poor technical quality that interpretation was not possible.

Two patients with intramural hematoma, both of the duodenum, were correctly diagnosed preoperatively. In 6 patients the diagnosis could not be established from the abdominal roentgenograms, either prospectively or retrospectively. Five patients had no abdominal roentgenograms, and in the final 2 patients the abdominal roentgenograms were of such poor quality that an interpretation could not be rendered.

TABLE I

TYPE AND SITE OF HOLLOW VISCUS INJURY IN 28 PATIENTS

Site	Perforations	Hematomas
Stomach	1	0
Duodenum	4	8
Jejunum	4	4
Ileum	3	3
Colon	1	0

TABLE II

DIAGNOSTIC ACCURACY IN THE ASSESSMENT OF THE ABDOMINAL ROENTGENOGRAMS

Site	Perforation					Hematoma				
	A	B	C	D	E	A	B	C	D	E
Stomach	1									
Duodenum					4	2	4	2		
Jejunum	1	2	1				2	2		
Ileum	1	1		1				1	2	
Colon				1						

A = the correct diagnosis was made preoperatively; B = the roentgenograms were normal, even in retrospect; C = the patient had no abdominal roentgenograms; D = the patient had abdominal roentgenograms of poor technical quality; E = the diagnosis could have been made on abdominal roentgenograms preoperatively but was missed.

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ROENTGEN FINDINGS

PERFORATION

Stomach. The only patient in this series with a ruptured stomach had a considerable amount of free air in the abdomen (Fig. 1). Gastric rupture is rare because the stomach is a thick-walled and mobile viscus.[8] After a heavy meal, however, distention might make it more susceptible to perforation.

Duodenum. The most common sites of duodenal blow-out are the second and third portions.[2] Because of the retroperitoneal location of these segments of the duodenum, intraperitoneal air would not be expected. The most common roentgen findings are either obscuration of the right psoas muscle outline or bubbly, mottled radiolucencies along the right flank caused by intestinal gas dissecting into the right perirenal area and right psoas muscle.[11] Both of these findings were present in 3 of the 4 patients with duodenal blow-out. In 1 patient the only abnormality was obscuration of the right psoas muscle outline (Fig. 2). This finding can be falsely positive if the patient is rotated or if feces are present in the ascending colon. In patients with blunt abdominal trauma, however, obscuration of the right psoas muscle outline should arouse a suspicion of retroperitoneal fluid or blood if there is no rotation or there are feces in the ascending colon and the roentgenograms are of good technical quality. Further appropriate studies, such as an upper gastrointestinal series or intravenous urography, should be performed. The mottled radiolucencies in the right flank are often mistaken for feces and gas in the ascending colon. In 3 patients in whom this finding was present, the mottled radiolucencies were fairly uniform in appearance and unchanged by positioning, as shown on the supine and upright roentgenograms (Fig. 3, *A* and *B*). By contrast, the gas and feces in the ascending colon usually contain some elements of solid density and change position on the supine and upright roentgenograms. In 1 of the 4

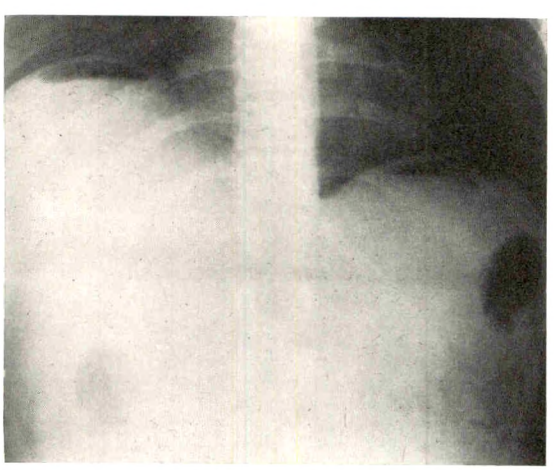


Fig. 1. Ruptured stomach in a 28 year old man, caused by an automobile accident. Free air is present under the diaphragm.

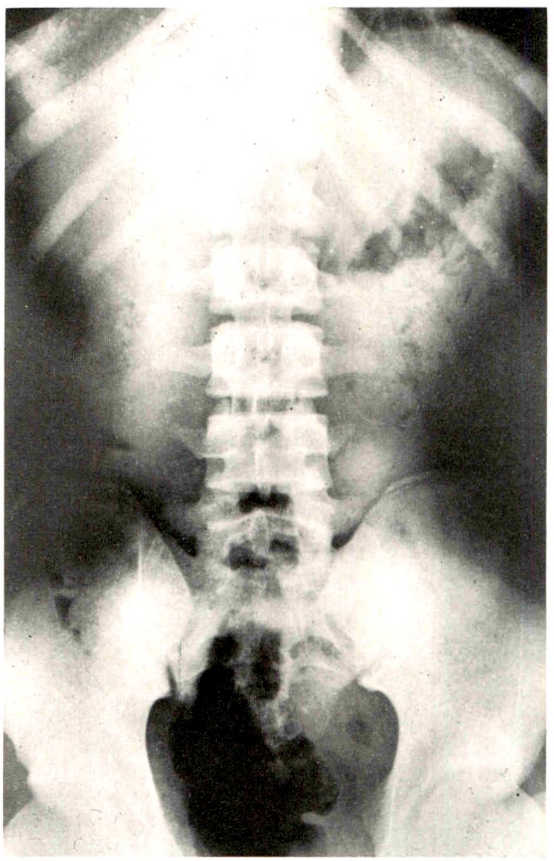


Fig. 2. Retroperitoneal hematoma and blow-out injury of the third portion of the duodenum in a 19 year old man, caused by an automobile accident. The right psoas muscle outline is poorly delineated.

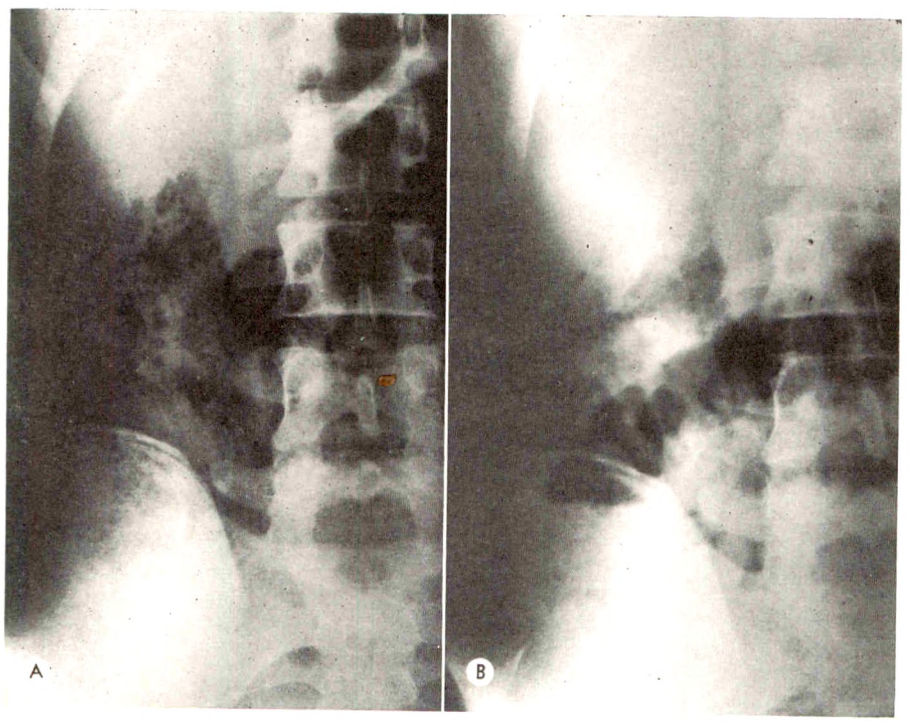


Fig. 3. Duodenal blow-out injury in a 24 year old man, caused by an automobile accident. (*A*) Bubbly radiolucencies are present in the right flank on a supine roentgenogram, and the right psoas muscle outline is obscured. (*B*) On the upright roentgenogram, a fluid level is present in the cecum. The bubbly radiolucencies do not change position compared with the supine roentgenogram. At operation, the third portion of the duodenum was found to be ruptured, the right retroperitoneal soft tissues were necrotic, and an abscess was present in the right pericolic gutter.

patients the perforation of the third portion of the duodenum was missed at operation. Postoperatively, she had a stormy course with spiking fever. One week following operation, an upper gastrointestinal series showed rupture of the third portion of the duodenum with a fistula and abscess (Fig. 4). This patient illustrates that a duodenal blow-out injury may even be missed at the time of operation.

Jejunum. In 1 patient with jejunal rupture, the correct diagnosis was made preoperatively because of amorphous, mottled radiolucencies beyond the confines of bowel (Fig. 5*A*) in the left upper quadrant of the abdomen on the supine roentgenogram. No free intraperitoneal air was present. An upper gastrointestinal study with water soluble contrast medium showed the site of the perforation (Fig. 5*B*).

Ileum. One of the 3 patients with perforated ileum had a small amount of free intraperitoneal air. The roentgenograms of another patient were of such poor quality that interpretation was not possible. In the final patient the diagnosis could not be made on the roentgenogram, even in retrospect, illustrating that normal abdominal roentgenograms in patients with blunt abdominal trauma do not exclude a bowel perforation.

Colon. The only patient in this series with colon perforation had roentgenograms of such poor quality that a diagnosis could not be rendered. The abnormal roentgenographic findings to be expected from intraperitoneal perforation of the colon are free intraperitoneal air and peritonitis; in retroperitoneal perforation of the colon, free intraperitoneal air would not be expected and the findings would be those of a retroperitoneal abscess.[8]

INTRAMURAL HEMATOMA

The only patients with intramural hematomas who had abnormal roentgenographic findings were those with duodenal hematomas. Two such patients were diagnosed correctly prior to operation because of persistent vomiting, indicating a high small bowel obstruction. Upper gastrointestinal series with water soluble contrast medium showed the typical roentgen findings of an intramural hematoma of the duodenum. The mucosa proximal to the intramural mass was thickened, almost completely obstructing the outflow of the contrast medium. The intramural mass was eccentrically placed and the mucosa over the mass was compressed, resulting in a coil-spring appearance (Fig. 6, *A* and *B*). At operation a 14 cm. in length intramural hematoma was found in the second and third portions of the duodenum. Four patients with duodenal intramural hematomas had no symptoms and their abdominal roentgenograms were normal, even in retrospect. The other 2 patients with duodenal intramural hematomas did not have roentgenograms.

The patients with intramural hematomas in the jejunum and ileum had few clinical symptoms. Two patients with jejunal hematoma and 1 with ileal hematoma did not have abdominal roentgenograms, and in 2 patients with ileal hematoma the studies were of very poor technical quality. The roentgenograms in the 2 patients with jejunal hematomas who had satisfactory studies had normal abdominal roentgenograms, even in retrospect. All of the intramural hematomas in these patients were found incidentally at celiotomy.

DISCUSSION

Injury to a hollow viscus in this series of patients with blunt abdominal trauma is higher than has generally been reported.[6] The relative lack of symptoms early in the course of hollow viscus injury and the masking effect of more obvious accompanying trauma frequently causes the diagnosis to be missed early. Moreover, as shown in our series of patients, the roentgenographic findings on abdominal studies can be absent or very subtle, and abnormalities may be missed even when they are present. Free intraperitoneal air was not a constant or prominent finding. Only the patient with a ruptured stomach and one of the patients with a ruptured ileum had free air. The lack of free intraperitoneal air may be attributed to the following: (1) perforations are usually short, 0.4 to 2 cm. in length;[9] (2) the interval between the injury and the roentgenograms is relatively short; and (3) the healthy, adult small bowel is usually devoid of gas.[6] An additional explanation for the absence of free air in patients with proximal jejunal rupture is that the omentum of the transverse mesocolon may form an effective barrier to the upward escape of gas.[7] In order to im-

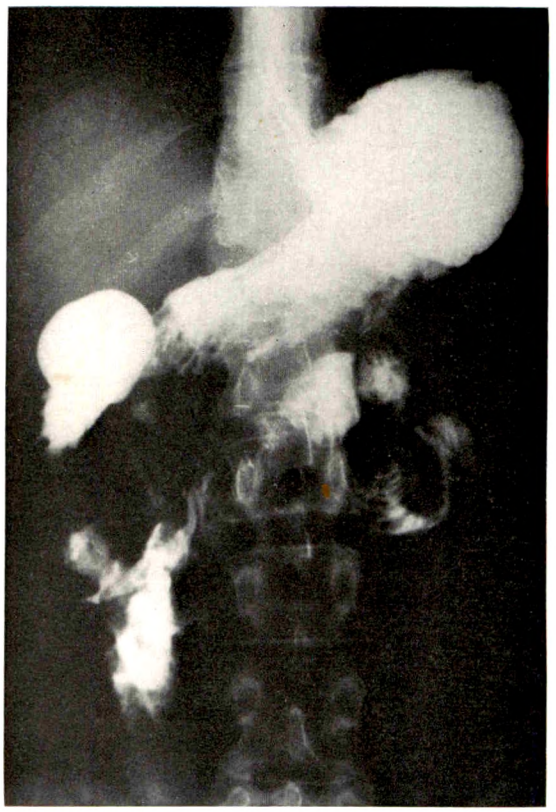


Fig. 4. Duodenal fistula complicating a duodenal rupture in a 35 year old woman. An automobile accident was the cause.

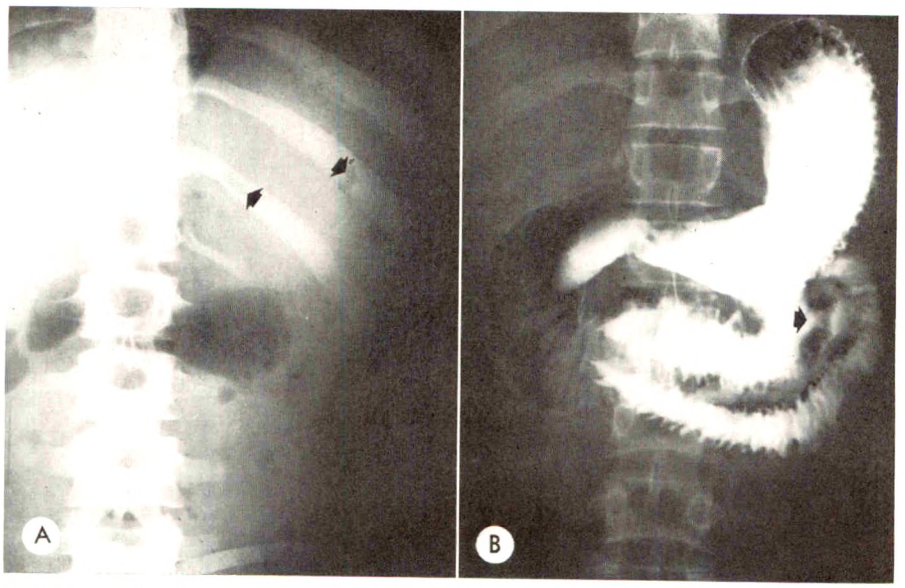


Fig. 5. Jejunal perforation in a 30 year old man injured in an automobile accident. (*A*) Supine roentgenogram. Air bubbles (→) in the left upper quadrant are outside the confines of the gastrointestinal tract. (*B*) Upper gastrointestinal series with water soluble contrast medium shows the site of jejunal perforation (→).

prove the early diagnosis of a ruptured hollow viscus, a careful search must be made for unusual, mottled radiolucencies which do not conform to the pattern or distribution of bowel. The constant position of these air collections in the upright and supine projections confirms their retroperitoneal location in patients with duodenal rupture. Obscuration of the psoas muscle outline should arouse suspicion of a retroperitoneal hematoma or abscess.

Because of the frequently precarious condition of trauma patients, abdominal roentgenograms are often of poor technical quality. At the same time, it is these very patients in whom roentgenograms must be of the highest quality because of the generally subtle nature of the roent-

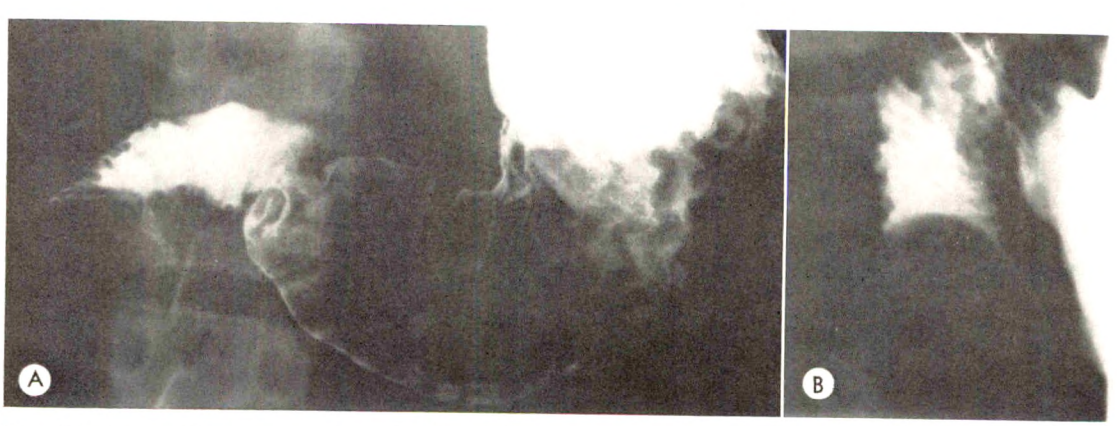


Fig. 6. Intramural hematoma of the duodenum in a 17 year old man injured in a basketball game. (*A*) An upper gastrointestinal series with water soluble contrast medium shows obstruction of the second portion of the duodenum. (*B*) A spot roentgenogram of the duodenal loop shows the eccentric position of the intramural hematoma. The mucosal fold proximal to the obstruction is thickened, and the mucosa at the site of obstruction is compressed.

genographic abnormalities. If any suspicion of bowel injury is present, an upper gastrointestinal series or a barium enema examination with water soluble contrast medium should be performed. Early diagnosis is the most important factor in reducing the morbidity and mortality in patients with hollow viscus injury.[1]

SUMMARY

The roentgenograms in 28 patients with hollow viscus injury caused by blunt abdominal trauma were reviewed.

The findings re-emphasize the frequently subtle abnormalities found in these patients. Free intraperitoneal air was uncommon. Loss of distinctness of the psoas margin or constant, mottled bubbles of gas lying outside the usual confines of colon warrant further investigation with water soluble contrast medium.

The initial abdominal roentgenograms in these frequently difficult to examine patients must be of the highest diagnostic quality to avoid missing the diagnosis and because of the markedly increased morbidity and mortality that accompanies delayed exploration.

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OSSEOUS METASTASES FROM CARCINOMA OF THE LARGE BOWEL*

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THE roentgenographic appearance of an osseous metastasis from a primary colon neoplasm may simulate an inflammatory process, masquerade as a primary bone tumor such as chondrosarcoma, or mimic findings usually associated with osteoblastic secondary deposits from the prostate, breast or lymphoma.

This report presents the roentgenographic findings in 8 cases of symptomatic osseous metastatic disease, primary in the large bowel. In all cases, the bone lesions were biopsied and histologic sections of the metastasis and primary lesion were reviewed by the same pathologist and considered to be essentially identical in appearance. The osseous lesion in every case in this series showed an area of increased density within the bone or calcium deposition in the portion of the mass extending into the soft tissue.

REPORT OF CASES

CASE I. A 46 year old man was referred for roentgenographic study because of pain in the left buttock and weight loss. The pain began 4 months earlier, had localized in the left hip and buttock, and gradually became more severe.

Roentgenographic study led to the tentative diagnosis of chondrosarcoma (Fig. 1).

Biopsy of the pelvic lesion was interpreted as mucus secreting adenocarcinoma originating in the bowel.

A barium enema examination revealed a lesion in the sigmoid colon with a histologic appearance identical to that of the metastasis.

CASE II. A 30 year old man with a strong family history for colon carcinoma had multiple polyps of the colon resected over the previous 4 years. Segmental resection of the sigmoid was done for Grade II adenocarcinoma, which did not appear to extend beyond the submucosa.

One year after surgery, roentgenographic examination of the pelvis (Fig. 2) was done because of perineal pain. There was destruction of the left ischium with a large soft tissue mass extending into the thigh containing scattered calcified areas. The roentgenographic appearance suggested a primary bone neoplasm such as chondrosarcoma.

Biopsy of the mass revealed adenocarcinoma with a histologic appearance similar to that of the previously resected sigmoid colon carcinoma.

CASE III. A 55 year old woman had a 6 month history of pain in the left shoulder radiating down the arm. A previous diagnosis of subdeltoid bursitis had been made on the basis of

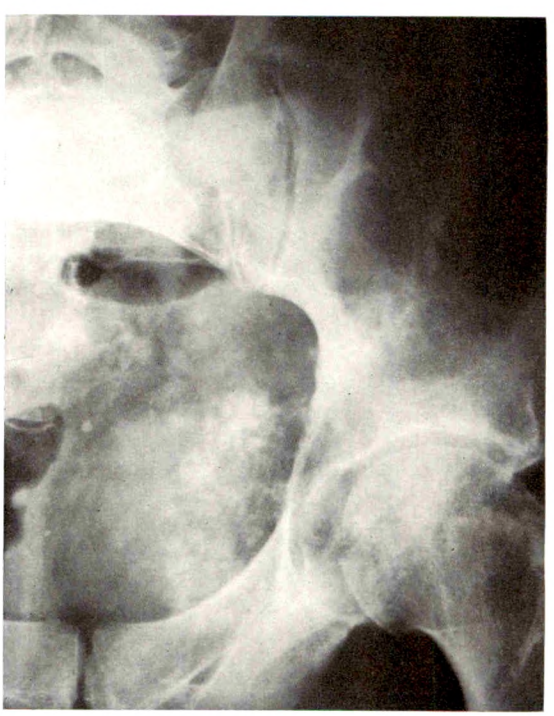


FIG. 1. Case I. The lesion involves the acetabulum and there is a large soft tissue mass which displaces the rectum. The appearance of this lesion suggested chondrosarcoma.

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soft tissue calcification near the shoulder. No relief was obtained after treatment with roentgen ray, diathermy, and ultraviolet light. Subsequently, an indurated mass developed over the left scapula with no lymphadenopathy.

A biopsy of the axillary border of the scapula disclosed metastatic adenocarcinoma.

A barium enema examination revealed a lesion in the sigmoid colon and incomplete obstruction of the bowel. The lesions in the scapula (Fig. 3) and colon had an identical histologic appearance.

Case iv. A 65 year old man noted increasing constipation. Barium enema study revealed a lesion of the transverse colon.

Fourteen months after resection of the bowel carcinoma, a mass was noted in the region of the left hip. There was a fracture of the upper femoral shaft and the acetabulum was involved as shown in Figure 4.

Biopsy of the metastasis showed a histologic appearance identical to that of the primary lesion.

The roentgenologic appearance suggests chondrosarcoma.

Case v. A 53 year old woman was admitted for metastatic disease 2 years after resection of a primary carcinoma of the sigmoid colon with extension to the serosa. Metastasis to a single abominal lymph node was seen at the time of surgery.

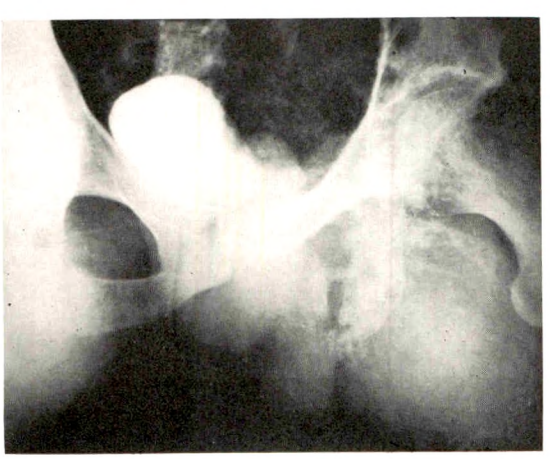


Fig. 2. Case ii. Involvement of both ischia. On the left there is extensive destruction of bone and a soft tissue mass containing considerable calcium. The right ischium shows sclerosis with loss of trabecular architecture.

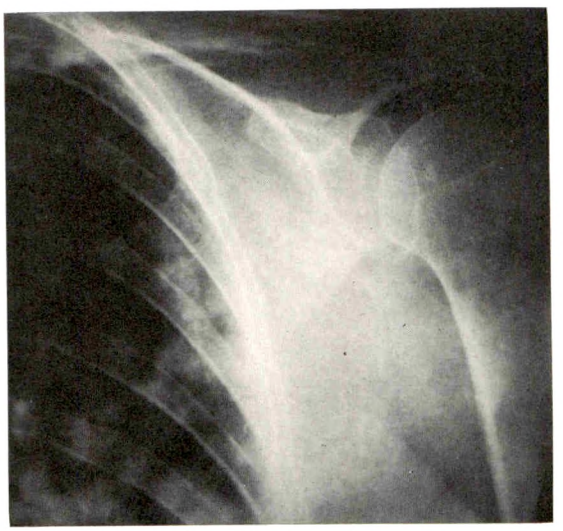


Fig. 3. Case iii. The scapula shows a large area of destruction with an associated soft tissue mass containing calcium. There are also metastases to the lung. The roentgenographic appearance of the scapula would suggest a primary bone tumor with extension to the soft tissue.

The first symptomatic metastasis was in the ilium (Fig. 5*A*), with later foci diagnosed in the lung, liver and left foot (Fig. 5*B*).

The histologic appearance of all lesions biopsied was similar to the primary lesion.

Case vi. A 64 year old woman had the sig-

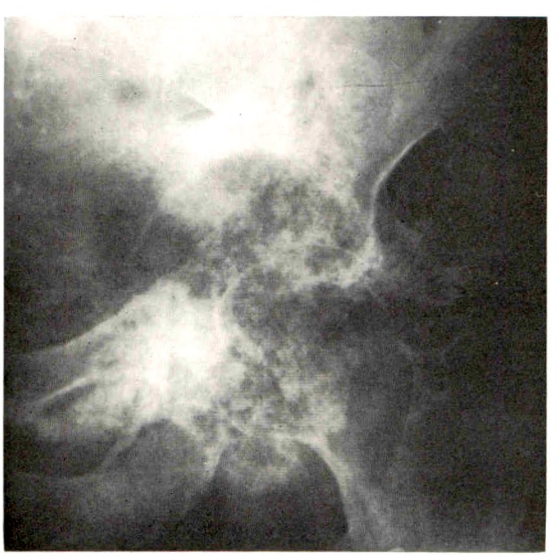


Fig. 4. Case iv. Extensive destruction of the pelvis adjacent to the acetabulum with considerable calcium present in the soft tissue mass.

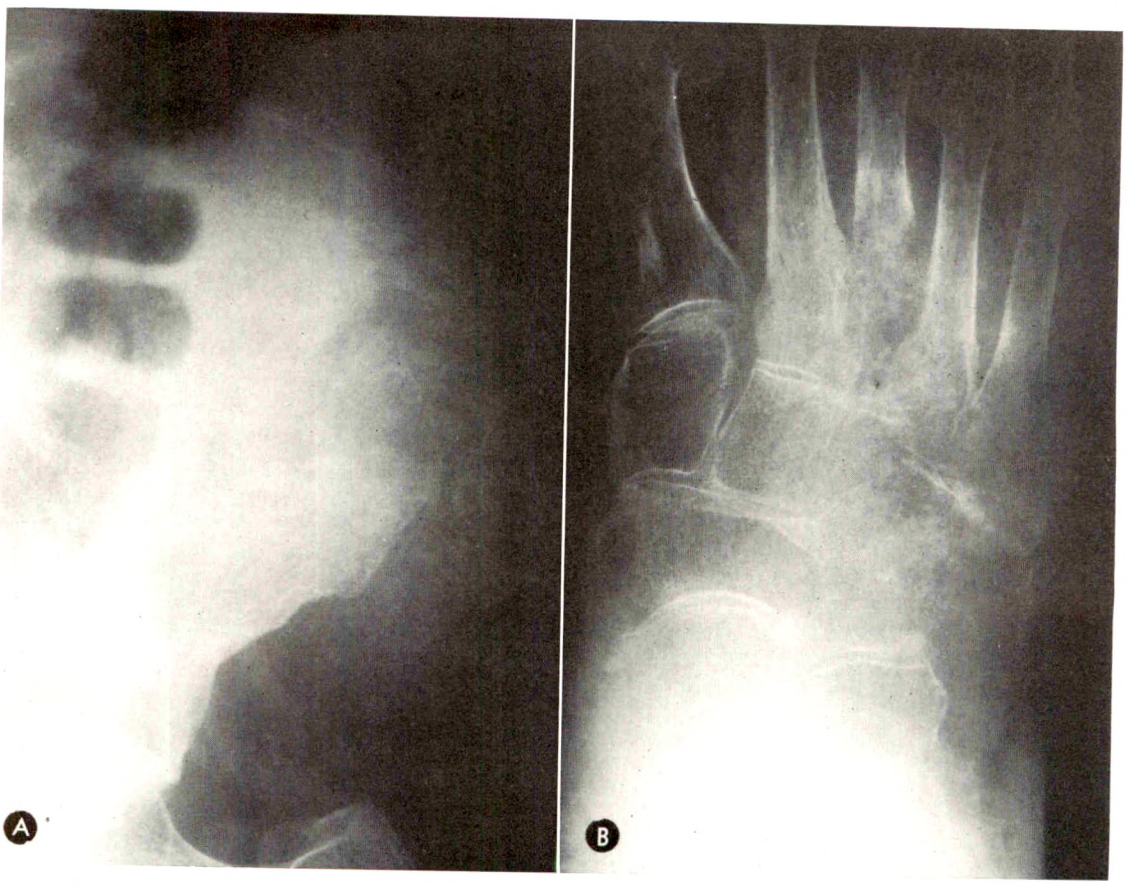


Fig. 5. Case v. (*A*) An expanding lesion of the ilium with an associated soft tissue mass. Calcium can be seen in the mass which has a roentgenographic appearance similar to chondrosarcoma. (*B*) The lesion in the foot is predominantly osteolytic and has involved the cuboid, cuneiform, navicular and 3 metatarsal bones.

moid resected for carcinoma 2 years earlier. Seven months after surgery, the patient developed back pain and was given palliative radiation therapy to the lumbar spine even though the roentgenographic study did not demonstrate overt metastatic disease. Twenty months after her colon resection, an enlarged liver was palpated.

Roentgenographic examination of the right foot 22 months after surgery showed sclerotic changes in the os calcis and tarsal navicular bone (Fig. 6*A*). Progressive destructive changes were seen in the proximal metatarsal and tarsal bones over the next 3 weeks (Fig. 6*B*).

Aspiration biopsy of the foot revealed adenocarcinoma, similar in appearance to the primary sigmoid colon lesion.

Case vii. A 57 year old man presented with stenotic colostomy, a small pericolostomy hernia, back pain and swelling of the left foot. Three years earlier, this patient had had an abdominal-perineal resection for carcinoma of the rectum. The histologic appearance obtained on biopsy of the foot was similar to the patient's primary lesion.

Figure 7 shows unusual expanded appearance of the metatarsal and 2 phalanges of the great toe. The joint spaces appear preserved. The roentgenographic appearance closely resembles bone lesions of yaws.

Case viii. A 44 year old man had severe back pain.

Roentgenographic examination of the lumbar spine and pelvis (Fig. 8) revealed multiple sclerotic and lytic areas, suggestive of the changes seen with lymphoma or metastases from prostatic carcinoma.

Clinically, carcinoma of the prostate was

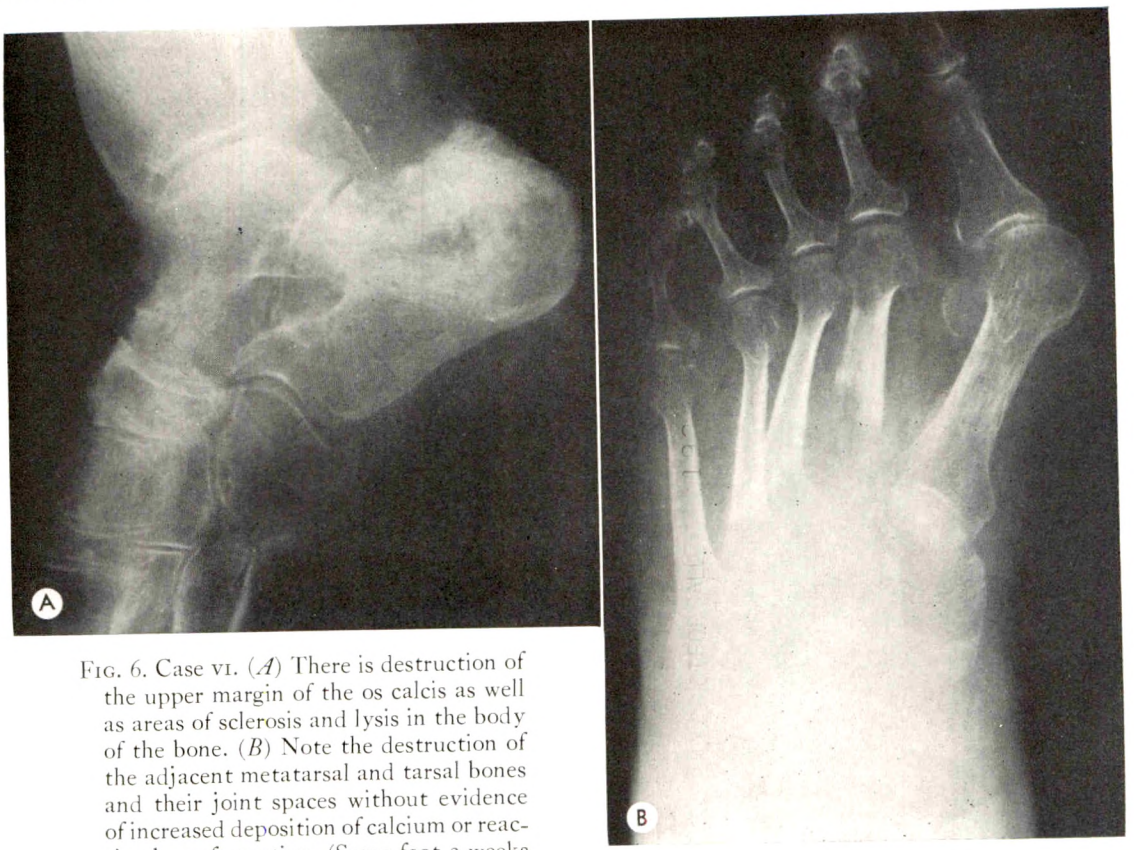


Fig. 6. Case VI. (*A*) There is destruction of the upper margin of the os calcis as well as areas of sclerosis and lysis in the body of the bone. (*B*) Note the destruction of the adjacent metatarsal and tarsal bones and their joint spaces without evidence of increased deposition of calcium or reactive bone formation. (Same foot 3 weeks later.)

thought to be a possibility, in spite of normal serum acid phosphatase levels. Eight years previously a portion of the patient's colon had been resected for carcinoma.

Aspiration biopsy of the right sacroiliac area was described as metastatic carcinoma of uncertain histologic type. The pathologist suggested renal or prostatic primary lesions.

The patient became paraplegic 10 months later and a laminectomy in the lower thoracic region showed invasion of the laminae by tumor and compression of the spinal cord.

Histologic examination of material from the laminae and spinal cord suggested primary sites in the thyroid or lung.

The patient died 11 months following laminectomy. On postmortem examination a recurrent colon carcinoma, 26 cm. proximal to the anus at the old resection line, was seen. Multiple lymph nodes were replaced by tumor and massive involvement of the lumbar vertebrae and pelvis was described.

Review of the histologic sections from the original colon resection, the biopsy of the sacroiliac joint, the laminectomy and the postmortem study resulted in the conclusion that all of the lesions had a common origin—the colon.

DISCUSSION

Metastasis to the skeleton from large bowel primary neoplasm has been the subject of sporadic case reports.[1–4,6,7] Such metastases are more frequently noted as incidental findings in postmortem examinations than symptomatic lesions in living patients. Although primary carcinoma of the gastrointestinal tract has a documented potential for the production of osteoblastic change in bone[1,4] and soft tissue ossification,[3] previous reports did not suggest the predominance of these findings over purely lytic lesions. A soft tissue mass with calcification or increased

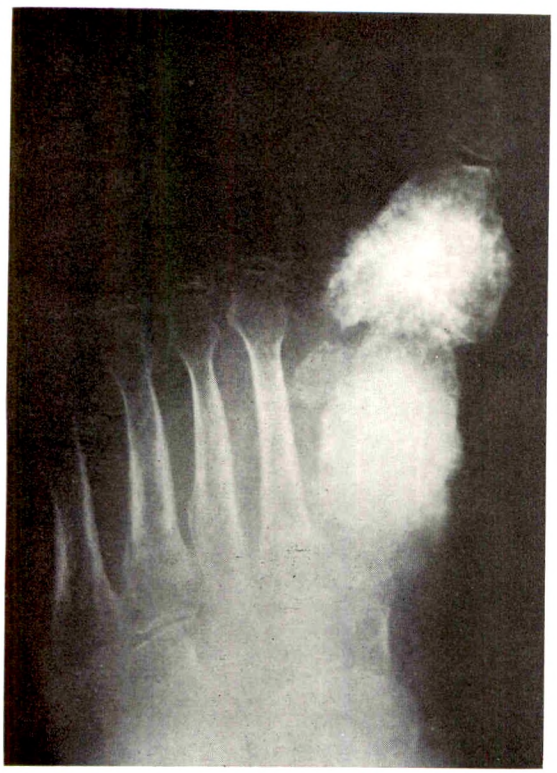


FIG. 7. Case VII. Bizarre expanded appearance of the metatarsal and 2 phalanges of the great toe. The joint spaces appear preserved.

density of the bone is present in all of the cases reported.

In 3 of these cases, lesions were found in the foot. The bizarre appearance of the lesion in Case VII was typical of yaws. The author offers no hypothesis or explanation for this most unusual location, but notes that one of the rather small number of lesions reported in the literature was also in the foot[6] and another in the tibia.[1]

CONCLUSION

Symptomatic osseous metastases from carcinoma of the colon are uncommon.

The roentgenographic appearance of these lesions is frequently osteoblastic. Even when they are predominantly osteolytic, reactive changes are also provoked within the bone, periosteum, and in soft tissue masses. The resemblance of these unusual metastases to primary bone tumors may be striking.



FIG. 8. Case VIII. Numerous osteoblastic lesions involving the sacrum, ilium and vertebral bodies. Many similar lesions were present in the thoracic vertebrae. The roentgenographic appearance of the lesions suggested Hodgkin's disease or a primary focus in the prostate.

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ROENTGENOLOGIC DEMONSTRATION OF SPONTANEOUS REVERSAL OF PORTAL BLOOD FLOW IN CIRRHOSIS AND PRIMARY CARCINOMA OF THE LIVER

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THE hemodynamics of liver blood flow are altered in cirrhosis as the disease progresses, and similar changes occur in primary carcinoma of the liver, since the latter often arises on the basis of the former. They include portal hypertension, reduced portal blood flow due to increased intrahepatic resistance, compensatory increase of hepatic arterial blood flow, and development of intra- and extrahepatic shunts which divert blood from the liver parenchyma.[18]

In 1959, Warren and Muller[22] studied hemodynamics in cirrhotic patients during portacaval shunt operations, and found in 3 out of 8 patients, a sinusoidal pressure higher than the free portal pressure preoperatively. In 1 such patient with a complete oxygen study, the oxygen saturation increased in the lower portal vein blood after a side-to-side anastomosis. Based on these findings, they suggested the possibility of spontaneous conversion of the portal vein to the outflow tract in a cirrhotic liver. A similar suggestion was made also by Hales *et al.*[8] who demonstrated, using the injection-corrosion technique, consistent enlargement of the hepatic arteries and arterial beds with an increased intercommunication between hepatic arteries and portal veins in cirrhotic livers. By wedged hepatic venography, Gliedman *et al.*[7] demonstrated filling of the intrahepatic portal veins following injection of contrast medium and construed it to be evidence of a retrograde portal blood flow. However, Britton *et al.*[2] showed cinephlebographically that such retrograde portal flow occurred only during forceful injection of the contrast medium, and that the contrast medium swept back toward the sinusoids upon discontinuation of the injection. During shunt operations in 85 patients with cirrhosis, Moreno and his associates[15] found in 7, a to-and-fro motion of the portal blood with zero net flow as measured with a flow-probe. In none of these patients was there a continuous retrograde flow in the portal vein, however. Reynolds and Redeker,[18] based on their data, also favored continued forward portal perfusion of the liver in the unoperated cirrhotic patients. Despite the more recent publications by Warren *et al.*[21,23] based on angiographic observations, the question of whether or not in advanced cirrhosis with portal hypertension retrograde portal flow occurs spontaneously remains largely unsolved, because of the lack of unequivocal or direct demonstration of the reversed portal flow before portacaval shunt operations.

In the course of our roentgenologic studies of 96 patients with cirrhosis and/or

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hepatoma, the portal vein was visualized during celiac arteriography and continuously in the venous phase in 5 with hepatoma and in 1 with cirrhosis. In the latter, an unequivocal retrograde portal flow was evident by the stop and push-back of the contrast medium injected into the portal vein through a catheter inserted in a mesenteric vein, and by a retrograde flow of the contrast medium during umbilicoportography. Thus, reversal of the portal flow does occur in cirrhosis spontaneously, but very rarely, and it occurs more often in primary liver cell carcinoma which has a tendency to grow in the hepatic vein,[5] causing an outflow block.[16] Retrograde portal flow in hepatoma has not been emphasized in the past reports of angiographic studies.[1,11,25] It was also found that nonvisualization of the splenic and portal veins on splenoportography represented, more often than not, a two-way movement of the portal flow.

MATERIAL AND METHOD

A total of 96 patients consisting of 49 with cirrhosis and 47 with proven hepatoma, who were admitted to Kurume University Hospital, Chiba University Hospital or one of the affiliated hospitals, were subjected to at least two of the following angiographic examinations, combined with a barium swallow study specifically designed for the detection of esophagogastric varices.

Splenoportography was carried out in 63 patients who had had no bleeding tendency. The pretreatment consisted of intramuscular injection of 35 mg. of meperidine hydrochloride and 0.25 mg. of atropine 30 minutes prior to the examination. A 19 gauge venous needle with a stop-cock was inserted 1.5 to 2 cm. into the spleen through the 9th or 10th intercostal space in the left midaxillary line. The needle was connected to a syringe through a vinyl tubing and pumping was carried out with saline untill a smooth flow of blood from the spleen was attained. The syringe was changed to another one containing 76 per cent urografin (Na- and meglumine diatrizoate) and serial films were exposed at 0.5 second intervals, while the contrast medium was injected manually at a maximal possible speed. There was 1 incident in which postprocedural bleeding necessitated splenectomy.

Celiac and superior mesenteric arteriography was carried out in 73 patients with the usual technique using a yellow or green KIFA catheter and 60 per cent urografin. Two to 3 films were exposed per second during the arterial phase, 1 per second during the capillary phase and 1 per 2 seconds thereafter. Hepatic venography was carried out in 52 patients according to the procedure of Celis *et al.*[4] A No. 7 Cournand catheter was inserted from the left antecubital vein, and pressures of the inferior vena cava, free hepatic vein and wedged hepatic vein were recorded before venography. Umbilicoportography was carried out in 22 patients with the procedure in which a transverse incision was made 3–6 cm. above the umbilicus, and a metal probe was inserted into the umbilical vein to break the junction of the umbilical and the left portal vein, followed by catheterization using a 2–3 mm. vinyl tubing.[10] Sixty per cent urografin was used for the visualization of the portal vein. For the demonstration of retrograde flow, the syringe containing the contrast medium, but without the plunger, was raised to the level where the contrast medium flowed forward at a minimal speed, and no force was applied.

In addition to a routine barium swallow examination, a second study for the detection of cardiac varices was carried out in all patients. In this procedure, the fornix and cardia were observed carefully with the patient erect, prone, supine and at angles, and after insufflation of 200–300 ml. air,[9] roentgenograms in many projections were made. In those patients in whom no obvious esophageal varices were found, endoscopy was carried out using an Olympus GTF-A fibergastroscope with the retrograde observation (U-turn) technique for the demonstration of cardiac varices.

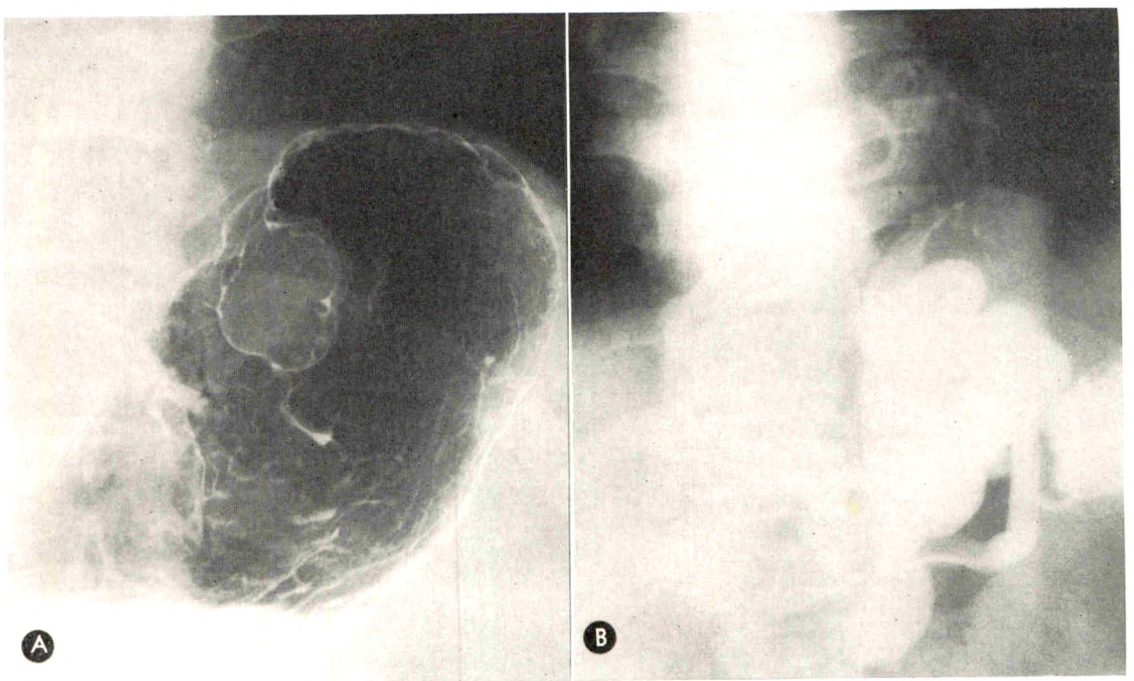


Fig. 1. Case 1. (*A*) A large mass is seen protruding in the cardia above the esophagogastric junction. Winding rugae-like lines across the body may be related to dilated veins. (*B*) Splenoportogram showing huge cardiac varices corresponding to the mass. The splenic vein is visualized only near the spleen, and the incomplete visualization of the main portion of the splenic vein is due to either a thrombus or collision with retrograde blood flow.

REPORT OF CASES

Case 1. F.E., a 52 year old male, was admitted because of a gastric mass and lethargy which developed a week prior to admission. He had been drinking 2 to 5 go's (360–800 ml.) of rice wine daily for the last 25 years, and had a history of ascites 5 years previously. He first noticed abdominal distention and decrease in urine volume about 6 months prior to admission, and recent roentgen examinations made by practitioners revealed a mass in the stomach.

Physical examination disclosed a medium sized man who responded clearly to questions. Vascular spiders were numerous around the neck, the abdomen was distended with positive fluid waves, and there was a slight pretibial edema.

Laboratory studies included: red blood cells 4,200,000/mm.^{3}; white blood cells 5,600/mm.^{3}, with a normal differential count; Hb 14.4 gm. per cent; total bilirubin 1.0 mg. per cent; serum protein 6.7 gm., albumin 2.7 gm., γ-globulin 2.3 gm. per cent; ammonia 260 μg. per 100 ml.; SGOT 40 u. (Karmen); SGPT 20 u.; and zinc turbidity test (ZnTT) 16.2 u.

Roentgen studies disclosed a tumorous varix in the cardia (Fig. 1*A*). Upon splenoportography, the contrast medium did not course in the splenic vein, but mostly ran into the dilated short gastric vein to form massive varices (Fig. 1*B*). This study was interpreted as indicating either thrombosis of the splenic vein or a collision of splenic venous blood with a retrograde flow. Celiac angiography disclosed a markedly dilated common hepatic artery and distorted intrahepatic branches typical of advanced cirrhosis (Fig. 2*A*). Caudad to the hepatic artery and parallel, the portal vein was visualized in the early arterial phase (arrows), and after cessation of injection, the contrast medium continued to flow into the portal vein and further into the collaterals (Fig. 2*B*). Subsequently, splenectomy and devascularization of the paraesophageal and paragastric veins were carried out. During the operation, a catheter was inserted into a branch of the superior mesentric vein and contrast medium was injected with a manual force. The contrast medium entered the portal vein halfway and was stopped short; the remainder flowed into

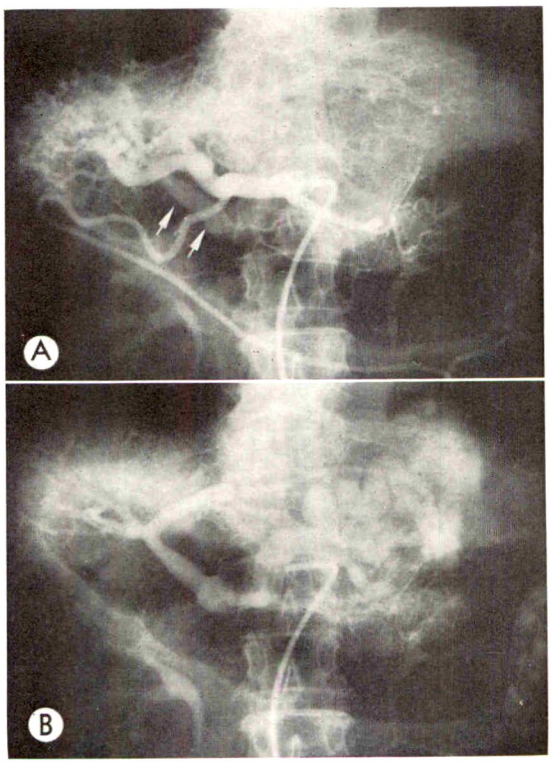


Fig. 2. Case 1. Celiac angiography. (*A*) The branches of the right and middle hepatic arteries show marked winding, and enlargement of the common hepatic artery is prominent. In the arterial phase, the portal vein is already visualized (arrows). (*B*) In the venous phase, the contrast medium continued to flow retrograde and the entire portal vein, the proximal portion of the splenic vein and tortuous gastric varices were clearly seen.

the collaterals through the coronary vein (Fig. 3*A*). Also, the contrast medium flow ceased sharply in the splenic vein, 2–3 cm. from the junction with the superior mesenteric artery, indicating a probable thrombus. The portal pressure was 430 mm. H_2O preoperatively and 340 mm. H_2O postoperatively. Two months after the operation, umbilicoportography was carried out and contrast medium was instilled into the left portal vein with a minimum necessary gravity. There was practically no antegrade flow, and the contrast medium flowed back into the portal vein (Fig. 3*B*).

Case II. T.K., a 58 year old female, was admitted because of abdominal distention. About 2 months prior to admission, she began feeling fatigued, and noticed an increasing abdominal girth. She consulted several doctors whose diagnoses varied from ovarian cyst to peritonitis, and the last physician, who found liver function abnormalities, referred her to us.

On admission, she was moderately nourished, and abdominal distention was marked with fluid waves. No jaundice was noted, but there were vascular spiders, moderate dilatation of the abdominal veins, and (+) pitting edema of the legs.

Laboratory data included: red blood cells 2,850,000/mm.^{3}; white blood cells 3,000/mm.^{3}; Hb 10.4 gm. per cent; prothrombin time 18 sec.; SGOP 60 u.; SPGT 15 u.; alkaline phosphatase 14 u. (K.A.); LDH 355 u. (LDH_4 1.0 per cent, LDH_5 2.8 per cent); total bilirubin 2.0 mg., direct bilirubin 0.9 mg. per cent; total protein 7.1 gm., albumin 3.6 gm., γ-globulin 2.0 gm. per cent; ammonia 90 μg. per cent; urea nitrogen 28 mg. per 100 ml. Alpha-fetoprotein was 20,850 ng./ml. by radioimmunoassay which increased rapidly during the ensuing month and a half. The ascites was yellow and slightly turbid with a protein concentration of 1.6 gm./100 ml. No malignant cells were seen in the sediment.

A liver scan showed a large spleen and a generally poor hepatic uptake of the colloid. No defect was discernible, probably because of the diffuse mottling of the liver. An upper gastrointestinal series disclosed varices in the lower esophagus and the cardia. Celiac arteriography disclosed irregular and marked neovascularizations in the right lobe (Fig. 4*A*), and the portal vein was visualized during the arterial phase. The contrast medium continued to flow retrograde, after cessation of injection, into the large winding collateral vein from the junction with the splenic vein which did not show at any time, suggesting thrombosis. From the beginning of the portal vein, the contrast medium flowed caudad into a small collateral which was traced down into the pelvis. Noteworthy was the presence of 2 small filling defects in the main collateral vein near the porto-splenic junction (Fig. 4*B*).

She responded to antidiuretics and ascites disappeared almost completely after 1 month. On the 45th day, while stepping down from her bed, she suddenly felt dizzy and vomited. She then lapsed into a shock state with sudden increase of the abdominal girth. A hemorrhagic ascites was aspirated. She died several hours later.

Autopsy disclosed a massive hemorrhage from the surface of a hepatoma which had developed on the basis of cirrhosis of the post-

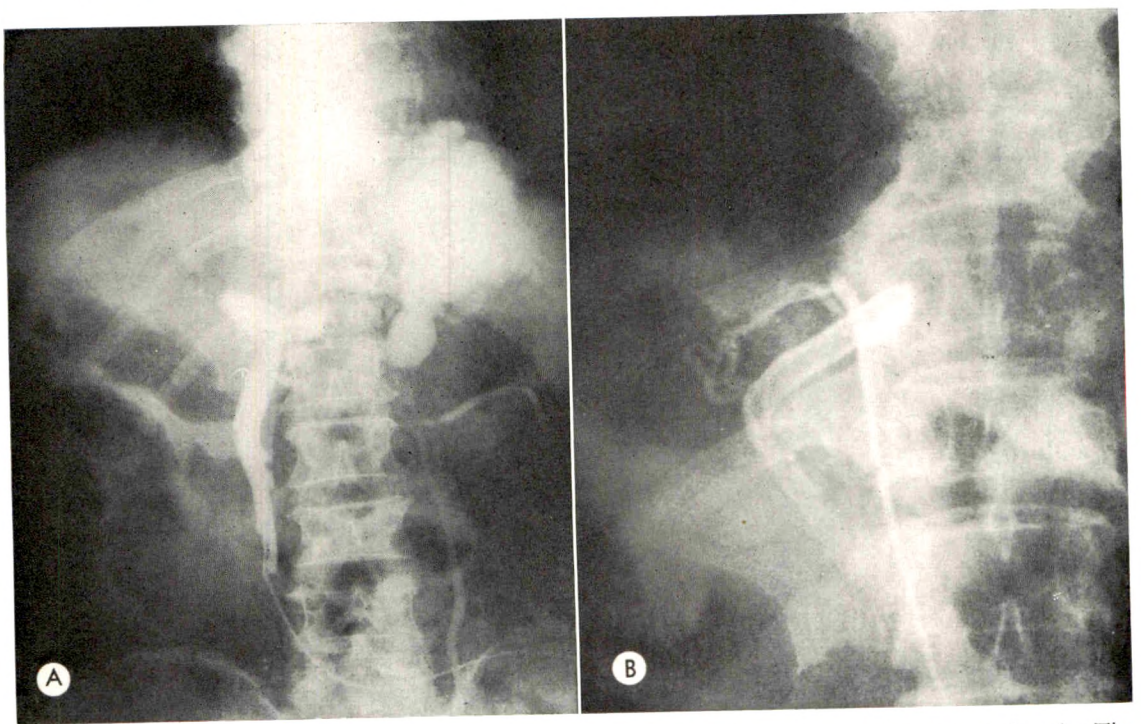


Fig. 3. Case I. (*A*) Operative portography through a catheter passed into the superior mesenteric vein. The flow of contrast medium was stopped halfway in the portal vein, and the contrast medium entered the coronary vein and the varices from a point along the splenic vein a few centimeters to the right of the junction with the superior mesenteric artery. (*B*) Umbilicoportography. Contrast medium which was instilled with a minimum pressure flowed retrograde into the portal vein and a winding collateral vein. No contrast medium flowed antegrade or into the right portal vein.

hepatitic type. As expected from the angiography, tumor thrombi were seen in the portal vein, in the collateral vein and further up in the esophageal varices—a clear indication of retrograde transport of tumor cells from the portal vein. Contrast medium injected from the right hepatic artery flowed into the portal vein (Fig. 5, *A* and *B*).

Case III. M.K., a 47 year old male, was admitted because of epigastric pain and abdominal distention. He had had a history of jaundice 15 years previously. He began feeling tired and noting distention of abdomen 2 months prior to admission. The abdominal distention was marked with a positive fluid wave. A firm liver was palpated 2 fingerbreadths below the right costal margin with an uneven surface and edge, but the spleen was not felt.

Laboratory studies disclosed: SGOT 27 u.; SGPT 22 u.; alkaline phosphatase 19.5 u.; total protein 7.8 gm., γ-globulin 1.6 gm. per cent; total cholesterol 299 mg. per cent; total bilirubin 1.6 mg., and direct bilirubin 0.8 mg. per 100 ml. Alphafetoprotein was positive by the single radial immunodiffusion method. A liver scan showed several large defects in the right lobe and in the middle area, and needle biopsy aimed at a defect disclosed liver cell carcinoma.

Celiac angiography demonstrated marked neovasculature immediately cephalad to the common hepatic artery, and the left and middle hepatic arteries were not discernible (Fig. 6*A*). In the capillary phase, the contrast medium started flowing into the portal vein, and the dilated portal vein was apparent, with a filling defect at the point where the left vein joins (arrow), and a tumor growth in one of the branches of the right portal vein was also seen (Fig. 6*B*).

Case IV. T.Y., a 44 year old man, came to the hospital because of milk jaundice and a recent weight loss of 3 kg. He began having a slight fever and malaise 3 months prior to admission, and a right upper quadrant pain with mild jaundice about a month later. On physical

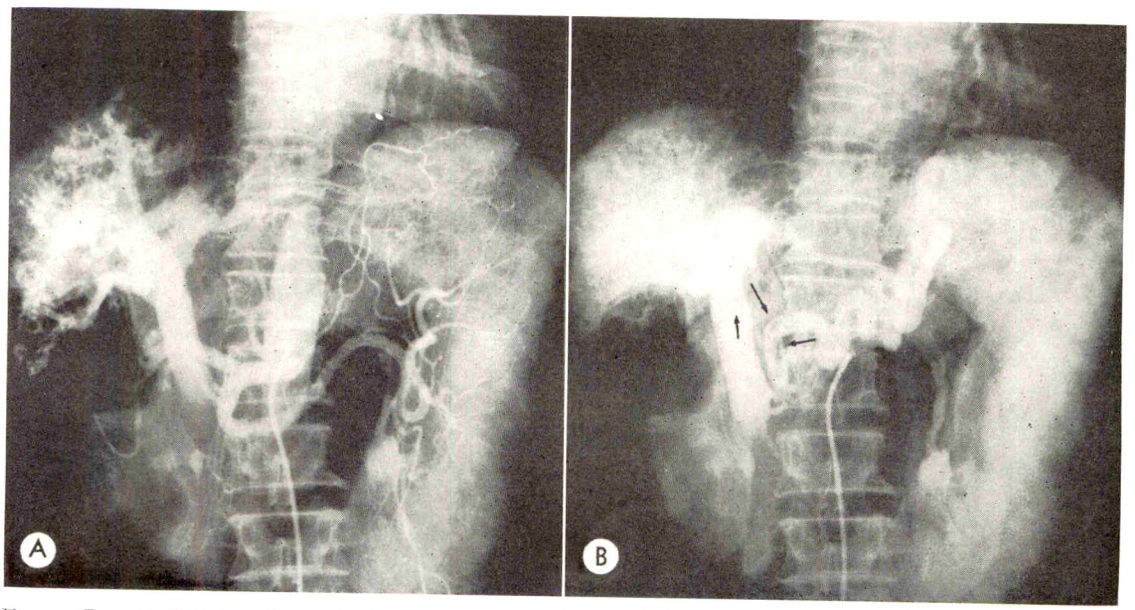


Fig. 4. Case 11. Celiac angiography. (*A*) There is marked, irregular neovascularization in the right lobe and the portal vein is visualized during the arterial phase. Splenomegaly is evident. (*B*) Contrast medium continued to flow retrograde into the tortuous coronary and collateral veins. There were a large filling defect in the portal vein and several small defects in the coronary vein (arrows), suggesting tumor thrombi. The portal vein did not flow into the superior mesenteric vein, indicating a collision of the two streams; no contrast medium entered the splenic vein from either side and splenic vein thrombosis was evident. A small collateral is flowing caudad from the beginning of the portal vein into the pelvis.

examination, he was rather poorly nourished, and several spider angiomata were seen over the anterior chest superiorly. A firm liver with an irregular surface was felt 3 fingerbreadths below the xyphoid process with slight tenderness. The lung-liver boundary was elevated to the 4th interspace in the mamillary line.

Laboratory studies disclosed: SGPT 108 u.; SGPT 82 u.; alkaline phosphatase 75.1 u.; total protein 7.6 gm., albumin 3.3 gm., γ-globulin 2.05 gm. per cent; total cholesterol 168 mg. per cent; total bilirubin 1.6 mg., direct bilirubin 1.1 mg. per cent; and ammonia 46 μg. per 100 ml. Alphafetoprotein was positive by the single radial immunodiffusion method.

The chest roentgenogram showed an irregular elevation of the right diaphragm, and on liver scanning, a large defect was seen in the right lobe. The barium swallow study disclosed esophageal and cardiac varices. Celiac angiography showed marked neovascularization in the middle of the right lobe, and the portal vein was visualized in the late arterial phase (Fig. 7*A*). In the capillary phase, the contrast medium entered the superior and inferior mesenteric and collateral veins from the dilated portal vein and the splenic vein. A round filling defect was apparent in the dilated portal vein around the porta hepatis (Fig. 7*B*).

Case v. M.Y., a 43 year old male, entered the hospital because of anorexia and general malaise. He gave a history of jaundice and anorexia for which he was hospitalized about 1½ years previously. About 4 months prior to the present admission, he again had anorexia and malaise. An upper gastrointestinal series was made by a radiologist who found a compression of the stomach, probably caused by an enlarged liver, and referred him to us for evaluation. On physical examination, he was well nourished and alert, but several vascular spiders were seen around the neck. The liver was palpable 2 fingerbreadths below the xyphoid process with a sharp tender edge. The spleen was not felt.

Laboratory examinations revealed: SGOT 93 u.; SGPT 24 u.; alkaline phosphatase 24.4 u.; ZnTT 28.0 u.; total protein 7.8 gm., albumin 3.7 gm., γ-globulin 2.2 gm. per cent; total bilirubin 0.6 mg. per cent; total cholesterol 146 mg. per 100 ml.; and BSP retention was 14.2 per cent at 45 minutes.

A barium swallow study disclosed varices in the cardia, and a liver scan showed an enlarged liver with generally poor uptake of the colloid. No apparent defect was noted. Upon splenoportography, marked collaterals and varices consisting of dilated short gastric and

coronary veins were found. The contrast medium was mostly diverted to the collaterals and a small amount of contrast medium entered from the splenic vein into the portal vein along the left superior side, seen as a thin radiopaque stream (Fig. 8*A*). When celiac angiography was carried out, contrast medium flowed into the portal vein toward the end of the arterial phase and in the capillary phase (Fig. 8*B*). Thus, the portal blood flowed in two directions, depending upon the direction of the force applied. He was subsequently found to have a hepatoma.

DISCUSSION

It has been well documented that the portal flow is reduced in varying degrees in cirrhosis, and that it becomes an outflow tract following side-to-side portacaval anastomosis in the majority of patients.[17,19,22]

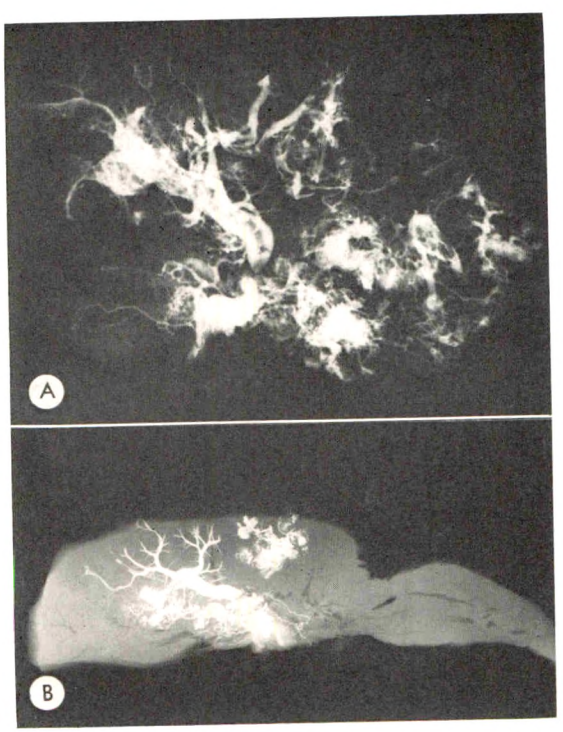


Fig. 5. Case 11. Right hepatic arteriography of the liver (removed at autopsy) with a mixture of barium sulfate and gelatin. (*A*) Roentgenogram of the whole liver. Winding arteries and tumor vessels are shown with the right portal vein, the lumen of which is filled with a tumor thrombus. (*B*) Soft tissue roentgenogram of a longitudinal slice of the liver shows the right portal vein and irregular masses of arteries below. A plaque-like deposit of contrast medium to the right of the portal vein is also a portion of the tumor.

It has also been shown in patients[6,20] as well as in animals[20] that the hepatic arterial blood increases upon reduction of the portal blood flow, although the exact mechanism for such compensatory changes is not known. Histopathologic studies in cirrhotic livers have demonstrated enlargement of the hepatic arterial beds with an increased intercommunication between hepatic arteries and portal veins.[12,13] In some patients with advanced cirrhosis, angiographic studies often disclose complete diversion of the splenic venous blood and/or of a large part of the superior mesenteric venous blood off the liver, advancing into the collateral and other mesenteric veins. All these data suggest that in cirrhosis, alterations of the portal hemodynamics result in an ever increasing reduction in the velocity of the portal flow. As Moreno *et al.*[15] demonstrated with a flow-meter during operations, one of the extreme situations is a to-and-fro motion of the portal blood, its direction being largely dependent upon respirations, posture and intraabdominal pressure.

A stagnant state of the portal flow may be suggested angiographically if a minimal forward portal flow following splenoportography together with a retrograde flow following celiac arteriography are demonstrated in the same patient, as in Case v. All of these operative and angiographic procedures are carried out with the patient supine, and do not represent a person in his daily activities. Upon rising and exertion with increased abdominal pressures, the portal flow may be reduced further, only to be replaced by the arterial blood which is less influenced by posture and will overwhelm the portal flow pushing it distally. The metabolic significance of arterial blood in patients with a retrograde flow following a shunt operation has been variously estimated, and the figures of BSP extraction vary considerably among patients as well as investigators.[3,14,17] Obviously, however, the dependence of the liver functions on arterial blood has already been established by the time the portal venous flow becomes minimal. Therefore, in such patients, a change of the flow

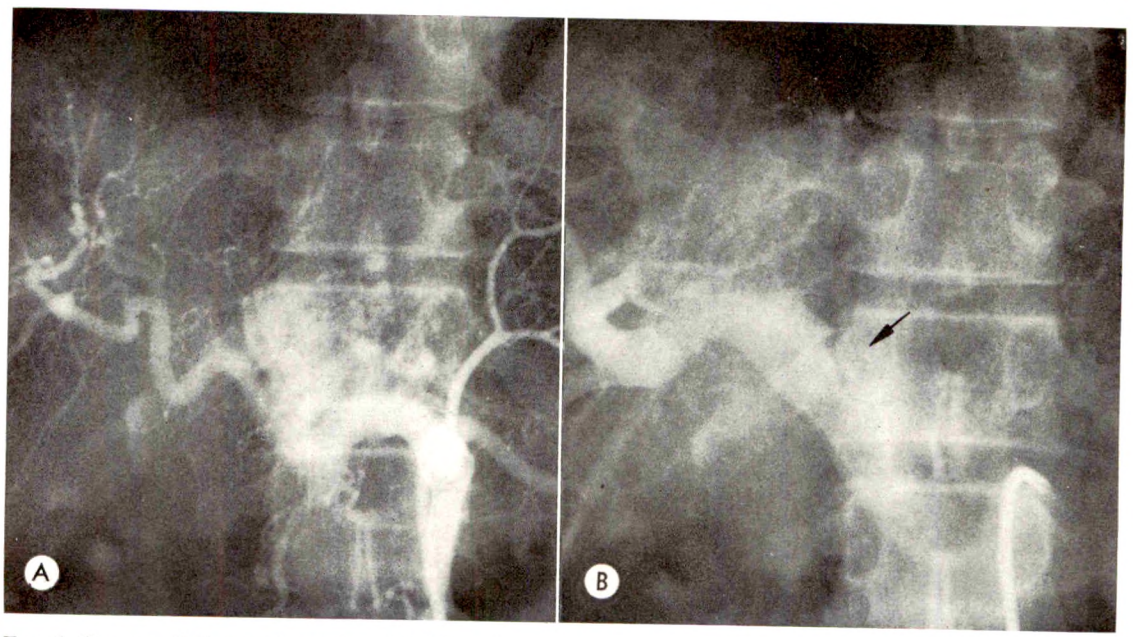


Fig. 6. Case III. Celiac angiography. (*A*) There is a marked neovasculature above the dilated hepatic artery, where the middle hepatic artery is usually seen. (*B*) A retrograde portal flow is seen in the capillary phase. There is a filling defect in the dilated portal vein where the left portal vein branches off (arrow). One of the major branches of the right portal vein has a rounded end, indicating a mass.

direction may not greatly affect the perfusion rate, because of the predominantly arterial perfusion and the small contribution of the portal blood.

The findings in Case I suggest that in an extreme situation of hemodynamic alterations, even a continuous retrograde portal flow occurs. However, in view of its low

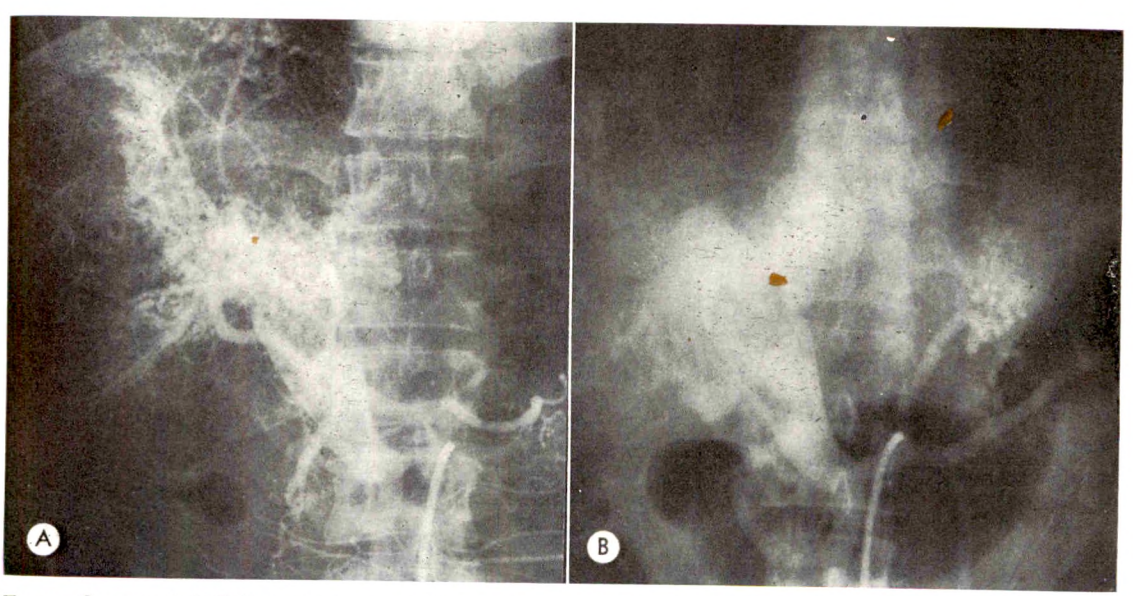


Fig. 7. Case IV. (*A*) Celiac arteriogram taken at the end of injection shows marked neovascularization in the right lobe as well as the markedly dilated portal vein. (*B*) The entire portal vein is clearly seen flowing retrograde into the coronary and further into the cardiac varices. A large filling defect is evident in the center of the portal vein where the diameter is greatest. Both the superior and inferior mesenteric veins are flowing retrograde.

incidence in our series and judging from the previous publications, spontaneous retrograde portal flow must be an extremely rare occurrence. One point that has to be made clear is that the mere fact that the contrast medium stops short of the portal vein while running from the spleen is by no means indicative of retrograde flow of the portal vein, and that a transient visualization of the portal vein following celiac arteriography may simply represent the to-and-fro motion of the portal flow, as in Case v. There were 4 cases in our series in which the splenic vein was not completely visualized following splenoportography, but the portal vein was flowing antegrade.

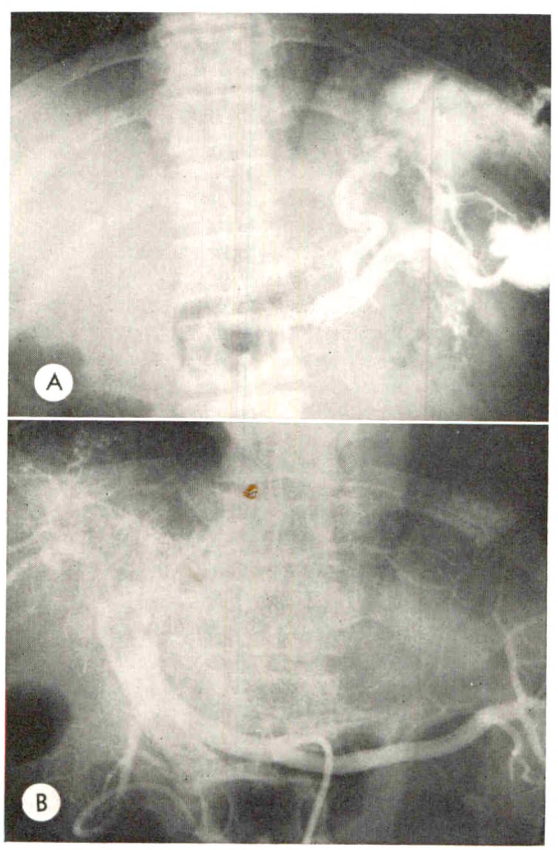


Fig. 8. Case v. (*A*) Splenoportogram. The first half of the splenic vein and the short gastric and right coronary veins were clearly visualized; the contrast medium in the proximal half of the splenic vein was diluted and diverted into the left coronary vein. The portal vein is barely visible, and a thin stream of the contrast medium is seen along the caudal and right side of the vein as far as the porta hepatis, but not beyond it. (*B*) Celiac arteriography showing a dilated common hepatic artery and winding of the branches of the right hepatic artery. The portal vein was visualized with a large filling defect near the porta.

Probably, for the same reason, retrograde portal flow occurs in primary liver cell carcinoma more often than in cirrhosis. It is well known that the arterial supply is markedly increased in hepatoma tissue,[12,24] as exemplified by neovascularizations in celiac arteriograms.[5,6,7] Arteriovenous anastomoses are often extensive[11] and portal branches are shown during the arterial phase of angiography. It is not unexpected that retrograde portal flow causes a hepatofugal tumor growth, as in Case iv, in whom not only were tumor thrombi seen in the portal and collateral coronary veins, but microscopic tumor thrombi were numerously found in the esophageal varices, providing clear evidence for retrograde portal flow. It is noteworthy that in all hepatoma cases in which a retrograde flow was seen angiographically, there were tumor thrombi in the portal vein. It is difficult to assess to what extent and at what stage the tumor growth in the hepatic vein contributes to the reversal of portal blood flow relative to the contribution of extensive arteriovenous anastomoses. It is also possible that tumor growth in the portal lumen directly contributes to the reversal of blood flow, since our autopsy studies have invariably shown a rich arterial supply to the intraluminal growth, a finding which will be published elsewhere.

SUMMARY

Angiographic studies were carried out on 49 cases of liver cirrhosis and 47 cases of primary liver cell carcinoma.

An unequivocal retrograde portal blood flow was demonstrated in 1 cirrhotic patient. The portal vein was visualized during celiac arteriography in 5 patients with hepatoma. In 1, the portal flow was to-and-from, the direction depending upon the force of the contrast medium injection. In 4 hepatoma patients in whom retrograde portal flow was seen, there were tumor

thrombi in the portal vein, and in 1 patient, in the collateral veins.

Thus, retrograde portal flow does occur in cirrhosis of the liver, but very rarely, and it occurs more often in primary liver cell carcinoma, probably due to arterial neovascularization and arteriovenous communications.

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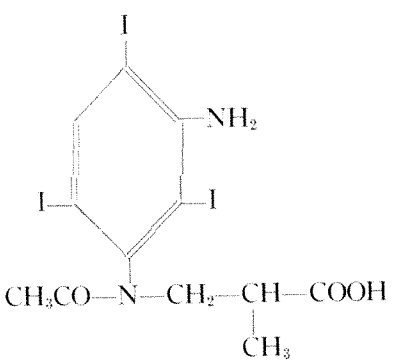
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COMPARISON OF THE OPACIFYING CHARACTERISTICS AND PHARMACOLOGIC RESPONSES OF IOCETAMIC ACID (CHOLEBRINE), A NEW ORAL CHOLECYSTOGRAPHIC AGENT, WITH SODIUM TYROPANOATE (BILOPAQUE)*

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IOCETAMIC acid (Cholebrine, Mallinckrodt) is a cholecystographic agent presently classed as an investigational drug in the United States, chemically related to sodium tyropanoate (Bilopaque, Winthrop), iopanoic acid (Telepaque, Winthrop) and sodium ipodate (Oragrafin, Squibb); first introduced for sale in the Netherlands in January 1968, and currently marketed in many European countries, Mexico, and Taiwan. It is estimated that more than a million doses have been used clinically. Its chemical designation is N-acetyl-N-(3-amino-2,4,6-triiodophenyl) amino-isobutyric acid.



Its iodine content is 62.08 per cent, and it is available as a light gray tablet (750 mg.) containing a mixture of 4 stereoisomeric active forms of the acid.

Appropriate animal and clinical investigations having been accomplished in the United States, the present study was undertaken to compare the cholecystographic characteristics and pharmacologic effects of Cholebrine with Bilopaque, a commercially available agent, chemically related to Telepaque and Oragrafin.

METHOD

A double-blind system was established, and standard dose packets (4.5 gm. iocetamic acid, 3.0 gm. sodium tyropanoate) of the drugs were given to adult patients referred for routine oral cholecystographic study. Patients were excluded from the study if they were diagnosed as:

1. Having advanced hepato-renal disease.
2. Having severe impairment of renal function.
3. Having gastrointestinal disorders which might interfere with absorption.
4. Having advanced cardiovascular disease.
5. Being pregnant.
6. Being sensitive to iodine-containing compounds.

After a light, fat-free supper, the compounds were given with water, and oral intake was restricted to water only thereafter until the roentgenographic examination had been completed the next morning. If unsatisfactory opacification of the gallbladder was noted, a second dose was given 24 hours later in the same manner.

Cholecystographic opacification was graded on a scale of zero to 4 by 2 radiol-

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TABLE I

EVALUATION OF OPACIFYING CHARACTERISTICS

Numerical Value	Grade	Criteria
4	Excellent	Equal to or exceeding the opacity of the cortex of the lumbar vertebral pedicle
3	Good	Greater than the opacity of rib but less than above
2	Fair	Equal to opacity of the 11th or 12th rib
1	Poor	Less than opacity of the 11th or 12th rib
0	None	Nonvisualization

ogists who evaluated the films independently (Table I) without knowledge of which agent had been employed. The amount of residual contrast agent visible in the bowel also was estimated.

Possible side effects were sought by standardized patient interviews and laboratory examinations. Prior to the administration of the drugs, baselines were established for the following laboratory analyses: complete urinalysis; hemoglobin; hematocrit; white blood cell count; platelet estimate; Lee and White clotting time; alkaline phosphatase; serum glutamic oxaloacetic acid (SGOT); total bilirubin; serum urea nitrogen (BUN); prothrombin time; serum creatinine; and calcium. These were repeated after drug administration, at 36 hours and 7 days or on the day of discharge from the hospital, whichever came first. Ten randomly selected patients who received Cholebrine and 10 who received Bilopaque (T_3, T_4, and free T_4 indices), performed according to the same time schedule as the basic laboratory examinations.

RESULTS

Twenty-four patients received Cholebrine and 27 received Bilopaque. Patient groups were evaluated in respect to age, sex, height, weight, history of allergy, and the condition for which the drug was administered, as well as primary and final diagnoses. The 2 groups were found to be comparable except that those who received Cholebrine were somewhat heavier (average weight 156 compared to 141 pounds for the Bilopaque group), while the group which received Bilopaque had a greater number of preexisting allergies (12, as compared with 5).

In evaluating the diagnostic quality of the roentgenograms, a diagnostic index was obtained by dividing the sum of the numerical values assigned by each of the 2 film readers by the number of patient observations. All patients who received a single dose (regardless of whether they were later given a second dose) were included. The 2 readers were in close agreement on individual cases. The results are presented in Tables II and III. Chi-squared testing revealed no statistically significant difference between Cholebrine and Bilopaque in respect to the cholecystographic quality of the roentgenograms.

Six of 7 patients whose gallbladders did not visualize after the first dose of Cholebrine had repeat studies. Four of these resulted in improved visualization. Repeat examination was done in 8 who got Bilopaque, with improved visualization in 4. The numbers are insufficient for valid statistical analysis.

Both groups had comparable amounts of residual contrast material in the bowel; in neither was there interference with gallbladder visualization.

Seven patients reported side effects after receiving Cholebrine, and 10 after taking

TABLE II

COMBINED SCORING OF OPACIFICATION CHARACTERISTICS

Score	Grade	Cholebrine	Bilopaque
4	Excellent	10 (21%)	9 (17%)
3	Good	10 (21%)	14 (26%)
2	Fair	12 (25%)	10 (18%)
1	Poor	8 (16%)	7 (13%)
0	None	8 (16%)	14 (26%)

Bilopaque. The types of side effects reported were comparable (Table IV). There was no statistical difference in incidence or severity of side effects.

The massive data accumulated as a result of the laboratory studies were meticulously analyzed and subjected to tests of statistical validity. Average values for blood chemistries were within normal limits at each observation period, except for SGOT and alkaline phosphatase among patients taking Cholebrine, and SGOT and BUN among those who had Bilopaque. Variances in average values were attributed to several patients whose values were markedly elevated due to their underlying clinical condition, and thus influenced the group average. In both groups, the values were outside normal limits before the drugs were given. The only statistically meaningful change was a decrease in serum creatinine noted at 36 hours after administration of Bilopaque, a matter of dubious clinical importance. No significant changes occurred in the thyroid studies within either group.

Among the hematologic determinations, hemoglobin levels decreased about 0.5 gm. per cent in both groups 60 hours after drug administration. This may well have been due to diagnostic and treatment manipulation rather than to drug effect. The average hematocrit dropped from 36.2 to 33.5 per cent among repeat dose Bilopaque patients, but there was no concomitant drop in hemoglobin. Another statistically evident change was an increase in the relative monocyte count more than 60 hours after repeat doses of Bilopaque (from 5 to 13 per cent). The clinical significance of this observation (as well as its validity) are conjectural.

Urine pH decreased in the repeat-dose Bilopaque patients, after the greater-than-60-hour interval (pre-drug average pH 6.8; post-drug average 5.8).

An additional statistically apparent finding was an increase in the average number of red blood cells in the urine of patients who received a single dose of Bilopaque, occurring more than 60 hours after receipt of the drug. Investigation revealed that 2 particular patients in the group were responsible for the increased average values; one had a neurogenic bladder secondary to nerve interruption during a bladder suspension procedure and developed clinical cystitis 48 hours after removal of an indwelling catheter; the other had cholecystectomy and hiatal hernia repair at 36 hours, with insertion of an indwelling catheter and subsequent hematuria. These events were regarded as not related to oral cholecystographic agent.

TABLE III

DIAGNOSTIC INDICES (SEE TEXT)

Drug	Reader	Diagnostic Index	Average Diagnostic Index
Cholebrine	1	2.1	2.1
	2	2.1	
Bilopaque	1	2.0	
	2	1.9	2.0

TABLE IV

TYPE AND NUMBER OF SIDE EFFECTS

Side Effect	Number of Patients Reporting	
	Cholebrine	Bilopaque
Nausea	1	5
Nausea and vomiting	2	3
Mild flush	1	0
Heartburn	1	0
Metallic taste	1	0
Flatulence	1	0
Diarrhea	0	2
Perioral tingling	1	0
Headache	0	1
Dizziness	0	1

DISCUSSION

Statistically valid differences were found in 7 out of the total of 106 separate tests included in this study. By chance alone, statistical analysis would be expected to disclose validity in 5.

The over-all impression gained from this study is that Cholebrine and Bilopaque are closely comparable in terms of cholecystographic characteristics, and pharmacologic effects as reflected in laboratory examination of hepatic, renal, thyroid and hematologic functions. Such changes as were assigned statistical validity are of dubious clinical significance in some categories; the drop in serum creatinine and an elevation of relative monocyte count among patients receiving Bilopaque are examples. The slight drop in hemoglobin noted in both patient groups is not necessarily due to the administration of the drugs used in this study. Prior animal and human studies[1,3] have failed to show this trend in respect to Cholebrine. Most of the patients in this study were at least moderately ill (nearly all were inpatients, hospitalized because of illness and not simply "for diagnosis"). Lacking a control group, it is therefore impossible to attribute the observed hematologic variations solely to the effects of the drugs under study; similar changes occurred in both groups and might well have occurred if neither drug had been given. Reported symptoms occurring in both patient groups must be interpreted with due regard for the difference between correlation and causal relationships. (Two clocks strike the hour at the same time—does one trigger the other?) Six of the 8 side effects reported by the patients who got Cholebrine were related to the gastrointestinal tract, while 10 of 12 of the side effects reported by Bilopaque patients were gastrointestinal. Two complaints of diarrhea were registered by the latter group, while no diarrhea occurred among Cholebrine patients. These differences lacked statistical significance.

This study confirms prior reports[1,3] that Cholebrine is a safe and effective cholecystographic agent. In these respects, it appears to be comparable to Bilopaque.

SUMMARY

A double-blind comparative study of Cholebrine, 4.5 gm., and Bilopaque, 3.0 gm., was conducted in 51 patients to determine the incidence, type, and severity of side effects, gallbladder opacifying characteristics, and pharmacologic effects as evaluated by a series of laboratory tests of hematologic, renal, thyroid and liver functions.

The observed changes in laboratory values were small, tended to occur with both drugs, were questionably related to drug administration and of dubious clinical significance.

Cholebrine and Bilopaque are comparable with respect to the properties forming the basis of this comparative study.

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SPLENOPORTOGRAPHY WITH MULTIPLE SIDEHOLE CATHETER*

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PORTAL venography performed by injection of contrast medium through the portal vein or one of its tributaries at laparotomy was first described by Blakemore and Lord,[3] in 1945. The value of splenoportography and its aid in the surgical management of portal hypertension has been emphasized by Child *et al.*,[5,6] and Rousselot *et al.*[11] The possibility of a simple transparietal approach by injection into the splenic pulp in the dog was described by Abeatici and Campi,[1] in 1951, and in the same year in man by Pereira,[10] and Boulvin *et al.*[4] Also, a technique for splenoportography with a single endhole polyethylene tube was described by Seldinger[12] in 1957, and with a multihole needle by De Sousa and Lopes,[8] in 1965.

The diagnostic potentialities and principal aims of the roentgenographic visualization of the splenic and portal veins are demonstration of hemodynamic alteration and investigation of morphology of the portal venous system.[9] For these purposes, good visualization of the portal and tributary branches is essential. The technique using a metallic needle with sideholes (De Sousa) has been useful. However, the disadvantage is that difficulty arises when the patients are not cooperative and cannot hold their breath during the procedure.

A polyethylene catheter with a single endhole for splenoportography (Seldinger) has an advantage because of softness and flexibility as compared to the metallic needle, but the disadvantage is insufficient opacification of all of the tributary branches of the splenic hilum because of the compartment-like nature of the spleen.[7] Therefore, in combining both advantages, we anticipated that the multiple sidehole configuration in a soft catheter would ensure

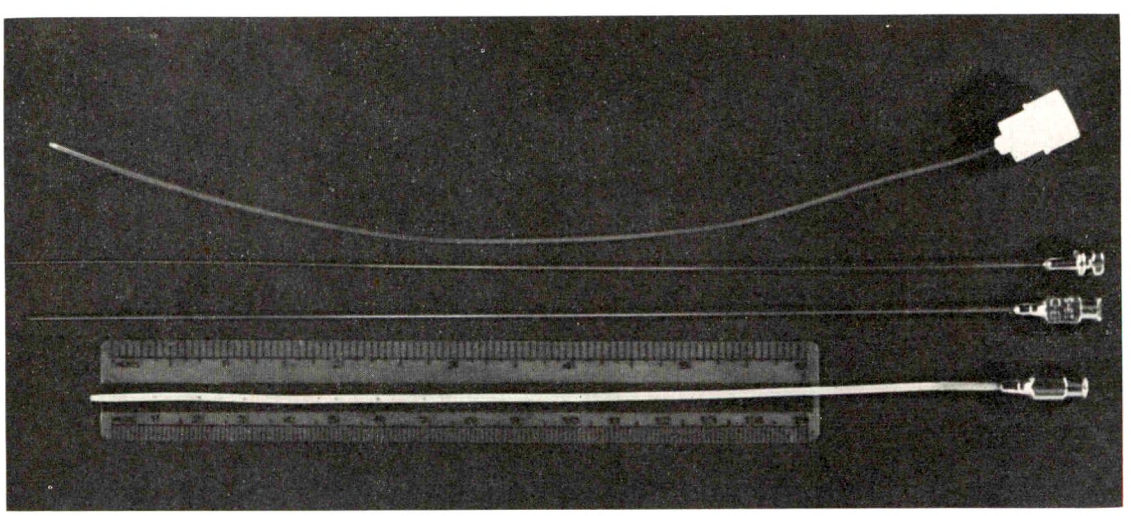


FIG. 1. B. D. Longdwel Catheter (Teflon) 18 G×8′ Thin Wall with 7 holes (1.0 cm. interval) and the metallic needle, inner metal and teflon cannula.

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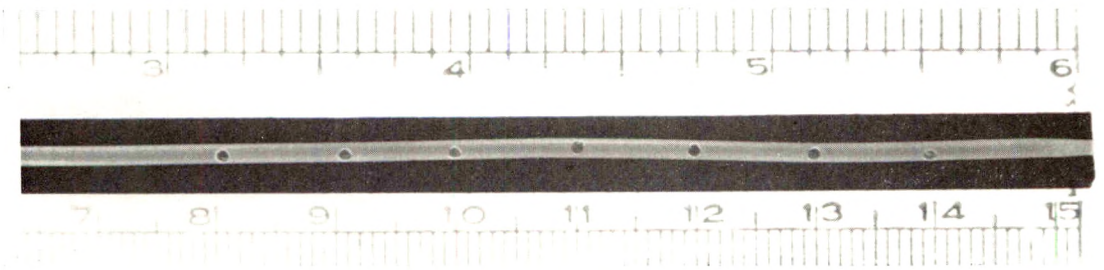


Fig. 2. Magnified view of the distal portion of the multiple sidehole catheter (7 holes with 1.0 cm. interval) on the ruler.

safety and also produce better distribution of the opaque medium to the splenic hilum and tributary branches (Fig. 1; and 2).

The purpose of this paper is to report the technique and the result of experimental splenoportography performed on a dog using this catheter and the subsequent human clinical trial.

METHOD AND TECHNIQUE

The experiment was performed on a dog because of the elongated shape and moderate size of the spleen. The dog was anesthetized by injection of intravenous sodium pentothal (10 cc.), then the abdominal cavity was opened. Splenoportography was performed with a single endhole catheter and a first injection of 60 per cent meglumine diatrizoate (20 cc.) was made (Fig. 3*A*; and 4). After the complete disappearance of the opaque medium from the portal venous system, a second puncture was made with a specifically designed multiple sidehole catheter (5 holes, 1.0 cm. interval). The metallic needle was then withdrawn and a second injection of 60 per cent meglumine diatrizoate (20 cc.) was made (Fig. 3*B*; and 5). Opacification of the splenic hilum was better than in the initial experiment.

Following the dog experiment the multiple sidehole catheter* was used in humans

* Multiple Sidehole Catheter: B. D. Longdwel Catheter (Teflon).

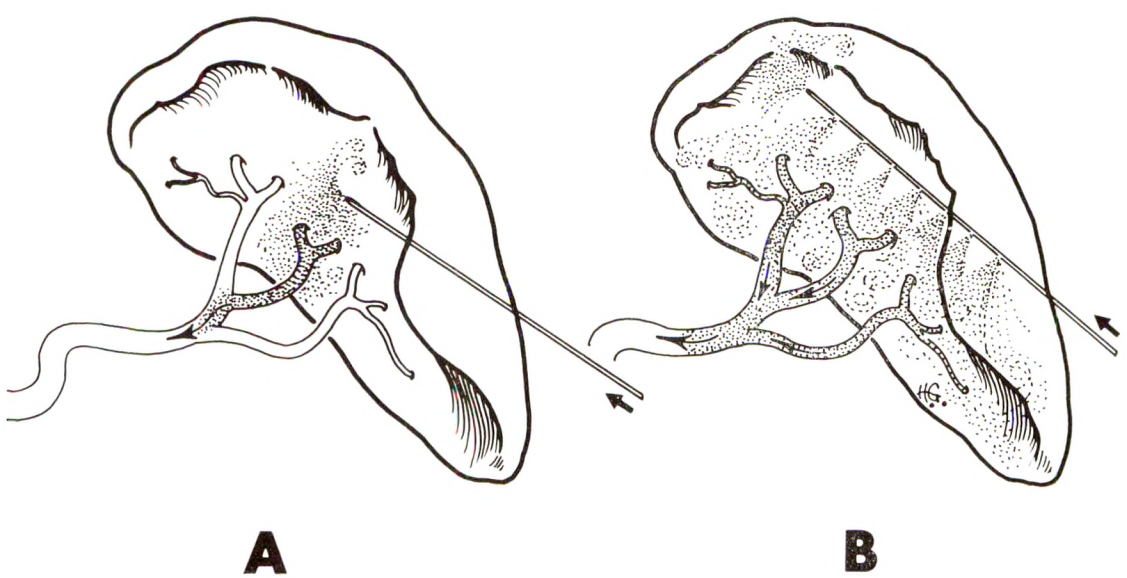


Fig. 3. Schematic illustration of splenoportography in the dog. (*A*) Conventional injection without sideholes. (*B*) Injection made with multiple sidehole catheter.

(Fig. 6; and 7). One hour before the examination, the patient received atropin (0.4 mg.), Dilaudid (0.1 mg.), and Vistaril (25 mg.), as premedication. Attempts have been made to assess the splenic size before the puncture, including physical examination (palpation) and a plain scout roentgenogram of the abdomen in order to ensure the puncture of the spleen along the long axis. Depending upon the size of the spleen in its long axis, 4 to 7 holes are made in the catheter at 1.0 cm. intervals with a hole punch. The catheter and needle are sterilized in activated dialdehyde solution or by gas sterilization before the puncture.

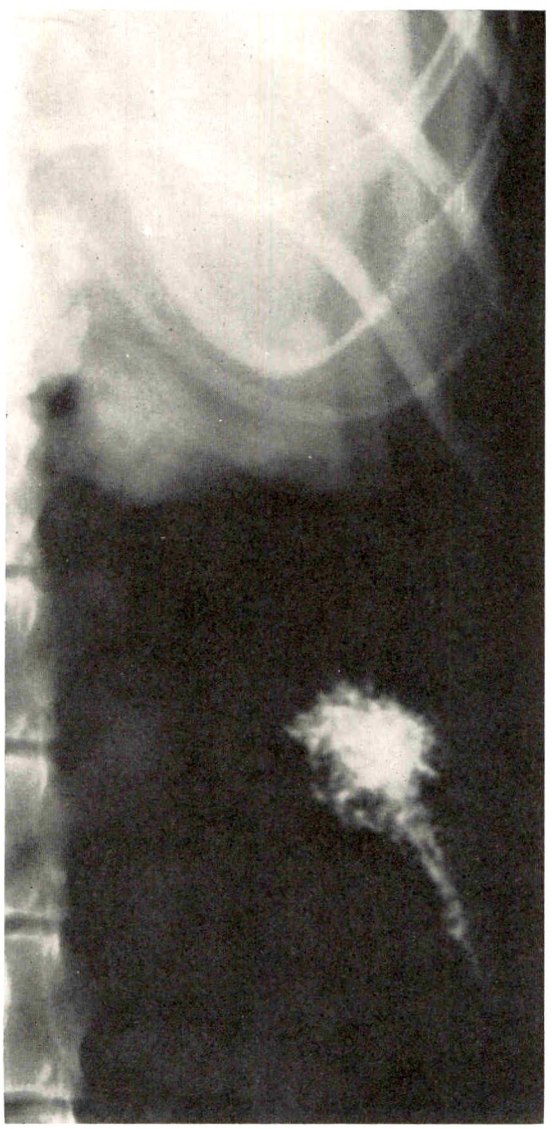


Fig. 4. Experimental splenoportography in the dog with conventional technique.

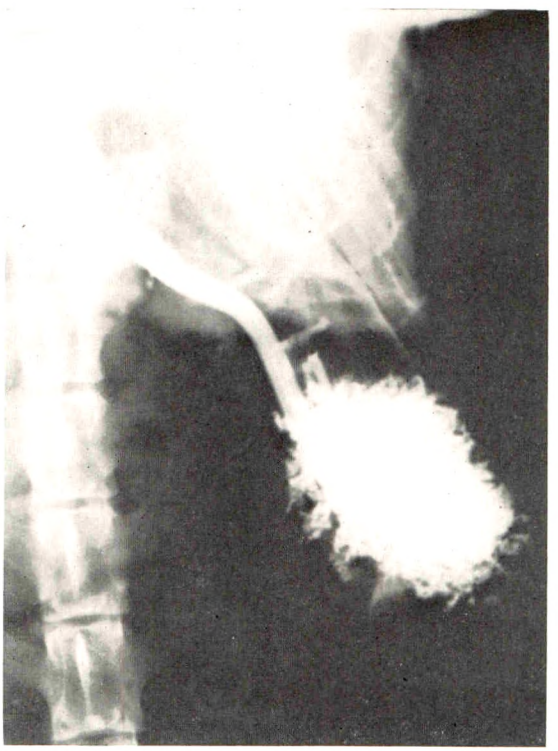


Fig. 5. Experimental splenoportography in the dog with multiple sidehole catheter.

Local anesthesia of the skin is sufficient for the puncture. The puncture is made at the tip of the spleen along the long axis,

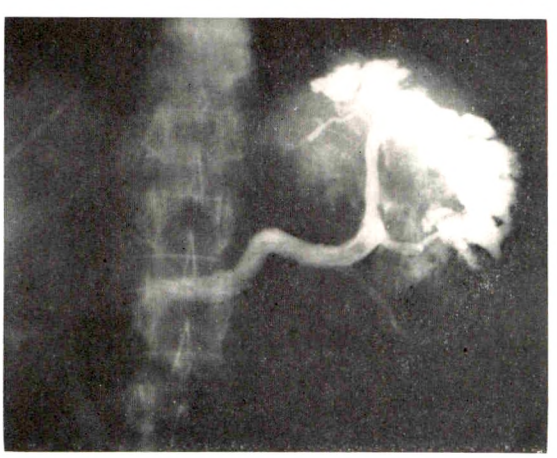


Fig. 6. Splenoportography in man, performed with multiple sidehole catheter, shows multiple splenic hilar branch opacifications.

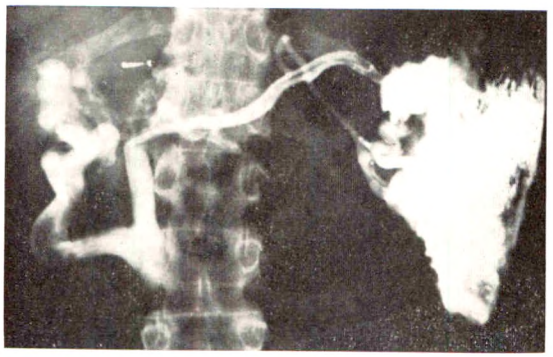


FIG. 7. Splenoportography in man, performed with multiple sidehole catheter, shows complete thrombosis of the splenic and portal vein with visualization of the collateral vessels (dorsolateral, portal venous ramus, and short gastric vein).

facing the multiple sideholes toward the hilum of the spleen. After puncture is made, the metallic needle is removed from the catheter and the catheter is connected to a 3 way stopcock. To this is connected a 10 cc. syringe filled with saline and a 50 cc. syringe filled with contrast medium. The injection of contrast medium is rapid (about 7–8 cc. per second). The contrast medium should be concentrated in order to secure satisfactory definition of vessels, a dose of 30–50 cc. of 50 per cent sodium diatrizoate or 60 per cent meglumine diatrizoate usually being sufficient. The patient is instructed not to strain during the injection. The catheter should be removed immediately after injection.[2]

This technique was used on 12 patients with improved visualization of the splenic hilum and portal vein and greater ease of study. Minute bleeding was noted at the puncture site at the time of surgery in 2 patients, but no serious bleeding or complications have been observed.

SUMMARY

An improved splenoportographic technique, with a multiple sidehole catheter, which proved to be useful after animal experimentation and human trial, is described.

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Fifty-sixth Annual Meeting: Surfer Hotel, Maui, Hawaii, April 21–25, 1974.

EDITORIAL

THE SOCIETY OF GASTRO-INTESTINAL RADIOLOGISTS



GASTRO-INTESTINAL radiology had its beginning almost immediately following the announced discovery of x rays by Konrad Röntgen. In the autumn of 1896, Walter Bradford Cannon and Albert Moser initiated gastro-intestinal radiology by studying the nature of swallowing using x rays and radiopaque contrast medium, at the suggestion of Dr. Bowditch, Professor of Physiology at Harvard Medical School.[1] They reported their first studies in 1898.[2] During the ensuing years, Cannon studied motility of the gastro-intestinal tract and established a base for studies which still has relevance today.[3]

[1] Davenport H. W. Walter B. Cannon's contribution to gastroenterology. *Gastroenterology*, 1972, *63*, 878–889.

[2] Cannon W. B., and Moser A. The movements of the food in the oesophagus. *Am. J. Physiol.*, 1898, *1*, 435–444.

[3] Cannon W. B. Early use of the roentgen ray in the alimentary canal. *J.A.M.A.*, 1914, *62*, 1–3.

Although early radiologists had to be generalists, even in those days in larger departments there were radiologists who had particular interest in and subspecialized in gastro-intestinal radiology. Guido Holzknecht and Martin Haudek in Vienna, Gösta Forssell and Åkerlund in Sweden, Rieder and Haenisch in Germany, Francis Williams, Pfahler and Carman in the United States, were among the pioneers who started the subspecialty.

The number of radiologists who devoted most of their professional energy towards the development of advances in gastro-intestinal radiology is too extensive to list. Since World War II particularly, almost every major teaching Department of Radiology in the United States has a faculty member, whose primary interest is the teaching of and clinical research in gastro-intestinal diagnostic roentgenology.

As time progressed and especially with the development of image-intensified fluoroscopy and the advent of television, other physicians and gastro-enterologists retreated from the active performance of fluoroscopy and began to depend increasingly on consultation with these radiologists. Furthermore, as in cardiovascular medicine, pediatrics and the neurologic specialties, interdepartmental teams developed in gastro-enterology with internists, surgeons and radiologists, having as much in common with each other as with members of other subspecialties in their own specialty. This resulted in joint research projects as well as in improved clinical work-ups.

Such joint efforts have advanced gastro-intestinal radiology, opening new areas of

diagnostic procedures; *e.g.*, physiologic studies using esophageal cineradiography, newer approaches to the roentgen study of the biliary tree and small bowel, and the use of hormones, like cholecystokinin and glucagon, and drugs, like neostigmine and probanthine. The entire field of gastro-intestinal radiology is in a state of excitation with progress made in too many areas to be listed here. Since so many general radiologists are active in performing gastro-intestinal examinations, these advances are finding rapid clinical application and common usage.

The National Institutes of Health research training grants in diagnostic radiology have given further opportunities to young academic radiologists interested in pursuing research in this field. Multiple areas are currently being attacked to obtain knowledge about the physiology of the gastro-intestinal tract by using radiologic methods, as well as to improve radiologic methods themselves in the study of the gastro-intestinal tract.

At present, work is being performed on motility of the entire gastro-intestinal tract, on digestion, or absorption and excretion of biliary contrast media, and on the use of pharmacologic agents in improving contrast studies both of the gut and the blood vessels supplying it. In addition to experimental laboratory research, clinical investigations flourish throughout this country; many entities previously felt to be fully understood are being re-evaluated with interesting results.

For several years now, gastro-intestinal radiologists have been getting together informally at national meetings and instructional courses to exchange their research and clinical experiences. It is not surprising therefore that the idea originated among several of them to form a society which would be dedicated to further advances in gastro-intestinal radiology.

The Society of Gastro-Intestinal Radiologists was formed after most of the practicing subspecialists in gastro-intestinal radiology answered an inquiry and expressed their enthusiastic support for such an organization. The founding committee prepared By-Laws and circulated these with invitations for papers. The criteria for eligibility to membership are past and present contributions to progress in gastro-intestinal radiology, and commitment to at least half-time activity in this subspecialty.

It is the intent of the Society to keep the membership small to allow for personal contact, discussion, and to keep the meeting atmosphere informal and intimate. The meetings are to be a forum for discussion of proposed areas of research, cooperative studies and work-in-progress, as well as an occasion for presentation of clinical and laboratory investigations which have been completed.

Initially, membership is limited to 100 members, but it is expected that exceptions will be made, in order not to exclude active contributors to advances in the field. To remain on the active list, members must attend at least 1 out of 4 consecutive Society meetings. In addition to active and emeritus members, gastro-intestinal radiologists from foreign countries who are unable to attend meetings regularly may be elected corresponding members. Honorary members are elected only from among non-radiologists and radiologists from abroad.

The first meeting of the Society combined an organizational business session with a scientific program; it was held in Chicago on November 29, 1971. The second meeting was chaired by the Society's first President, Richard H. Marshak, and also was held in Chicago on November 27, 1972. In addition to numerous scientific papers, the first Walter Bradford Cannon Lecture was given by Professor Horace W. Davenport, Chairman of the Department of Physiology at the University of Michigan. His Lecture is the lead article in this issue of the JOURNAL.

The Society of Gastro-intestinal Radiologists has established the Walter Bradford Cannon Medal to be awarded for excep-

tionally significant contributions to gastrointestinal radiology. The first recipient of the medal was Dr. Marshak.

With the continued enthusiastic support of its membership and the dedicated leadership of its officers, it is hoped that the Society of Gastro-Intestinal Radiologists will flourish and its members will continue to advance the field for the benefit of mankind.

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JAMES PICKER FOUNDATION FELLOWSHIPS AND GRANTS

The National Research Council was organized by the National Academy of Sciences in 1956 in order to provide for a broader participation by American scientists and engineers in the work of the Academy.

On July 17, 1973, the National Research Council (NRC), on behalf of the James Picker Foundation, announced the awarding of 27 fellowships and grants to medical scientists for support of research and training in radiology and nuclear medicine.

The awards program, valued this year at approximately $400,000, is supported by the James Picker Foundation and administered by the Committee on Radiology of the NRC's Division of Medical Sciences. Twelve of the awards are renewals for work already in progress, while 15 are being given for the first time.

Those selected to participate in the program for the 1973–1974 academic year are:

ADVANCED FELLOWSHIPS IN ACADEMIC RADIOLOGY

Dr. Jason C. Birnholz has been awarded his first-year Advanced Fellowship to investigate quantitative ultrasound with specific identification of intraabdominal neoplasms by sonic imaging techniques. This work will be conducted under the sponsorship of Dr. Juan M. Taveras at the Massachusetts General Hospital and Harvard Medical School.

Dr. Robert L. Carangi, as a second-year Advanced Fellow, under the sponsorship of Dr. George Jacobson will continue his research at the University of Southern California involving an immunological problem associated with depletion of the lymphocytes by thoracic duct cannulation and immunosuppression problems.

Dr. Conrade C. Jaffe, during his first year as an Advanced Fellow, will investigate radiographic imaging of dynamic anatomic structures under the joint sponsorship of Dr. Wiliam B. Seaman of Presbyterian Hospital, Columbia University, New York, and Dr. Murray Eden of Massachusetts Institute of Technology.

Dr. Charles A. Mistretta, as a second year Advanced Fellow, will continue his investigation of new imaging techniques in diagnostic radiology, under the sponsorship of Dr. John Juhl, at the University of Wisconsin Medical School.

RESEARCH FELLOWSHIPS

Dr. Pejavar S. Rao received his B.Sc. and M.Sc. degrees from the University of Madras, India, and his Ph.D. in high-energy physics from Syracuse University in 1968. He will spend his first year as a Research Fellow investigating the detection and delineation of soft tissue tumors under the direction of Dr. Earle C. Gregg at Case Western Reserve University.

SCHOLAR AWARDS

Dr. Robert E. Anderson received his M.D. and M.S. in radiology from the University of Minnesota. As a first year Scholar, he will investigate applications of ultrasonics, particularly holography, to diagnostic radiology at the University of Minnesota.

Dr. Warren A. Hinchcliffe received his M.D. degree from New York Medical College in 1966, and he came to the University of California, San Francisco, in 1968. Under the direction of Dr. Alexander R. Margulis, Dr. Hinchcliffe will investigate radiologic and physiologic studies of the pulmonary veins in intact animals.

Dr. Melvyn Korobkin received his M.D. degree from Yale Medical School in 1967. His research, "Urographic and Angiographic Studies of Renal Ischemia," will be conducted at the University of California Medical Center, San Francisco.

Dr. James A. Nelson received his A.B. and M.D. degrees from Harvard Medical School. Since 1965 he has been at the University of California School of Medicine, San Francisco,where he will now investigate the problems associated with clinical use of oral and intravenous contrast media for visualization of gallbladder and biliary ducts.

Dr. Bernard E. Oppenheim is in his second year as a Picker Foundation Scholar. He received his B.S. in mathematics with highest honors from the University of Arizona in 1959, and his M.D. from the University of Chicago in 1963. His work in the past year has involved

the theoretical aspects of motion correction with computers, and he has developed equations that give the relationships of the various factors involved.

Dr. Robert W. Parkey, a third year Picker Foundation Scholar, is a graduate of the University of Texas at Austin and Dallas. During his first year as a Scholar, he and his associates developed reliable computer programs capable of deriving regional myocardial blood flow values from the scintillation camera image of a bolus of xenon 133 injected into the coronary artery of the dog. His second year was devoted to attempting to establish the identity of the first 2 components of his multi-compartment curve.

RESEARCH GRANTS

Dr. Robert N. Berk has been awarded a second-year Research Grant to continue his evaluation of factors which may influence radiological visualization of the biliary tree. He has conducted research in related areas at the Salk Surge Facility's Department of Radiology, investigating cholecystography and biliary physiology.

Dr. Murray K. Delinka received his B.S. and M.D. degrees from the University of Michigan. He has received a second-year Research Grant from the Foundation to continue his research in intravenous cholangiography-physiologic studies.

Dr. Michael A. Davis earned his B.S. and M.S. degrees from the Worcester Polytechnic Institute and his S.M. and S.D. degrees from the Harvard School of Public Health. A principal associate in radiology at Harvard Medical School, his goal is to establish a simple, rapid, and reproducible procedure for the labeling of white blood cells and their evaluation as diagnostic agents in a variety of pathologic conditions.

Dr. Peter A. Dietrich received his M.D. in 1965 from Western Reserve Medical School, Cleveland. During his second-year Research Grant, Dr. Dietrich will continue his investigation of problem-oriented ordering and reporting of diagnostic radiology.

Dr. Norman A. Frigerio received his Ph.D. from Yale University in 1957. He has been awarded a first-year Research Grant to investigate the localization of radiolabeled compounds in spontaneous and induced malignancies. As a previous Picker Research Grantee, he proposes to chemically modify the localizing compounds and devise purification methods and administration schedules, in order to obtain optimum localization in, and optimum demonstration of, the tumors.

Dr. E. Stephen Garnett, as a second-year Research Grantee from the Foundation, will continue to work at McMaster University, Hamilton, Ontario, in an effort to develop F^{18} 3,4-dihydroxy-5-fluoro phenylalanine (F^{18}-5-Fluoro-DOPA) as a radiopharmaceutical with a specific activity high enough to make it useful as a brain scanning agent in man to delineate vascular and neoplastic lesions with equal facility.

Dr. Michael T. Gyepes received his M.D. degree in 1959 from the University of California School of Medicine, San Francisco. His research will involve evaluation of fetal circulation generally and in specific organ systems through angiographic techniques. His project is expected to permit *in vivo* imaging of the fetal circulation with advanced recording techniques.

Dr. George S. Harell, born in Vienna, Austria, received his M.D. degree from Columbia University College of Physicians and Surgeons in 1963. With a second-year Research Grant, he will continue his investigation of esophageal blood flow.

Dr. John C. McAfee received his M.D. degree from the University of Toronto in 1948. The goal of his Research Grant project is to develop a new radiopharmaceutical labeled with Tc^{99m} which can be safely administered to humans intravenously and which will concentrate in the normal myocardium sufficiently well to demonstrate areas of infarction as filling defects.

Dr. Andrew K. Poznanski, a native of Czestochowa, Poland, has received a second-year Research Grant to continue his investigation of pattern profiles in the diagnosis of congenital disorders. He received his B.Sc. and M.D. degrees from McGill University in Montreal. His project is to systematically plot pattern profiles in a number of additional congenital malformation syndromes to determine which are useful for diagnosis and to develop methods of diagnosis of these conditions which will be simple to use.

Dr. Josef Rösch, after receiving his M.D. in 1950 from Charles University in Prague, Czechoslovakia, remained there to become a radiology specialist, teacher, and lecturer until 1966. In 1967, he became a research associate at the University of Oregon Medical School.

With a second-year Research Grant, he will continue his study of radiologic treatment of acute gastrointestinal bleeding.

Dr. Salvador Treves received his M.D. degree in 1966 at the University of Buenos Aires, Argentina. With a first-year Research Grant, he plans to investigate radionuclide assessment of regional lung function in babies and small children using a radioactive gas (xenon 133), a gamma scintillation camera, and an on-line dedicated digital computer. These are expected to optimize data acquisition and analysis, so that this technique can be applied to patients.

Dr. Manuel Viamonte, Jr., received his M.D. in 1955 at Havana University School of Medicine in Havana, Cuba. He received his M.Sc. in radiology in 1960 from the University of Pennsylvania. His second-year Research Grant will enable him to continue his study of the development of cationic cholecystographic agents at the University of Miami School of Medicine.

Dr. Joseph P. Whalen received his M.D. in 1959 from the State University of New York at Syracuse. With a second-year Research Grant, he will continue his investigation of bone remodeling during development with particular interest in normal and abnormal influence of calcitonin and parathormone.

Dr. Jack Wittenberg received his M.S. in microbiology in 1958 and his M.D. in 1962 at the State University of New York, Upstate Medical Center, Syracuse. With a first-year Research Grant, he will study infusion cholangiography determination of dose and duration.

Dr. Gerald L. Wolf has been awarded a first-year Research Grant to investigate a microelectrode study of contrast agent induced arrhythmias. He received his B.S., M.S., and Ph.D. degrees from the University of Nebraska and his M.D. from Harvard Medical School in 1968. The goal of his project is to elucidate, at cellular level, the mechanisms of cardiac arrhythmias produced by contrast agents (C.A.).

The James Picker Foundation, headquartered in White Plains, New York, was established in 1947 by James Picker and his son Harvey with the broad purpose of fostering research in the field of radiology. Grants and fellowships in radiological research and nuclear medicine have been awarded annually since 1950 under the administration of the National Research Council's Committee on Radiology. James Picker, who died in 1963, was a pioneer in the manufacture of X-ray equipment and had had a life-long devotion to the advancement of diagnostic X-ray technology.

NEWS ITEMS

CONFERENCE ON RADIOLOGY IN OTOLARYNGOLOGY AND OPHTHALMOLOGY

The Departments of Radiology, Otolaryngology and Ophthalmology of the University of Illinois are presenting the Seventh Annual Conference on Radiology in Otolaryngology and Ophthalmology at the Illinois Eye and Ear Infirmary, University of Illinois at the Medical Center, Friday and Saturday, November 23 and 24, 1973.

The purposes of the program are to make radiologists more aware of the applications of radiology in otolaryngology and ophthalmology; also to present recent advances in otoradiology. One-and-one-half days will be spent on radiology and otolaryngology. Saturday afternoon will be devoted to radiology and ophthalmology.

The Faculty Coordinator is Galdino E. Valvassori, M.D.

For further information write to: Herman H. Slayman, Ed.D., Associate Professor, Continuing Education, Division of University Extension, University of Illinois at the Medical Center, 715 S. Wood Street, Chicago, Illinois 60612.

NATIONAL CONFERENCE ON VIROLOGY AND IMMUNOLOGY IN HUMAN CANCER

American Cancer Society-National Cancer Institute

The National Conference on Virology and Immunology in Human Cancer will be held at the Waldorf-Astoria Hotel in New York City, November 29–December 1, 1973.

This Conference, which is sponsored by the American Cancer Society and the National Cancer Institute, will present to the medical and related professions the current developments in research and clinical investigation in virology and immunology and the assessment and implications of this work in the prevention and treatment of human cancer.

The Sessions are open to all members of the medical and related professions. Advance registration is requested.

Additional programs, advance registration cards and hotel reservation cards may be obtained from the Divisions of the American Cancer Society or from: S. L. Arje, M.D., Coordinator, National Conference on Virology and Immunology in Human Cancer, American Cancer Society, Inc., 219 East 42nd Street, New York, New York 10017.

SPECIAL PROCEDURES IN DIAGNOSTIC RADIOLOGY

The Department of Radiology at Mount Sinai Medical Center, in collaboration with the Department of Radiology at Jackson Memorial Hospital, University of Miami School of Medicine will present the Fourth Annual Seminar on "Special Procedures In Diagnostic Radiology" at the Playboy Plaza Hotel, Miami Beach, Florida, January 8–11, 1974.

Twenty-six instructors, experts in radiologic special procedures will conduct informal refresher courses each morning (a total of 30), and will meet with small groups in the evenings for round table discussions (a total of 20).

There will be a one-hour general session each morning and each afternoon during which basic concepts, controversial methods and recent advances in special procedures will be discussed.

During this course, quizzes will be given for self-evaluation and there will be audience participation with ample opportunities for exchange of information.

For further information please write to: Miss Peggy Litka, Department of Radiology, Mount Sinai Medical Center, Miami Beach, Florida 33140.

A Post-Convention Seminar will be offered again this year immediately following the Special Procedures Seminar (January

12–16, 1974). For additional information please write to: Post-Convention Seminar in Radiology, 1200 N.W. 10th Avenue, Miami, Florida 33136.

OPTICS IN DIAGNOSTIC MEDICINE

The Department of Radiology, College of Medicine, University of Arizona, Tucson, Arizona, sponsors a Tutorial Seminar on Optics in Diagnostic Medicine, to be held in Tucson, Arizona, January 9–11, 1974.

Radiologists are vitally interested in the applications of optical instruments and techniques to improve diagnostic capabilities and patient care, while the optical scientists are primarily concerned with the engineering of new devices and techniques. The fields of research of these two groups are entirely different, and the language of each is largely foreign to the other.

This tutorial seminar is organized in an attempt to overcome some of these difficulties. Experts will discuss such topics as imaging in diagnostic radiology and nuclear medicine, thermography, medical holography, ultrasound, image recording and storage, image processing and information extraction, fiber optics, and light microscopy.

The interdisciplinary relationship between the College of Medicine and the Optical Sciences Center, and their cooperative relationship with the Optical Society of America, provide a unique opportunity for this tutorial seminar, while the sunny climate and warm hospitality of Tucson provide an ideal atmosphere for it.

Additional information on arrangements is available from: Joel E. Gray, Arrangements Chairman, Optics in Diagnostic Medicine, Department of Radiology, College of Medicine, University of Arizona, Tucson, Arizona 85724.

MIDWINTER RADIOLOGICAL CONFERENCE OF THE LOS ANGELES RADIOLOGICAL SOCIETY

The Twenty-sixth Annual Los Angeles Midwinter Radiological Conference for 1974 will again be held at the Century Plaza Hotel, Los Angeles, California 90067, on February 1st, 2nd and 3rd.

Outstanding speakers will deal with Diagnostic and Therapeutic Radiology as well as Nuclear Medicine and Ultrasound.

Small workshops will be featured, covering all modalities and subspecialties of Radiology. A radiologic technical exhibit will be open for viewing on all 3 days.

For further information please write to: James F. Mack, M.D., Publicity Chairman, 4500 Marloma Drive, Rolling Hills Estates, California 90274.

INTRODUCTION TO NEURORADIOLOGY

This course, which will be held February 1 and 2, 1974, will provide a review of the field of Neuroradiology by experienced teachers in the field. Although intended primarily for radiologists, neurologists and neurosurgeons, the presentations will be of value for any clinician dealing with neurologic disease.

More detailed information is available through Irwin H. Brown, M.D., Director of the Office of Continuing Medical Education for Physicians, University of Oklahoma College of Medicine, P.O. Box 26901, Oklahoma City, Oklahoma 73190.

GASTROINTESTINAL RADIOLOGY POSTGRADUATE COURSE

The tenth Annual Postgraduate Course in Radiology, sponsored by the Department of Radiology, Medical College of Virginia-Virginia Commonwealth University, will be held at the Williamsburg Conference Center in Williamsburg, Virginia, March 10–14, 1974.

This course is designed for the general radiologists and the interested allied physicians in other medical specialties. A distinguished Guest Faculty and members of the Faculty of the Medical College of Virginia will participate.

For further information, please write to: Gary G. Ghahremani, M.D., Associate Professor and Chief of Gastrointestinal Radiology, MCV Station Box 728, Richmond, Virginia 23298.

SEMINAR ON RADIOLOGY IN MEDICAL AND SURGICAL EMERGENCIES

The Twelfth Annual Seminar "Radiology in Medical and Surgical Emergencies," will be held at the Playboy Plaza Hotel in Miami Beach, Florida, March 23–30, 1974.

This Seminar is sponsored by the Department of Radiology, University of Miami School of Medicine (at Jackson Memorial Hospital).

For further information, please contact: Manuel Viamonte Jr., M.D., Department of Radiology, University of Miami School of Medicine, Post Office Box, Biscayne Annex, Miami, Florida 33152.

AMERICAN CANCER SOCIETY'S NATIONAL CONFERENCE ON CHILDHOOD CANCER

The purpose of this conference, to be held at the Fairmont Hotel, Dallas, Texas, May 16–18, 1974, is to alert the medical community to the progress being made in the diagnosis and treatment of childhood cancer and to emphasize the need for early diagnosis and the importance of prompt treatment by a team of medical specialists and allied health personnel capable of rendering optimal care.

Sessions are open to all members of the medical and related health professions. Preregistration is requested. There is no registration fee.

For further information please write to: American Cancer Society's National Conference on Childhood Cancer, 219 East 42nd Street, New York, New York 10017.

BRITISH NUCLEAR MEDICINE SOCIETY

The Second Annual Meeting of the British Nuclear Medicine Society will be held in the Middlesex Hospital Medical School, London, March 28 and 29, 1974.

The program will include review and original papers, as well as specialized "teach-in" sessions for technicians.

Please address enquiries to: The Honorary Secretary, Dr. E. R. Davies, Radiodiagnostic Department, Bristol Royal Infirmary, Marlborough Street, Bristol BS2 8HW.

INTERNATIONAL ACADEMY OF PROCTOLOGY

The Twenty-sixth Annual Congress and Teaching Seminar of the International Academy of Proctology will be held in beautiful Montreux and Zürich, Switzerland, May 3–17, 1974.

Waldo W. Stiles, M.D., of Quito, Ecuador, President of the Academy, will preside.

Scientific Sessions are planned in both cities to be visited, under the direction of Alvin D. Yasuna, M.D., of New York, Program Chairman.

All physicians are invited to attend the Scientific Sessions at which time leading doctors from many parts of the world are scheduled to present very worthwhile scientific papers.

Further information may be obtained by writing directly to the Executive Offices, 147-41 Sanford Avenue, Flushing, New York, 11355, attention of Alfred J. Cantor, M.D., Executive Secretary.

VII CONGRESSO DA ASSOCIAÇÃO MÉDICA BRASILEIRA

The Brazilian Medical Assocation will hold its 7th Congress together with the 5th Congress of the Medical and Surgical Society of Rio de Janeiro, organized by the Medical and Surgical Society of Rio de Janeiro, November 4–9, 1973 at the up-to-date Convention Center of the National Hotel (São Conrado shore) in Rio.

The Brazilian Society of Neuroradiology is among the 28 specialized Societies which have confirmed their participation. An outstanding program has been arranged.

The Committee is also preparing an especially attractive social program.

For further information please write to: Dr. A. Tomaz Rezende, Secretary, Prof. Associado de Radiodiagnostico da PUC, Neuroradiology 73, Caixa Postal 9031 ZC-02, 20 000 Rio de Janeiro, GB, Brazil.

BOOK REVIEWS

THE LYMPHATICS: DISEASES, LYMPHOGRAPHY AND SURGERY. By John B. Kinmonth, M.B., M.S. (Lond.), F.R.C.S. (Eng.), Professor of Surgery, University of London; Director, Surgical Unit, St. Thomas's Hospital, London; Consultant in Vascular Surgery to the Royal Air Force. Cloth. Pp. 420, with many illustrations. Price, $43.50. Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, 1972.

Dr. Kinmonth is the originator of the modern technique of lymphography and this work is the culmination of his years of experience with this diagnostic method. It contains detailed descriptions of the applications of lymphography in the various portions of the human anatomy, diseases of the lymphatic system, and the therapy of various disease states.

The descriptions of the technique of performance of the examination may be a bit scanty for the neophyte who might desire a more step by step presentation. The section on lymphedema is superb and represents the most complete discussion of this subject available in contrast to the section on lymph node disease, which occupies a considerably more limited portion of the book.

The illustrations are excellent, and the paper of good quality. The bibliography is ample, but heavily weighted toward the European literature.

This contribution is an outstanding effort by a pioneer in the field and should be in the library of all physicians who are involved in the diagnosis and treatment of diseases of the lymphatic system.

THEODORE E. KEATS, M.D.

ATLAS OF ORBITAL RADIOGRAPHY: CONVENTIONAL RADIOGRAPHY. By Judah Zizmor, M.D., Director, Department of Radiology, Manhattan Eye, Ear, and Throat Hospital, New York, N. Y.; Attending Radiologist, The New York Hospital; Clinical Associate Professor of Radiology, Cornell University Medical School, New York, N. Y. ORBITAL CONTRAST RADIOGRAPHY AND ANGIOGRAPHY. By Guido Lombardi, M.D., Chief Radiologist, Istituto Neurologico; Consultant Radiologist, Istituto Oftalmico, Milano, Italy. Cloth. Pp. 180, with many illustrations. Price, $25.00. Aesculapius Publishing Company, 903 South 21st Street, Birmingham, Ala. 35205, 1973.

This book is divided into 2 Sections.

The first Section by Dr. Zizmor reviews the abnormal orbital roentgenographic findings seen in patients with disease of the orbit. There is a separate chapter for each roentgenographic finding such as fossa formation, dehiscence of bone, osteolysis of bone, etc. Each of these chapters begins with a differential diagnosis of conditions which might produce the abnormal roentgenographic finding. The differential diagnosis is then followed by cases illustrating each entity in the differential. In addition to the chapters on differential diagnosis of abnormal orbital roentgenographic findings, there is a chapter explaining the technique of foreign body localization.

The second Section of the book, by Dr. Guido Lombardi, is a review of the use of contrast media in orbital roentgenography. Positive and negative contrast orbitography are discussed, as well as orbital venography and arteriography.

Since the book is organized on the basis of roentgenographic findings, it makes it easy for the radiologist presented with an abnormal finding in roentgenograms of the orbit to turn to the pertinent chapter to help him with the differential diagnosis of the case in hand.

One especially useful feature of the book is the extensive bibliography at the end of each chapter. Thus, if one wants to know more about a subject, the references are there available.

GUY D. POTTER, M.D.

BOOKS RECEIVED

RADIOLOGICAL ATLAS OF BONE TUMOURS. Volume 2. By The Netherlands Committee on Bone Tumours. Cloth. Pp. 600, with many illustrations. Price, $77.00. Williams & Wilkins Company, Baltimore, Md. 21202 exclusive U. S. agents, 1973.

THE NATURE OF CANCER. By Manu L. Kothari, M.B., M.S., M.Sc., and Lopa A. Mehta, M.B., M.S., Department of Anatomy, Seth G. S. Medical College, Bombay, India. Cloth. Pp. 902, with

132 illustrations. Price, $30.00. Kothari Medical Publications, Acharya Donde Marg, Bombay 400 012, India, 1973.

The Radiologic Clinics of North America. August, 1973, Volume XI, Number 2. Symposium on Cardiopulmonary Inflammatory Disease. David G. Bragg, M.D., Guest Editor. Pp. 186, with many illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

Modern Thin-Section Tomography. Edited by Arnold Berrett, M.D., Associate Clinical Professor of Radiology, New York Medical College, New York, N. Y.; Sam Brünner, Radiologist-in-Chief, Gentofte Hospital, Copenhagen, Denmark; and Galdino E. Valvassori, Professor of Radiology, University of Illinois College of Medicine, Chicago, Ill.; with a foreword by Sölve Welin, M.D., Professor of Diagnostic Radiology, University Hospital of Lund, Malmö, Sweden. Cloth. Pp. 337, with some figures. Price, $26.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

The Roentgenographic Diagnosis of Diseases of the Thoracic Aorta. By Steven H. Cornell, B.S., M.D., F.A.C.R., Professor of Radiology, The University of Iowa College of Medicine, Iowa City, Iowa. Cloth. Pp. 284, with many illustrations. Price, $15.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

Clinical Pediatric Oncology. Edited by Wataru W. Sutow, M.D., Pediatrician and Professor of Pediatrics, The University of Texas M. D. Anderson Hospital and Tumor Institute at Houston, Houston, Texas; Teresa J. Vietti, M.D., Professor of Pediatrics, Mallinckrodt Department of Pediatrics, Washington University School of Medicine; Associate Pediatrician, Barnes and Associated Hospitals; Director, Division of Hematology and Oncology, St. Louis Children's Hospital, St. Louis, Mo.; and Donald J. Fernbach, M.D., Professor of Pediatrics, Baylor College of Medicine; Chief, Hematology and Oncology Service at Texas Children's Hospital; Director, Research Hematology Laboratory, Texas Children's Hospital, Houston, Texas. Cloth. Pp. 602, with 216 illustrations. Price, $35.50. C. V. Mosby Company, Publishers, 11830 Westline Industrial Drive, St. Louis, Mo. 63141, 1973.

A Manual of Radiographic Positioning. By George B. Greenfield, M.D., Professor and Chairman, Department of Radiology, University of Health Sciences/The Chicago Medical School, Mount Sinai Hospital Medical Center, Chicago, Ill.; Attending Radiologist, Cook County Hospital, and the Hektoen Institute for Medical Research at the Cook County Hospital, Chicago, Ill.; Consultant Radiologist, Hines Veterans' Administration Hospital, Hines, Ill.; Professor of Radiology, Cook County Graduate School of Medicine, Chicago, Ill.; Medical Director, Baccalaureate Program of Radiologic Technology, School of Related Health Sciences, University of Health Sciences/The Chicago Medical School, Chicago, Ill.; and Steven J. Cooper, M.Ed., R.T.(ARRT), Chairman, Department of Radiological Sciences, School of Related Health Sciences, University of Health Sciences/The Chicago Medical School, Chicago, Ill. Cloth. Pp. 388, with 500 illustrations. Price, $20.00. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1973.

Atlas of Tumor Pathology: Second Series, Fascicle 7. Tumors of the Esophagus and Stomach. By Si-Chun Ming, M.D., Associate Professor of Pathology, University of Maryland School of Medicine, Baltimore, Md.; Present address, Professor of Pathology, Temple University Medical School, Philadelphia, Pa. Paper. Pp. 279, with many illustrations. Published by the Armed Forces Institute of Pathology, Washington, D. C., under the auspices of Universities Associated for Research and Education in Pathology, Inc., Bethesda, Md. For sale by the American Registry of Pathology, Armed Forces Institute of Pathology, Washington, D. C. 20306, 1973.

Tomographic Imaging in Nuclear Medicine. The proceedings of a symposium held September 15–16, 1972. Edited by Gerald S. Freedman, B.M.E., M.D., Department of Radiology, Yale University School of Medicine. Cloth. Pp. 271, with many figures. Price, $12.00. Society of Nuclear Medicine, Inc., 305 East 45th Street, New York, N. Y. 10017, 1973.

La Letteratura Radiologica Italiana. Volume XIV. By I. Belli, A. Caresano, and L. A. De Santis. Paper. Pp. 297. La Tipografica Varese, Via Tonale, 49, Varese, Italy, 1973.

Practica Limfografică în Clinică. By T. Roxin, and H. Buhar. Paper. Pp. 172, with some figures. Editura Academiei Republicii Socialiste România. Str. Gutenberg, nr. 3 bis, Sectorul 6, Bucureşti, România, 1973.

Radionuclide Carcinogenesis. Proceedings of the Twelfth Annual Hanford Biology Symposium at Richland, Wash., May 10–12, 1972. Sponsored by Battelle Memorial Institute, Pacific Northwest Laboratories and U. S. Atomic Energy Commission. Edited by C. L. Sanders, R. H. Busch, J. E. Ballou, and D. D. Mahlum. Paper. Pp. 500, with 210 illustrations. Price, $13.60. AEC Symposium Series No. 29. Available as CONF-720505 from the National Technical Information Service, U. S. Department of Commerce, Springfield, Va. 22151, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Queen Elizabeth Hotel, Montreal, P. Q., Canada, September 25–28, 1973.

American Radium Society
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Surfer Hotel, Maui, Hawaii, April 21–25, 1974.

Radiological Society of North America
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 25–30, 1973.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Roosevelt Hotel, New Orleans, La., April 1–6, 1974.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Chicago, Ill., June 15–19, 1974.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel; New York, N. Y., June 10–14, 1974, New York Hilton Hotel; and Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers in June 1974.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1974 is September 30, 1973.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Chairman, Dr. Merrill A. Bender, Suite 2402, 211 E. 43rd St., New York, N. Y. 10017.

American Association of Physicists in Medicine
Secretary, D. John Wright, Ph.D., Department of Radiology, Temple Univ. Health Sciences Center, Philadelphia, Pa. 19140. Annual Meeting.

American Society of Therapeutic Radiologists
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting: Royal Sonesta, New Orleans, La., Oct. 24–28, 1973.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting: Ann Arbor, Mich., Oct. 14–18, 1973.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

Thirteenth International Congress of Radiology
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

Eleventh Inter-American Congress of Radiology
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907. Meeting: Feb. 16–21 1975.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia.

Inter-American College of Radiology
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

Third Congress of the European Association of Radiology
Meeting in June, 1975 in Edinburgh, Scotland.

Second Asian and Oceanian Congress of Radiology
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society, Chapter ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

American Thermographic Society
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Richard H. Greenspan, Dept. of Radiol., Yale University School of Medicine, 333 Cedar St., New Haven, Conn. 06510. Twenty-second Annual Meeting, New York, N. Y., May 9–11, 1974. Dr. Norman E. Chase is in charge of arrangements, New York Medical Center, New York, N. Y. 10016.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Charles J. Masters, Maj. MC, Department of the Air Force, United States Air Force Hospital, Wiesbaden (USAFE) APO New York 09220.

Blockley Radiological Society
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Giacomo Pantaleo, 69-13 Forest Ave., Brooklyn, N. Y. 11227. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Ru-Kan Lin, 191 North St., Buffalo, N. Y. 14201. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER OF ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Emmett R. White, P.O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL TEXAS RADIOLOGY SOCIETY
Secretary-Treasurer, Dr. William H. Roush, Radiology Service, Veterans Administration Hosp., Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

CHICAGO RADIOLOGICAL SOCIETY, DIVISION OF THE ILLINOIS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

COLORADO RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lee Radford, 6011 Harry Hines, Suite 900, Dallas, Tex. 75235. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

DELAWARE CHAPTER OF ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

EAST BAY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

GEORGIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. E. P. Rasmussen, 1968 Peachtree Rd. N.W., Atlanta, Ga. 30309. Fall meeting at Cloister, Sea Island, Ga., Sept. 15–16, 1973.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Stanley I. Worton, 1400 N.W. 10th Ave., Miami, Fla. 33136. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

HAWAII RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Raymond W. Brust, Jr., St. Francis Hosp., 2260 Liliha St., Honolulu, Hawaii 96817. Meets third Monday of each month at 7:30 P.M.

HEALTH PHYSICS SOCIETY
Secretary, Shirley D. Vickers, Catholic Medical Center of Brooklyn and Queens, 88-25 153rd St., Jamaica, N. Y. 11432. Annual Meeting: Houston, Texas, June 16–20, 1974.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

IDAHO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. John R. Dehner, Reid Memorial Hosp., Richmond, Ind. 47374.

IOWA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER OF ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional

Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Eli A. Etscovitz, 10 Stonewood Dr., Newton Centre, Mass. 02159.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. A. G. Fleurquin, Pontiac General Hosp., Pontiac, Mich. 48053. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Howard L. Copas, 827 South 131st St., Omaha, Neb. 68154. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

New England Roentgen Ray Society
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting: May 10, 1974.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 25–27, 1974. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 S. University Dr., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 2100 Rockwood Dr., Sacramento, Calif. 95825. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald W. Philips, 2305 Washington, Milwaukie, Ore. 97222. Meets on second Wesnesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
President-Secretary, Dr. Arch W. Templeton, Univ. of Kansas Med. Ctr., Kansas City, Kan. 66103. Meets 5 times a year on given dates.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. Robert M. Maurer, Santa Rosa Medical Center, San Antonio, Tex. 78207. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President, James R. Collins, 340 Fourth Ave., Suite 15, Chula Vista, Calif. 92010. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, Mary's Help Hospital, 1900 Sullivan Ave., Daly City, Calif. 94015.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Chairman, Dr. Henry W. Wiggins, Jr., Mercy Hosp., 2537 South Prairie Ave., Chicago, Ill. 60616. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Annual meeting: Queen Elizabeth Hotel, Montreal, P. Q., Canada, September 23–24, 1973.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting.

South Bay Radiological Society
Secretary, Dr. Virgil H. Voss, 900 Kiely Blvd., Santa Clara, Calif. 95051. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Associa-

tion meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane I. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 311 Medical Arts Bldg., Chattanooga, Tenn. 37402. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neil, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205. Annual Meeting: La Mansion Hotel, San Antonio, Tex., March 21–23, 1974.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada. Annual meeting.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Don H. Nichols, 5770 S. 300 East, Murray, Utah 84107. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 8952 Bellfonte Rd., Richmond, Va. 23229.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Isaiah J. Seligman, 16 Guion Pl., New Rochelle, N. Y. 10801. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, Gundersen Clinic, LaCrosse, Wis. 54601. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Francisco Miranbell S., Apartado 3352, San José, Costa Rica. Thirteenth Central American Congress of Radiology, San José, Costa Rica, Dec. 4–8, 1973.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. T. Kerr, 32 Welbeck St., London, W1M/7PG, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, 1555 Summerhill Ave., Montreal 109, Que., Canada. Thirty-seventh Annual Meeting, Regency Hyatt House, Vancouver, B. C., April 21–26, 1974.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London WC2A 3PN, England

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie

Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

THE HOSPITAL PHYSICISTS' ASSOCIATION
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

THE ROYAL AUSTRALASIAN COLLEGE OF RADIOLOGISTS
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

SOUTH AMERICA

COLÉGIO BRASILEIRO DE RADIOLOGIA
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

FEDERACIÓN ARGENTINA DE SOCIEDADES DE RADIOLOGÍA
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

SOCIEDAD ARGENTINA DE RADIOLOGÍA
Secretary-General, Dr. Tomás J. Gutiérrez F., Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

SOCIEDAD COLOMBIANA DE RADIOLOGIA
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

SOCIEDAD ECUATORIANA DE RADIOLOGÍA
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

SOCIEDAD PERUANA DE RADIOLOGIA
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

SOCIEDAD DE RADIOLOGÍA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

SOCIEDAD DE RADIOLOGÍA DEL NORDESTE ARGENTINO
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

SOCIEDAD DE RADIOLOGÍA DE LA PLATA
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

SOCIEDADE DE RADIOLOGÍA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

SOCIEDAD DE ROENTGENOLOGÍA Y MEDICINA NUCLEAR DE LA PROVINCIA DE CÓRDOBA
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

SOCIEDAD DE RADIOLOGÍA, RADIOTERAPÉUTICA Y MEDICINA NUCLEAR, DE ROSARIO
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

SOCIEDAD SALTEÑA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

SOCIEDAD VENEZOLANA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

CONTINENTAL EUROPE

ÖSTERREICHISCHE RÖNTGEN-GESELLSCHAFT
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

SOCIÉTÉ ROYALE BELGE DE RADIOLOGIE
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

SOCIÉTÉ EUROPÉENNE DE RADIOLOGIE PÉDIATRIQUE
Secretary, Dr. P. Hicken, The Children's Hosp., Ladywood Middleway, Birmingham B16 8ET, Great Britain.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

SOCIÉTÉ FRANÇAISE DE RADIOLOGIE MÉDICALE, MÉDECINE NUCLÉAIRE ET ÉLECTROLOGIE, and its branches: SOCIÉTÉ DU SUD-OUEST, DU LITTORAL MÉDITERRANÉEN, DU CENTRE ET LYONNAIS, DU NORD, DE L'OUEST, DE L'EST, DU MASSIF CENTRAL, D'OUTRE-MER ET D'ÉTRANGER.
Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

SOCIÉTÉ FRANÇAISE DE NEURORADIOLOGIE
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting: Salpetrière, Paris, Dec. 1, 1973.

ČESKOSLOVENSKÁ RADIOLOGICKÁ SPOLEČNOST
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

DEUTSCHE RÖNTGENGESELLSCHAFT
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

SOCIETÀ ITALIANA DI RADIOLOGIA MEDICA E MEDICINA NUCLEARE
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

SOCIETAS RADIOLOGICA DANICA
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK-8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland. Annual Meeting in Uppsala, Sweden in June 1974.

Socied ad pañola de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Japanese American Radiological Society
Secretary, Thomas J. Imray, Maj. MC, Chief, Radiol. Dep., US Army Hosp., Camp Zana, Japan, APO San Francisco, Calif. 96343. Meets 3rd Thursday, monthly at various locations in Tokyo.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. T. M. Kolawole, University College Hospital, University of Ibadan, Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Head

Underwood, Louis E., Radcliffe, William B., Strickland, Alva L., and Van Wyk, Judson J. Assessment of the sella turcica volume in dwarfed children. *J. Clin. Endocrinol. & Metabol.*, April, 1973, *36*, 734–741. (From: Departments of Pediatrics and Radiology, University of North Carolina School of Medicine, Chapel Hill, N. C.)

Estimation of sellar volume from skull roentgenograms has been shown to correlate well with pituitary gland volume determined at autopsy, although this is more accurate in adults than children.

Studies of sellar volume were made in 38 normal children, 34 growth hormone deficient dwarfs, and 36 dwarfs without growth hormone deficiency. The latter group included growth failure of uncertain cause, chronic disease with growth failure, prolonged corticosteroid therapy and short stature with numerous skeletal abnormalities. The sellar volume was determined by di Chiro's formula. The estimation of sellar area from lateral skull roentgenograms is not an acceptable method.

The sellar age index is the ratio of sellar volume to the mean volume for the corresponding chronologic age (the latter from Fisher and di Chiro). The authors found that the sellar age index for the dwarf controls with normal pituitary function was definitely less than that in the normal controls, but the hypopituitary group showed a marked decrease.

The sellar height index is a ratio of the sellar volume to the mean volume for corresponding height (Fisher and di Chiro's chart). The authors found that the sellar height index showed no significant difference between the controls and the dwarf controls, but a significant decrease in the hypopituitary group.

The patients with small sellas had objective evidence of multiple pituitary hormone deficiencies, had the lowest peak growth hormone responses to stimulation and were most severely dwarfed. Linear growth failure from causes other than growth hormone deficiency may cause proportionate retardation in sellar growth. A sellar age index of less than 0.45 or a sellar height index of less than 0.43 should be considered suggestive of hypopituitarism.

This method should be a useful means of screening dwarfed individuals; however, a normal sellar volume does not exclude pituitary hypofunction.—*Martha Mottram, M.D.*

Tchang, Stanley P. K. The small orbit sign in supraorbital fibrous dysplasia. *J. Canad. A. Radiologists*, March, 1973, *24*, 65–69. (Address: Stanley P. K. Tchang, M.D., Department of Diagnostic Radiology, University Hospital, Saskatoon, Saskatchewan, Canada.)

Monostotic fibrous dysplasia is localized to the cranium in approximately 1 out of every 10 cases as reported by Windholz. The most frequent sites were the frontal and sphenoid bones. When localized to the frontal bone and supraorbital area, the differential diagnosis is principally between fibrous dysplasia and meningioma.

The author reports 8 cases, ranging in age from 5–41 years, of supraorbital fibrous dysplasia and emphasizes the importance of an associated small orbit, which was present in all 8 cases. Each case presented with a small orbit with the reduced size limited to the roof of the orbit. The associated absence or hypoplasia of the corresponding half of the frontal sinus was often found as a secondary sign. Typical associated signs included a supraorbital area of sclerosis, lack of trabecular pattern, principal involvement of the diploic space and smooth outward bulging of the outer table of the cranial vault.

In reviewing the literature the author is unable to find a single case of meningioma giving rise to the combination of small orbit, marked sclerosis of the orbital plate and/or underdeveloped frontal sinus on the affected side. Radiation treatment may result in similar findings but can be excluded by history.—*John T. Underberg, M.D.*

Neck and Chest

Paraskos, John A., Adelstein, S. J., Smith, Roger E., Rickman, Frank D., Grossman, William, Dexter, Lewis, and Dalen, James E. Late prognosis of acute pulmonary embolism. *New England J. Med.*, July, 1973, *289*, 55–58. (Address: James E. Dalen, M.D., Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Mass. 02115.)

The long-term prognosis of acute pulmonary embolism varies between complete recovery from even massive episodes to the development of chronic cor pulmonale.

To evaluate factors which might affect outcome, and to determine if possible the incidence of cor pulmonale, this study of 60 consecutive patients who survived an episode of acute pulmonary embolism was carried out, with evaluation of the patients 1 to 7 years after the acute episode.

The original diagnosis was established by demonstration of unequivocal angiographic evidence of embolism. Out of a group of 289 patients suspected clinically of having pulmonary embolism, in only 75 was the diagnosis substantiated by angiography, emphasizing the difficulty in clinical diagnosis. Of these 75, 15 died during the hospital stay. Most of the others were treated by anticoagulants, venous interruption, or both. One of the 60 patients had a

normal repeat pulmonary arteriogram before discharge, and another was lost to follow-up.

One-third of the 58 remaining patients died during the follow-up period, and only 1 of these died of the long-term effects of pulmonary embolism: chronic cor pulmonale with progressive right ventricular failure. The 39 survivors were a slightly younger group. Surprisingly, the acute episode in those who survived involved more than 50 per cent of the lung in 41 per cent, but in only 21 per cent of those who died during the follow-up period. Shock had occurred in 28 per cent of the survivors, but in only 11 per cent of the nonsurvivors. The most striking difference between the 2 groups, however, was the much higher incidence in nonsurvivors of preexisting heart disease: 31 per cent of the survivors had prior heart disease, compared with 89 per cent of the nonsurvivors; 8 per cent of the survivors had prior left ventricular failure, compared with 68 per cent of the nonsurvivors.

Both survivors and nonsurvivors were evaluated as to the degree of resolution of the original embolism, the former by arterial blood gases and lung scans, and the latter, when possible, by postmortem examination. Of those in whom the data were adequate, resolution appeared to be complete in 65 per cent and partial in 23 per cent, while unresolved embolism persisted in only 5 patients. There was a documented recurrent episode of pulmonary embolism in only 1 patient, and there was evidence of chronic cor pulmonale due to pulmonary embolism in only 1.—*E. Frederick Lang, M.D.*

Kempf, K., Capesius, P., and Mugel, J. L. Un cas de calcinose pulmonaire au cours d'une myélomatose diffuse. (A case of pulmonary calcinosis during the course of diffuse myelomatosis.) *J. de radiol., d'électrol. et de méd. nucléaire*, Dec., 1972, *53*, 861–865. (Address: Pr agr. K. Kempf, Service de Radiologie de la Clinique médicale, C.H.U., F.67-Strasbourg, France.)

This rare complication had already been reported in 1855 by Virchow who coined the term "calcium metastases" to characterize calcium deposits in the stomach and lungs in association with hypercalcemia. Such a syndrome was attributed to an increased bone resorption coupled with a lowered excretion by inflammatory kidneys. "Metastatic calcification" is the term now widely used to describe calcium deposits in normal tissues, usually viscera, complicating a destructive bone process accompanied by a disturbed phospho-calcic metabolism. Such deposits may occur in the course of hyperparathyroidism, hypervitaminosis D, malignancies, multiple myelomas, and severe nephropathies. In decreasing order, the sites of predilection are as follows: kidneys, lungs, stomach, heart, arteries. The authors prefer the term "visceral" to "metastatic" calcinosis.

They report the case of a 66 year old female admitted to hospital in a state of cachexia. Chest roentgenograms showed multiple nodular and annular calcifications, round or oval, and distributed mainly in both lung bases. Tomography confirmed their peripheral localization. A skeletal survey disclosed a generalized demineralization and large punched-out areas in the skull. Laboratory tests revealed a mild anemia with thrombopenia and elevated sedimentation rate. Blood calcium was at 162 mg. per cent and phosphorus at 22 mg. per cent. A Bence-Jones protein K was demonstrated by immuno-electrophoresis. The urines contained traces of albumin but no Bence-Jones protein. Calciuria was increased at 417 mg. and phosphaturia at 339 mg. for 24 hours. Changes due to hypercalcemia were present on the electrocardiogram and electroencephalogram. The patient's condition deteriorated progressively; she became comatose and died within 10 days after admission. Autopsy was refused.

In their discussion, the authors refer to 2 exhaustive analyses of metastatic calcifications, one by Mulligan (1947) based on 100 cases, the other by Franke (1960) assembling 157 cases.

The pathologic analysis revealed that the calcium was precipitated more frequently at the periphery of the lung, where it was found mainly in alveolar septa on the capillary vessels and on the connective matrix. Acid and neutral mucopolysaccharides were seen accumulated with calcium in the connective matrix. Calcium was seen less frequently in the walls of the main vessels and bronchi.

Radiologic studies have demonstrated that these pulmonary calcifications may appear rapidly, since they were absent on the roentgenograms, but observed at autopsy a few weeks later. In the authors' case, no previous roentgenograms were available.

The authors discuss the radiologic differential diagnosis and then consider the physiopathology. Visceral calcinosis is due to the increase of calcium and phosphorus in the blood. Normally, the serum albumins absorb the excess of calcium. In a case of multiple myeloma, the protein loss in the urine could explain the calcium deposits in some viscera. The role of hyperphosphoremia is also brought to light by the fact that visceral calcinosis can occur following inorganic phosphate perfusions for the treatment of acute myelomatous hypercalcemia.

Local factors may also play a role in visceral calcinosis. Acid-excreting organs (lungs, kidneys, stomach) are a favorite site; a local alkalinization resulting in a lowered solubility of calcium salts could be a causative factor. In cases of renal insufficiency, metabolic acidosis is compensated by an increased CO_2 excretion from pulmonary hyperventilation. This mechanism could account for the extensive calcifications in the lungs. Finally, the presence of acid and neutral mucopolysaccharides

found in association with calcium deposits favors their formation by a process of ion exchange.—*H. P. Lévesque, M.D.*

Brandt, Peter W. T., Clarkson, Patricia M., Neutze, John M., and Barratt-Boyes, Brian G. Left ventricular cineangiography in endocardial cushion defect (persistent common atrioventricular canal). *Australasian Radiol.*, Dec., 1972, *16*, 367–376. (From: Green Lane Hospital, Auckland, New Zealand.)

The authors review their experience utilizing left ventricular oblique cineangiocardiography in diagnosing 39 cases of endocardial cushion defects. The angiographic appearances in systole and diastole in both the right anterior oblique (R.A.O.) and left anterior oblique (L.A.O.) are reviewed.

In the *L.A.O. projection* during diastole the mitral valve orifice appears more medially located overlying the septum. The inflow of unopacified blood accounts for the radiolucency and the goose-neck deformity. In systole the abnormal mitral leaflets can be seen bulging into the defect. They often apper septated and a horizontal radiolucent linear filling defect is seen near the middle of the leaflets representing the cleft anterior leaflet of the mitral valve. These are the typical findings in the incomplete endocardial cushion defect in which the ventricular septal defect component is not large. In the complete form of atrioventricular canal, the ballooning leaflets are not as discernible because of the large size of the ventricular septal defect and because of the common attachments of the anterior mitral leaflet and the septal tricuspid leaflet.

In the *R.A.O. projection*, the left ventricular outflow tract appears narrow from behind and below, depending in part on the extent of the ventricular septal defect. The posterior margin of the narrowed outflow tract consists of valve leaflet tissue and may show the typical scalloped margin with the radiolucent cleft in the anterior leaf of the mitral valve. This is the best view for assessing mitral regurgitation, while the L.A.O. projection is frequently misleading, since contrast material flowing across the ventricular septal defect or directly into the right atrium or left atrium may be confused with another.

The authors were able to assess the size of the ventricular septal defect quite well by angiocardiography. In general, all cases with the complete form of atrioventricular defect had systemic pressure in the right ventricle and a large ventricular septal defect. There was no correlation between the magnitude of mitral regurgitation and the presence or size of a ventricular septal defect.

The authors conclude that the cine technique with oblique positioning is essential for the assessment of the relative importance of the ventricular septal defect and the mitral regurgitation.—*Robert I. White, Jr., M.D.*

Berg, Gerald R., Hutter, Adolph M., Jr., and Pfister, Richard C. Electrocardiographic abnormalities associated with intravenous urography. *New England J. Med.*, July, 1973, *289*, 87–88. (From: Departments of Radiology and Medicine, Harvard Medical School and Massachusetts General Hospital, Boston, Mass.)

Reversible reactions to intravenous contrast media occur in approximately 1 out of 2,000 studies, and in various reports death occurs in between 8.6 and 19 times per million examinations. Death is usually sudden and is frequently associated with cardiac arrest.

In an attempt to evaluate the nature of cardiac changes associated with the use of the current larger doses of contrast medium, electrocardiographic studies were made on 30 patients before, during, and after introduction of the medium. In 20 of the patients the examination included injection of 150 ml. of meglumine diatrizoate (infusion urography), and in 10 the standard dose of 50 ml. was used.

Ten of the 30 patients presented abnormal post-injection electrocardiograms, with 6 major and 4 minor abnormalities. All the major changes occurred in those undergoing infusion study. Four patients showed ischemic changes, and 1 of these also showed intraventricular conduction delay associated with chest pain. Virtually all of the major abnormalities in the electrocardiograms occurred with the triad of high dose infusion studies, in older patients, and in the presence of clinical evidence of heart disease.

The mechanism of the changes is uncertain, but the introduction of a sudden hypertonic volume load, neurogenic stimulation, and direct myocardial toxicity should be considered. In a patient who has had a myocardial infarct, and in whom treatment with anticoagulants is associated with microhematuria, delaying the pyelography should be considered, because of the high risk of the immediate examination.—*E. Frederick Lang, M.D.*

Abdomen

Cotton, P. B., Rosenberg, M. T., Waldram, R. P. L., and Axon, A. T. R. Early endoscopy of oesophagus, stomach, and duodenal bulb in patients with haematemesis and melaena. *Brit. M. J.*, June, 1973, *2*, 505–509. (From: St. Thomas's Hospital, and Brook General Hospital, London, England.)

For the accurate diagnosis of the source of upper gastrointestinal bleeding, there is no substitute for actually seeing the bleeding point. Barium meal examinations cannot demonstrate erosions or small ulcers, even if a lesion is shown, it may not be the

actual source of the bleeding. The recent development of fiberoptic instruments has facilitated examination in acute hemorrhage and has extended the range of examination. Although peptic ulcer accounts for about half the cases in large series, there are many other causes of acute bleeding, and patients with known ulcers or varices may bleed from other sites.

In this series 135 males and 73 females were considered, and of these, 68 per cent presented with hematemesis with or without melena, and 32 per cent with melena alone. Seventy-two per cent were examined within 48 hours of overt bleeding, and the patients later in the series were seen after a shorter interval than the earlier patients. They were seen during or after blood replacement and were examined with a variety of flexible instruments without being moved from bed. One child aged 2 years was examined with a fiberbronchoscope. Three alcoholics could not tolerate the examination.

Instruments were advanced beyond varices unless they were actively bleeding or showed adherent clot. The pylorus was passed in all but 6, and in most patients, the examination included the descending duodenum. Blood was encountered in 33 per cent of patients examined within 48 hours of overt bleeding, but this interfered significantly in only 8 examinations. Many patients were found to have 2 lesions. Thus, 18 out of 68 duodenal ulcers, 4 out of 63 gastric ulcers, and 4 out of 11 varices were not responsible for the current bleeding episode. Lesions were not found in 29 adequate examinations. More distant lesions were found in 4 of these and another 4 were thought not to have bled.

A positive diagnosis was arrived at in 80 per cent of patients, and in an additional 14 per cent no lesion was found. Age, sex, and clinical history were poor guides to the cause of bleeding. Bleeding acute erosions were found in only 7 of the 49 patients who gave a history of recent salicylate ingestion.

The authors suggest early endoscopy in acute bleeding, within the first 24 hours after admission. If this fails to provide a diagnosis, it is logical to proceed to barium studies, unless there is excessive bleeding, which will also interfere with the latter study. In this case arteriography will be the most helpful examination.—*E. Frederick Lang, M.D.*

Baum, Stanley, Rösch, Josef, Dotter, Charles T., Ring, Ernest J., Athanasoulis, Christos, Waltman, Arthur C., and Courey, W. Robert. Selective mesenteric arterial infusions in the management of massive diverticular hemorrhage. *New England J. Med.*, June, 1973, *288*, 1269–1272. (Address: Dr. S. Baum, Department of Radiology, Massachusetts General Hospital, Boston, Mass. 02114.)

The combined experience from the Massachusetts General Hospital and the University of Oregon in treating massive diverticular hemorrhage by arterial infusion of pituitrin is presented.

The diagnosis was made in all 15 patients by correlation of the angiographic appearance and the findings on subsequent barium enema examination. Pitressin was infused into the inferior or superior mesenteric artery at an initial dose rate of 0.2 units per minute. Repeat angiography was performed, 15 minutes after the beginning of the infusion to document that vasoconstriction occurred.

Immediate control of bleeding was obtained in all cases. The infusions were gradually tapered over the next 24 hours and the catheters were left in place an additional 6 to 12 hours in case the bleeding should recur. Seven of the 15 patients received no further therapy and they have been followed for a period of 6 to 30 months without re-bleeding. Four of the patients had elective partial colectomies and 4 patients underwent surgery for re-bleeding.

In keeping with previous reports, there was a high incidence of bleeding from the ascending colon (11 of 15 cases). No complications occurred in this series—specifically there was no evidence of intestinal or myocardial ischemia.—*Robert I. White, Jr., M.D.*

Leger, L., Cachin, M., Chapuis, Y., Delaître, B., Launois, P., de Saint-Maur, P. P., and Bonnin, A. Hamartomes hépatocytaires du foie. (Hepatocytic hamartomas of the liver.) *Presse méd.*, Feb., 1973, *2*, 353–358. (Address: Pr L. Leger, Clinique chirurgicale, Hôpital Cochin, 27 rue du Faubourg Saint-Jacques, F 75674 Paris Cedex 14, France.)

A hamartoma, as defined by Albrecht, is a pseudotumoral malformation attributable to a disorganization. Hamartomas located in the liver are rare, but an increasing number (66 cases) has been reported in the past 10 years. This fact might be explained by a more enterprising surgery and also a more refined histologic analysis.

The authors report 3 cases of hepatocytic hamartomas.

Case 1. A 17 year old male. The physical examination disclosed a tumor in the left hypochondrium which was asymptomatic and even visible. Its lower border was firm, smooth, and moved with respiration.

Laboratory Data. The leukocytic count rose to 10,500 per mm.^{3} with 78 per cent polymorphonuclears; the sedimentation rate was 100; fibrinemia increased to 7 per cent; the functional hepatic tests were slightly disturbed. Electrophoresis: increased α_2 (14 per cent) and decreased γ (8.5 per cent) globulins.

Radiologic Investigation. Radiopaque calcifications were present in the left costovertebral angle with lateral displacement of the stomach. Hepatic scintigraphy revealed a large nonfunctioning area in the left lobe surrounded by hepatic tissue. Arteriography

showed a displacement of the main hepatic artery with its left branch and the splenic artery circling the lower half of the tumor. The vascularity was increased and stasic at the site of the tumor.

Surgery. Left hepatectomy and resection of 2 large inflammatory lymph nodes above the pancreas.

Pathologic Analysis. The tumor was composed at its periphery of proliferating hepatocytes, clearer and more voluminous than normal, with rare blood vessels and few biliary canaliculi. In the center, it was composed of dense hyalinized sclerosis. The fetal aldolases were found increased in the tumoral tissue.

Case II. A 24 year old male. The patient had complained of increasing pain in the right hypochondrium for the past 13 months, with loss of weight. A globular tumor was present in the right hypochondrium, overlapping the epigastric area. The tumor was smooth and painless on palpation, and mobile.

Laboratory Data. Sedimentation rate 123; alkaline phosphatase and cholesterol were elevated. The protein electrophoresis revealed lowered albumin (37 per cent) and increased α_2 (22 per cent) and β (19.2 per cent) globulins.

Radiologic Investigation. Scintigraphy showed a thin functional layer of heptic tissue in the upper part of the right lobe; the left lobe was normal. Arteriography revealed a great dilatation of the main hepatic artery and a displacement of its right branch in the direction of the heart. The tumor was hypervascularized by irregular branches and pools suggesting a malignant process.

Surgery. Right hepatectomy. The hepatic tumor was the size of an adult's head, soft and vascularized.

Pathologic Analysis. Proliferation of huge hepatocytes, foci of steatosis and biliary retention; dysplasic aspect of blood vessels with large sinusoids.

Case III. A 23 year old female. For the past 16 months, the patient had complained of severe epigastric cramps radiating to the lumbar area. The bouts would recur every half hour for about 10 days, with symptom-free intervals lasting 2 months. She also presented diarrhea, asthenia and loss of weight. A sensitive epigastric mass was palpated.

Laboratory Data. Normal.

Radiologic Investigation. Hypertrophy of the left hepatic lobe, by scintigraphy with homogeneous fixation. Arteriography revealed a median displacement of the left hepatic artery by a hypervascularized tumor displaying a fine arterial network mimicking an angiomatous tumor.

Surgery. Left hepatectomy.

Pathologic Analysis. The tumor measured 10 cm. in diameter and was attached to a thin layer of hepatic tissue. On cross-section, the tumor showed a nodular structure similar to a cirrhotic liver. It was composed of a proliferation of nearly normal hepatocytes interspaced by sinusoidal capillaries. Blood vessels were present in the sclerotic septa of the pseudolobules.

In their conclusion, the authors mention that the cellular polymorphism might explain the fact that these tumors have been described as adenomas or benign hepatocholangiomas. Malignant transformation has not been reported. Fetal aldolases contained in the tumor bespeak an embryonal origin.—*H. P. Lévesque, M.D.*

Genitourinary System

Conté, J., Guiraud, R., Conte-Viatge, A., Joffre, F., and Suc, J. M. Les fistules artério-veineuses rénales après ponction biopsie du rein; rôle favorisant de la dystrophie conjonctivo-élastique de Marfan: étude critique du concept d'ischémie rénale. (Arteriovenous fistulas after needle biopsy of kidneys; role favoring Marfan's conjunctivo-elastic dystrophy: critical study of the concept of renal ischemia.) *J. d'urol. et de néphrol.*, Sept., 1972, *78*, 776–782. (From: Service de Néphrologie, CHU Purpan, F-31-Toulouse, France.)

The authors report in detail 2 patients, who developed arteriovenous fistula after transcutaneous needle biopsies of the kidneys. In the discussion, they point out that recent studies show that 30 per cent of such renal biopsies develop arteriovenous fistulas, which is an impressive risk. This figure is as high as that of congenital and idiopathic renal arteriovenous fistulas.

It seems that the following factors enhance the development of such a complication: (1) the rich arterial and venous systems of the kidney in 2 perpendicular planes; (2) the pathologic condition of such kidneys with arterial hypertension, vascular sclerosis and necrotizing angiitis—all tending to prevent hemostasis in case of punctured arterial and venous vessels. Thus, a hypertensive patient's kidneys run 3 times a higher risk of such a complication, compared to one having a normal blood pressure; (3) the deeper the penetration of the biopsy needle, and the more medial, the higher is the risk.

The prognosis of such arteriovenous fistulas is good, as 50 to 70 per cent of these close spontaneously. However, if fibroelastic dystrophic changes are present in the kidneys (such as in Marfan's disease), the fistula will expand, rather than close, and nephrectomy will be necessitated (as in the second case of the authors).

Whatever their origin, arteriovenous fistulas do not seem to cause renal ischemia and once arterial hypertension appears, it will persist, 1 out of 2 cases, after nephrectomy.—*Jirair N. Sarian, M.D.*

Pontonnier, F. Compressions calicielles supérieures d'origine vasculaire: à propos de 5 observations. (Superior calyceal compressions of vascular origin: report of 5 cases.) *J. d'urol. et de néphrol.*, Oct.–Nov., 1972, *78*, 807–820. (From: Service d'Urologie du Pr

A. Lhez, Hôpital Lagrave, 31-Toulouse, France.)

A short review of the literature on the subject is first presented.

The author then discusses 5 cases of superior calyceal dilatation due to infundibular compression by intrarenal vessels, 4 of which were arterial and symptomatic (Fraley's syndrome), and 1 venous and asymptomatic. Each of these cases is illustrated with intravenous pyelograms and arteriographic roentgenograms.

Three of the 4 symptomatic cases were treated by excision of the superior pole of the kidney involved. The pathologic findings were: localized chronic pyelonephritis; interstitial pyelonephritis with atrophy of the renal parenchyma.

The fourth symptomatic case of superior calyceal compression was not treated surgically because both the radioactive hippuran nephrogram and the scintigraphic studies revealed no abnormality of the affected pole.

The author points out the significance of Fraley's syndrome in the differential diagnosis of tuberculosis and intrarenal calculus.—*Jirair N. Sarian, M.D.*

Traumann, G., Holsten, D. R., and Berndt, G. Extravasalbildung nach Fornixrupturen bei der Infusionsurographie. (Extravasation due to fornix rupture during infusion pyelography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Oct., 1972, *117*, 429–433. (Address: Priv.-Doz. Dr. D. Holsten, Zentrale Röntgenabt. im Krankenhaus Siloah, Austrasse 46, 3 Hannover, Germany.)

After the introduction of infusion pyelography, 10 cases of extravasation due to fornix rupture were observed within a period of 3 years.

In 1 case there was a history of blunt abdominal trauma, in 7 cases a ureteral stone with obstruction was demonstrated, and an obstruction of unknown cause was present in the remaining 2 cases.

The frequent occurrence of extravasation in patients with obstructing ureteral calculus is thought to be the result of 2 factors: (1) the increased intrapelvic pressure secondary to the ureteral obstruction; and (2) the additional rapid increase in the intrapelvic pressure following the administration of a large volume of contrast medium with the infusion pyelography.

It is, therefore, recommended that only conventional intravenous pyelography be performed in all patients with clinical suspicion of an obstructing ureteral calculus.—*Peter F. Winter, M.D.*

Gynecology and Obstetrics

Sirbu, P., Vasiliu, Christina, and Goranov, Mitko. La cicatrice utérine après les opérations césariennes: étude radiologique. (The uterine scar after cesarean section; a radiologic study.) *J. Gyn. Obst. Biol. Repr.*, Sept., 1972, *1*, 563–574. (From: Clinique d'Obstétrique et de Gynécologie, Maternité "Giulesti," Bucarest, Romania.)

This report is based on 40 patients who had been submitted to a cesarean section at least 6 months before the hysterographic examination. The "cervico-hysterography" was performed with a modified instrument composed of a short cannula (7 mm. long) and a large occlusive cone (3.5 cm. in diameter), which permitted a satisfactory opacification of the cervical canal. Lipiodol (40 per cent) at room temperature was the contrast agent used. Three films were exposed: in an oblique incidence; an anteroposterior incidence, while the cervix was under traction; and another anteroposterior incidence after removal of the cannula.

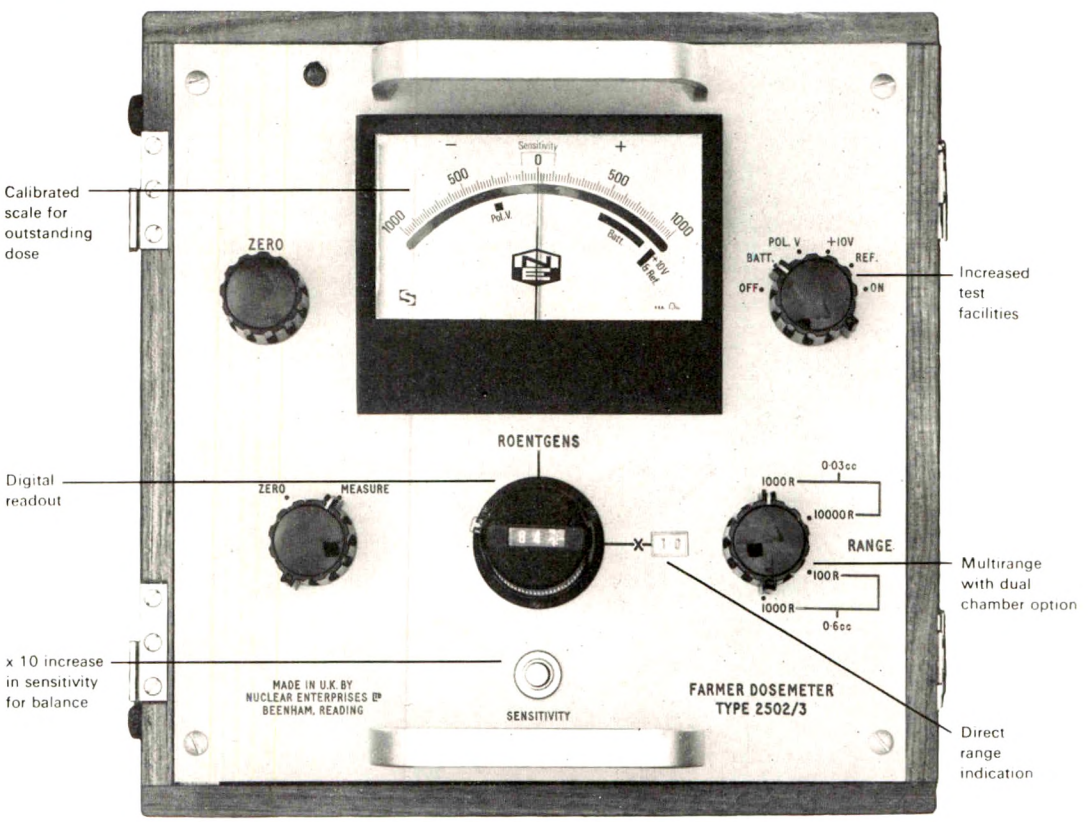
The hysterographic findings were divided into functional and morphologic anomalies. As a functional sign, spasm of the cervico-isthmic segment was considered a debatable one, since no comparable examination had been performed before the cesarean section. The morphologic changes were classified as direct and indirect signs.

The direct signs included: (a) opaque shadows (spicules, spurs, diverticular formations, sinuses and fistulae); and (b) filling defects created by an hypertrophied scar (invagination, notching or lacunae). These changes were localized around the isthmus in 70 per cent of the cases, and in the cervical canal in 30 per cent. According to the size of these changes, they were divided into 4 categories: Group I—no recognizable scar (2 cases or 5 per cent); Group II—anomalies of less than 3 mm. (23 cases or 57.5 per cent); Group III—direct and indirect signs ranging between 3 and 5 mm. (7 cases or 17.5 per cent); Group IV—anomalies greater than 5 mm. (3 cases or 7.5 per cent). Three cases presented changes that could not be interpreted.

The indirect signs consisted of: (a)—axial deviation of the uterus with isthmic angulation attributable to sclerosis surrounding the scar; (b)—elongation of the cervical canal; (c)—gaping of the cervico-isthmic junction; and (d)—vascular and lymphatic reflux by increased mucosal fragility.

The authors then present a correlation of the radiologic findings and the various factors involved in the abnormal scarring. They found faulty scars more frequently in women who had had more than 3 children. Faulty scars were also observed more often if the cesarean section had been performed more than 12 hours after induction of labor. Other factors were placenta previa, surgical suture in 2 planes, and postoperative infection or anemia.—*H. P. Lévesque, M.D.*

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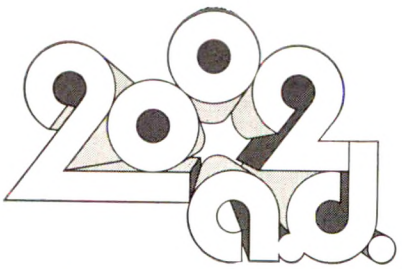
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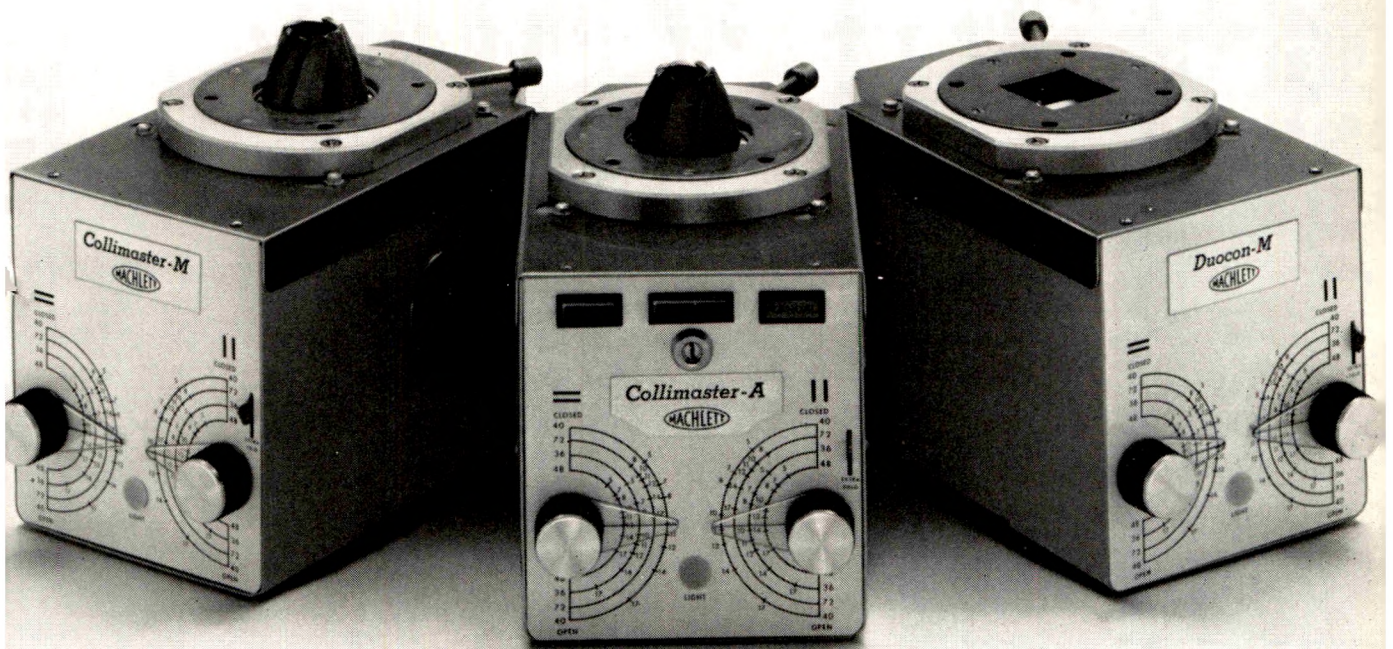


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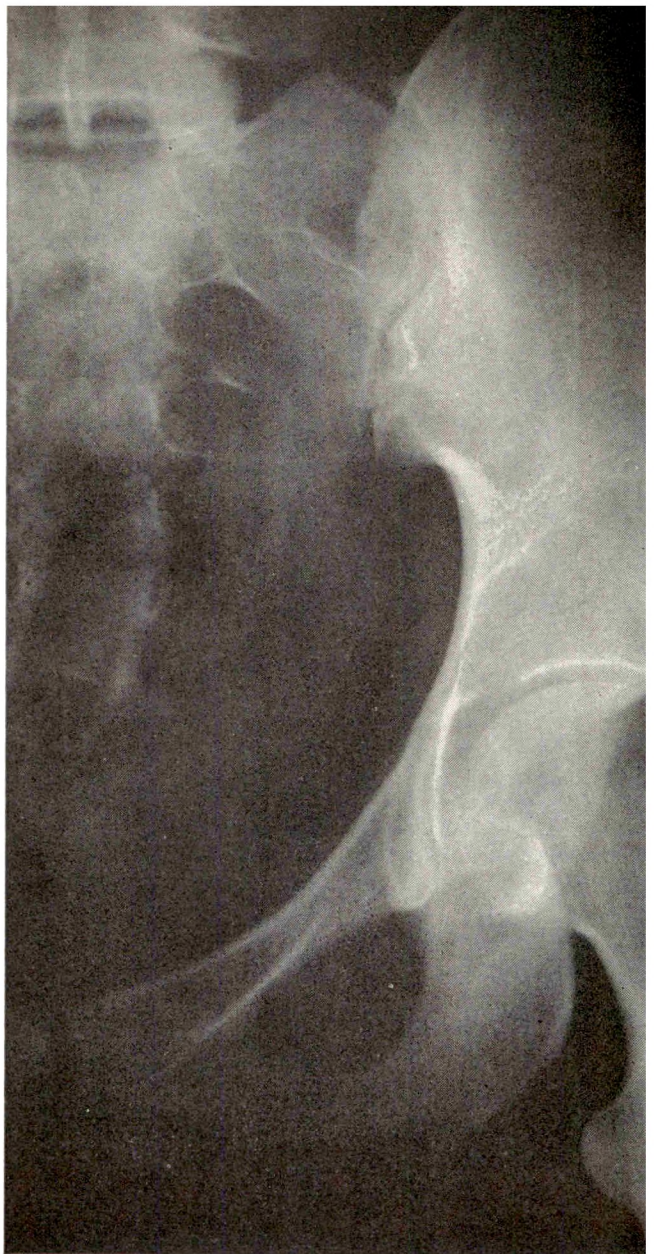
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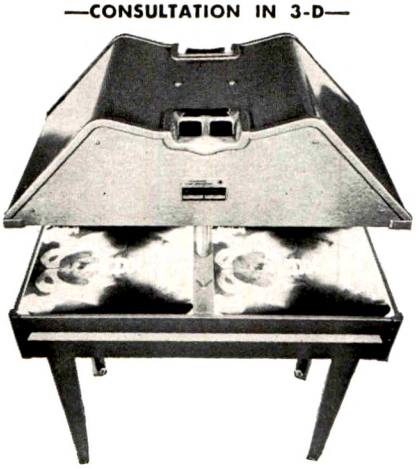
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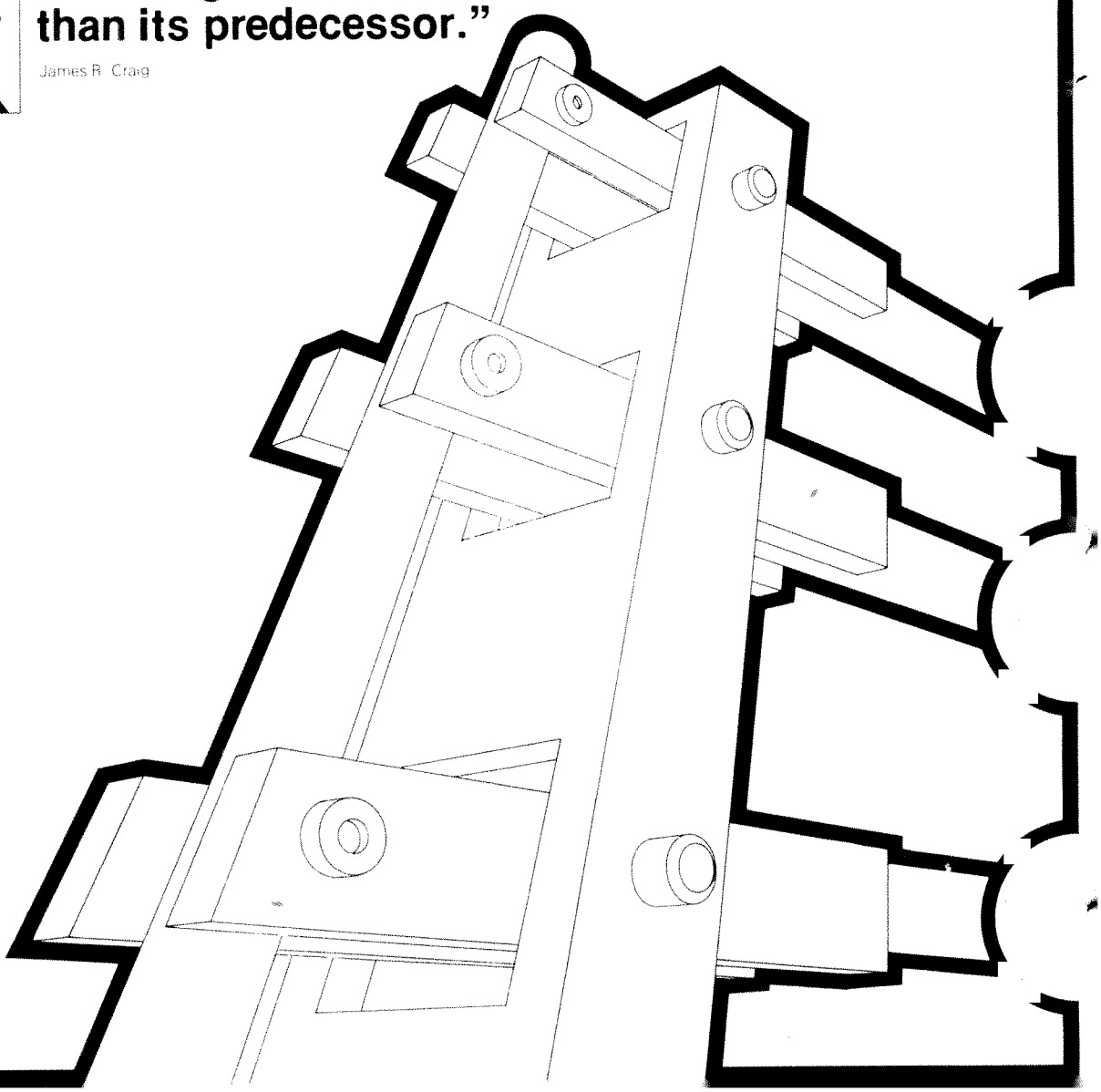
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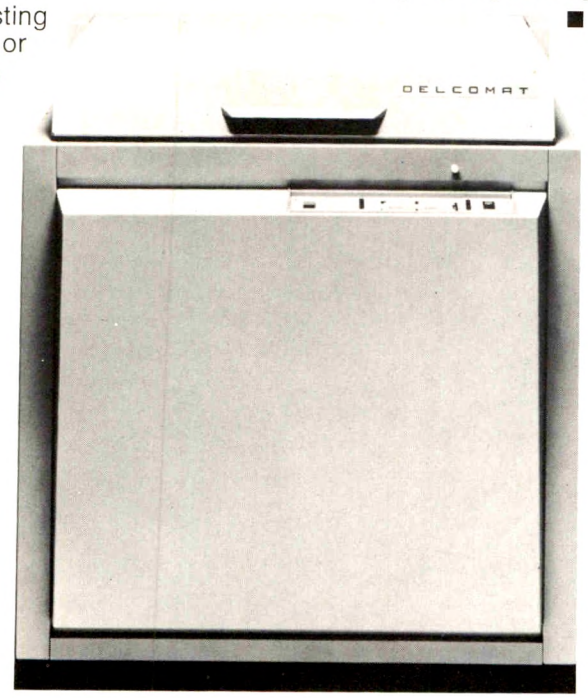
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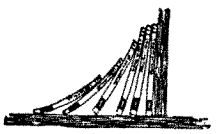
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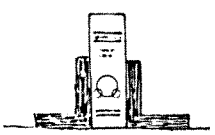
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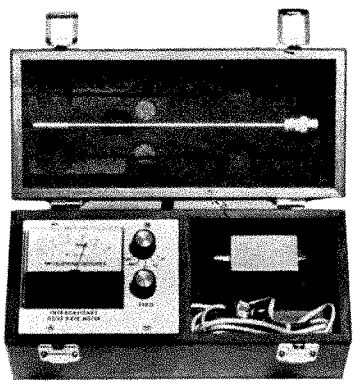
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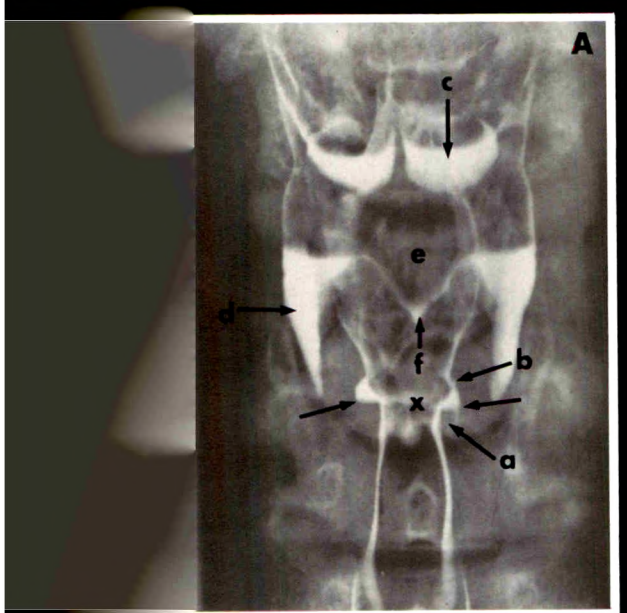
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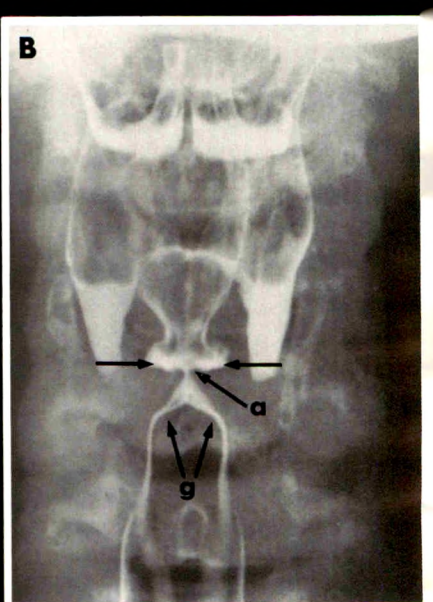
Here demonstrated are four different posteroanterior views of a normal larynx exposed during various respiratory maneuvers. For contrast, two abnormal studies follow.

Figure I. Normal Laryngogram.



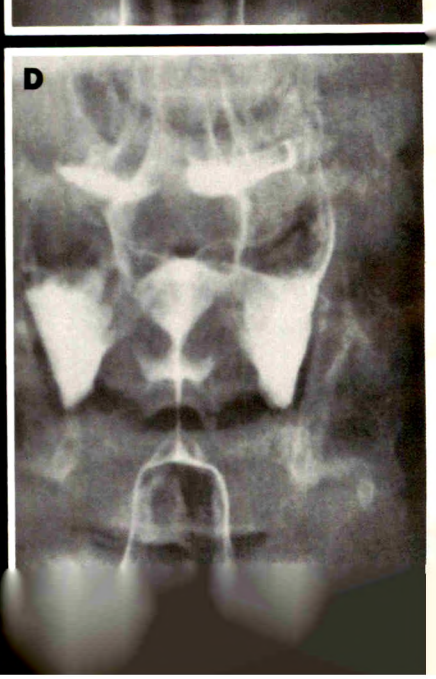
A. Quiet Inspiration: During inspiration, the true (a) and false (b) cords retract leaving the glottic aperture (x) wide open and the laryngeal ventricles (arrows) collapsed. The valleculae (c), pyriform sinuses (d), laryngeal vestibule (e) and interarytenoid groove (f) are well delineated.

B. Phonation: When the vowel "eee" is sounded at a high pitch, the cords approximate each other with the true cords (a) meeting in the midline to form the laryngeal ventricles (arrows). The subglottic angles (g) are now sharply defined.





C. Modified Valsalva Maneuver: This is likened to blowing the trumpet with lips closed and cheeks fully expanded. Now the entire hypopharynx distends; the ballooning of the lateral pharyngeal walls (arrows) reflects normal pliability.



D. True Valsalva Maneuver: As one strains against the closed glottis, the cords, ventricles and laryngeal vestibule contract even more and form a pattern so symmetrical that its slightest asymmetry is considered one of the cardinal roentgen

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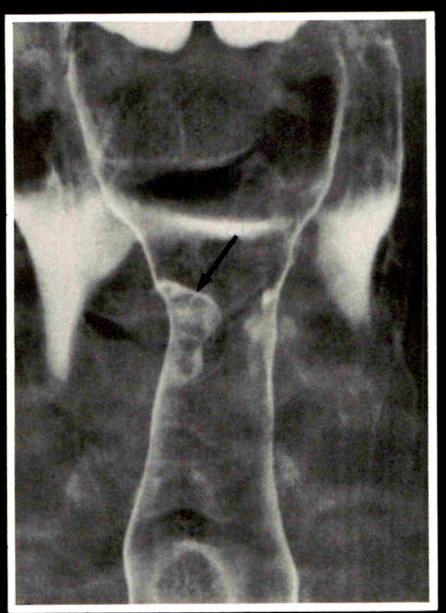


Figure II. Squamous-cell carcinoma of the right vocal cord (arrow); exposure made during quiet inspiration.

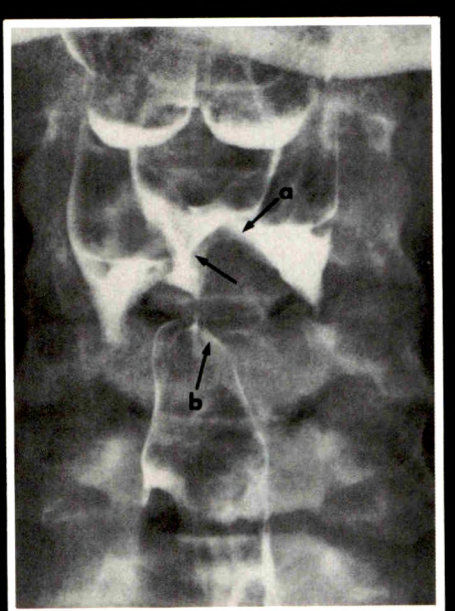


Figure III. Squamous-cell carcinoma of the left false cord (arrow) which has displaced the true cord and ventricle on the same side. Note the encroachment upon the medial aspect of the homolateral pyriform sinus (a) and subglottic angle (b); exposure made during phonation.

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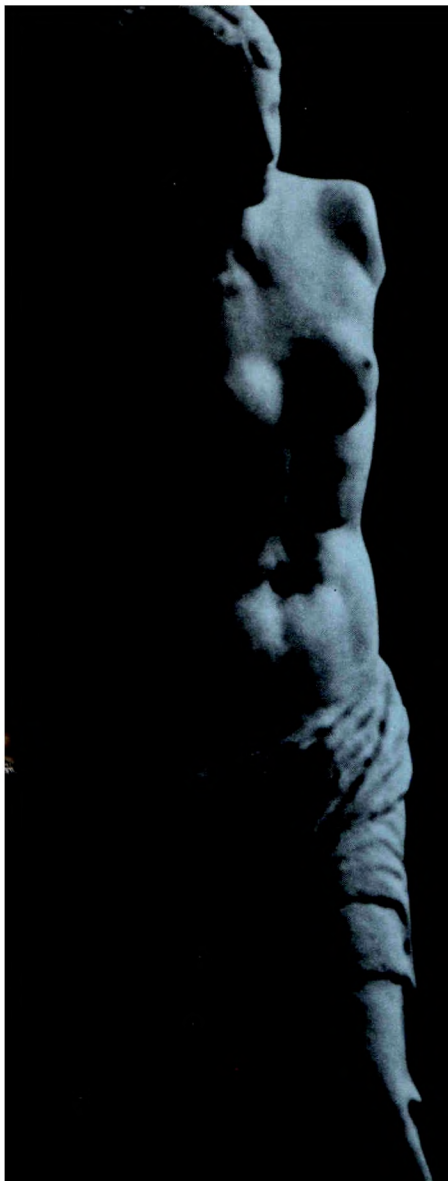
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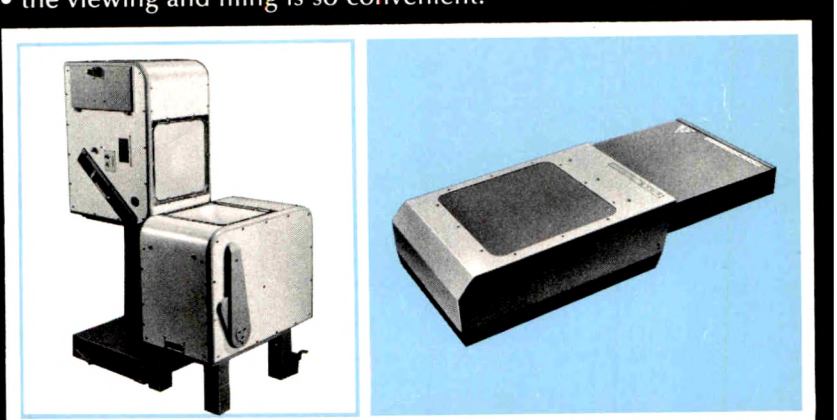
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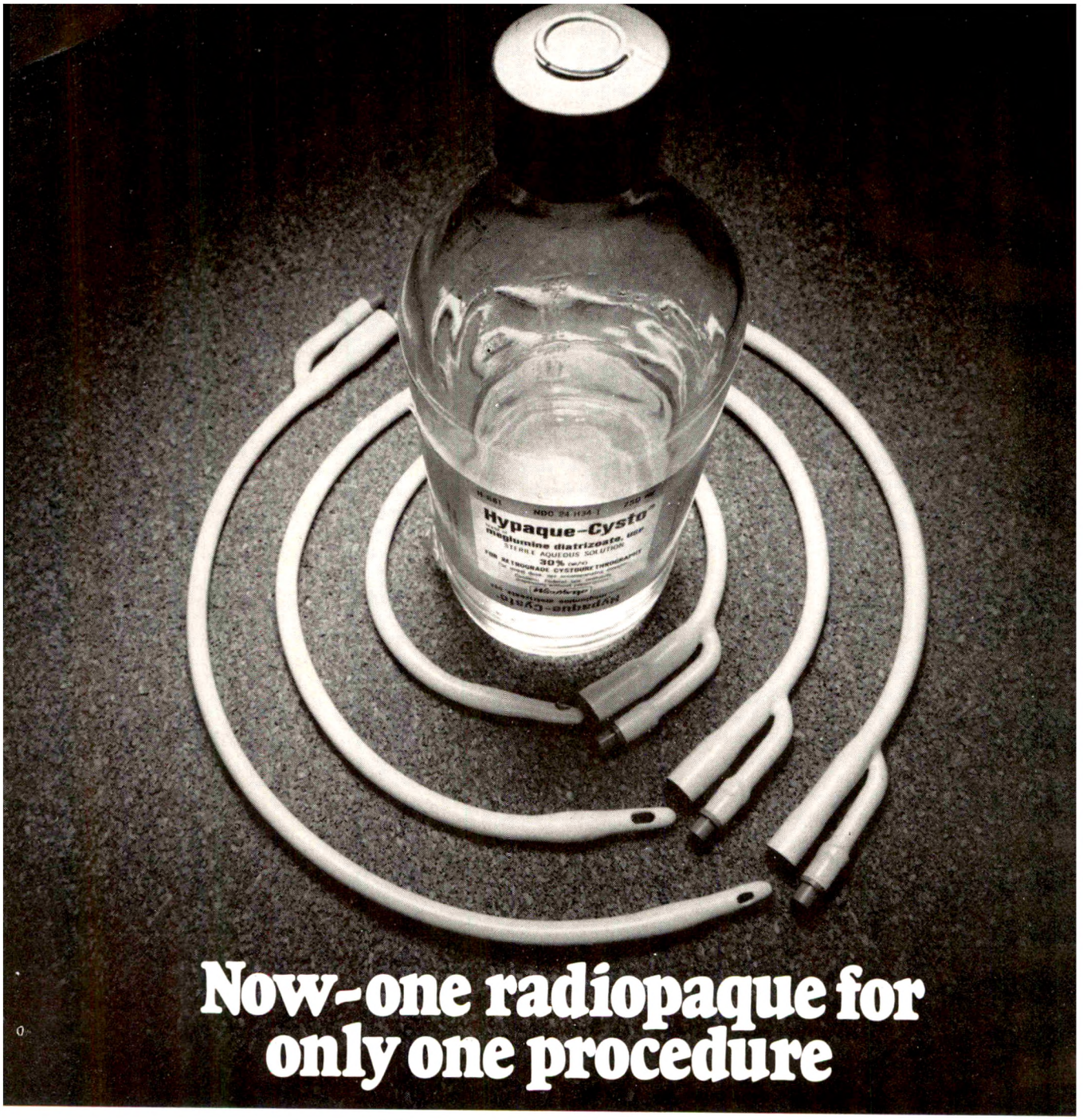


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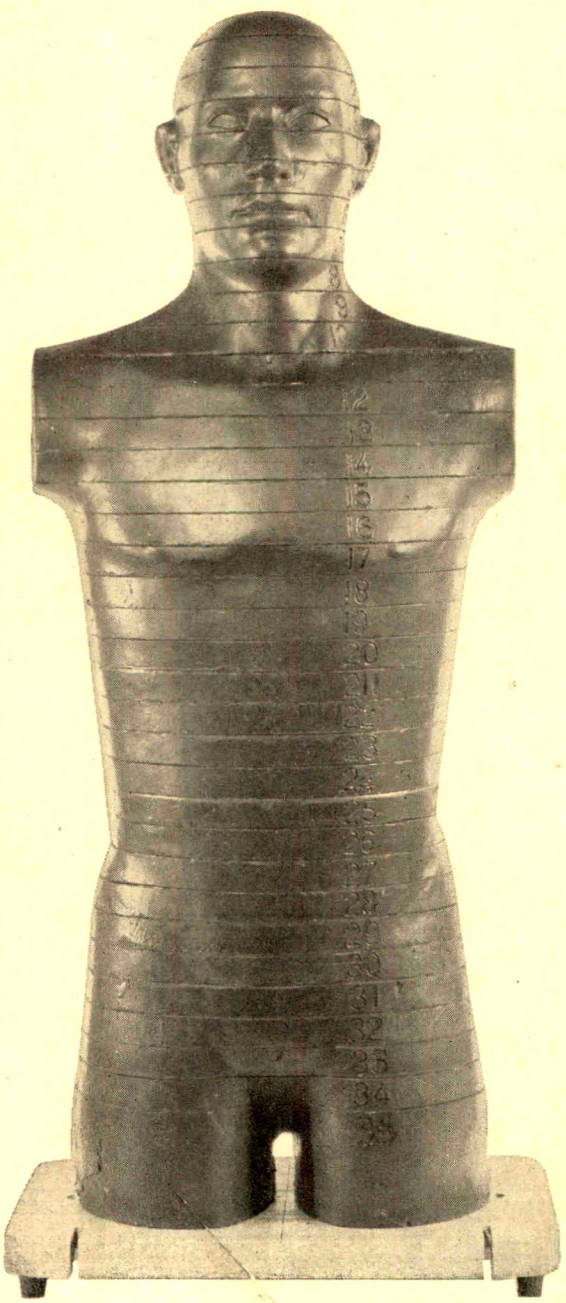
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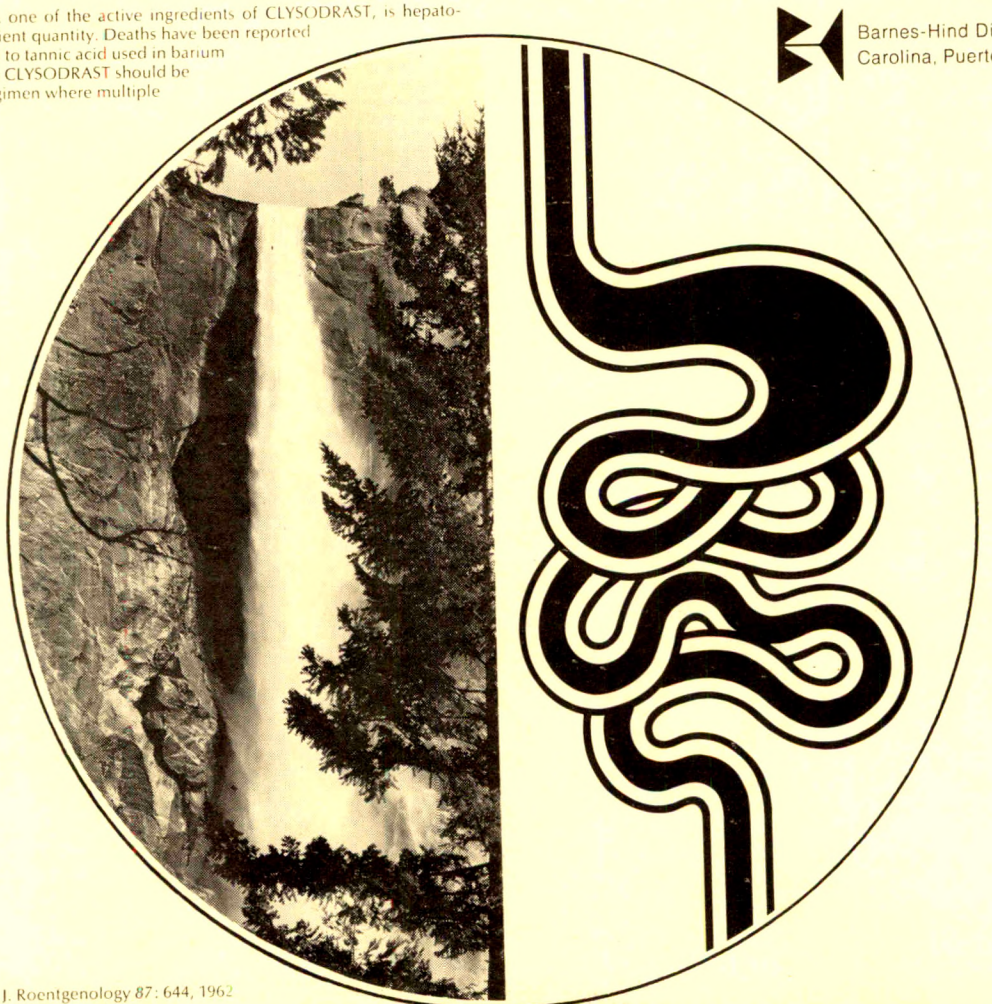


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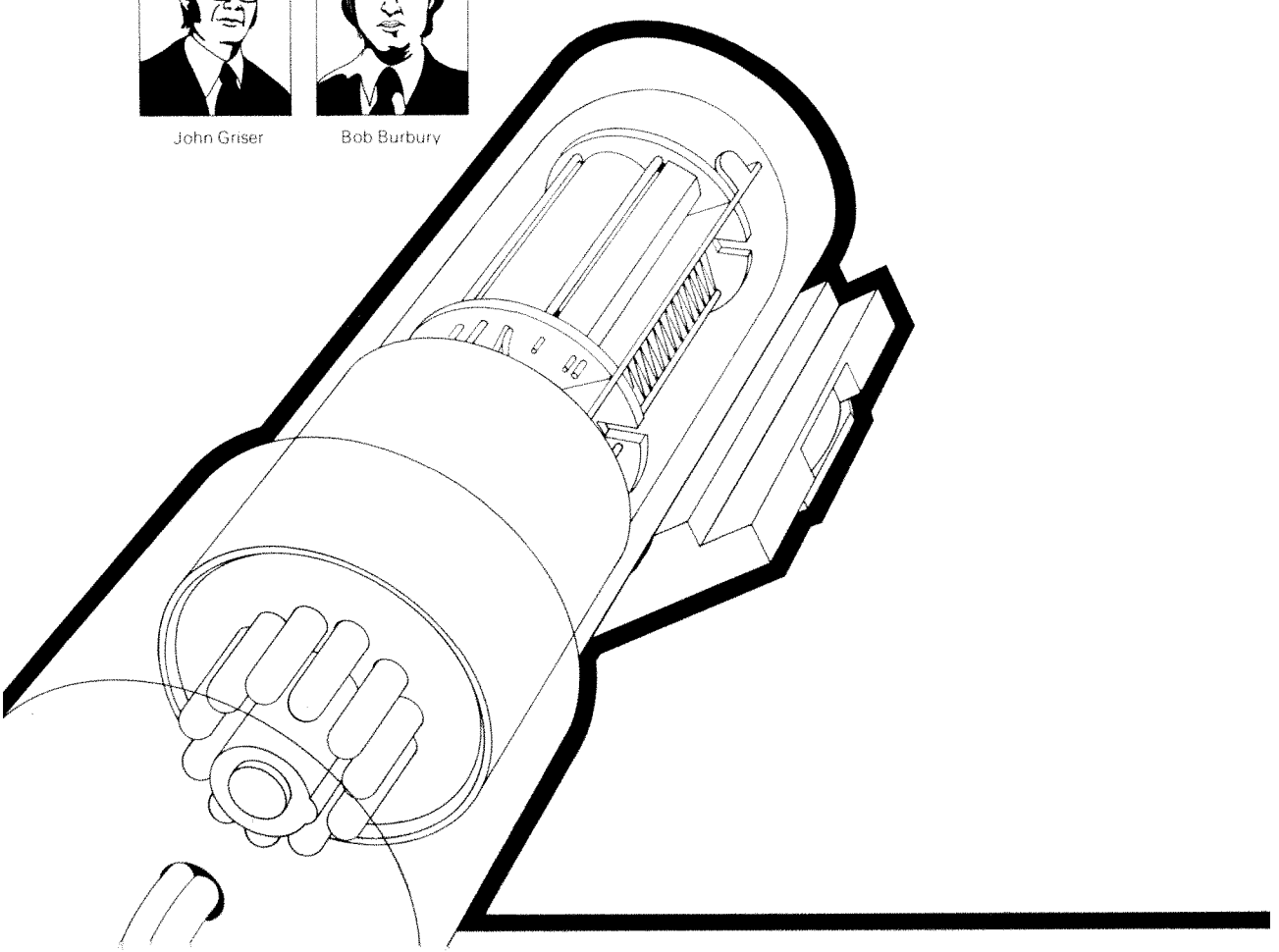
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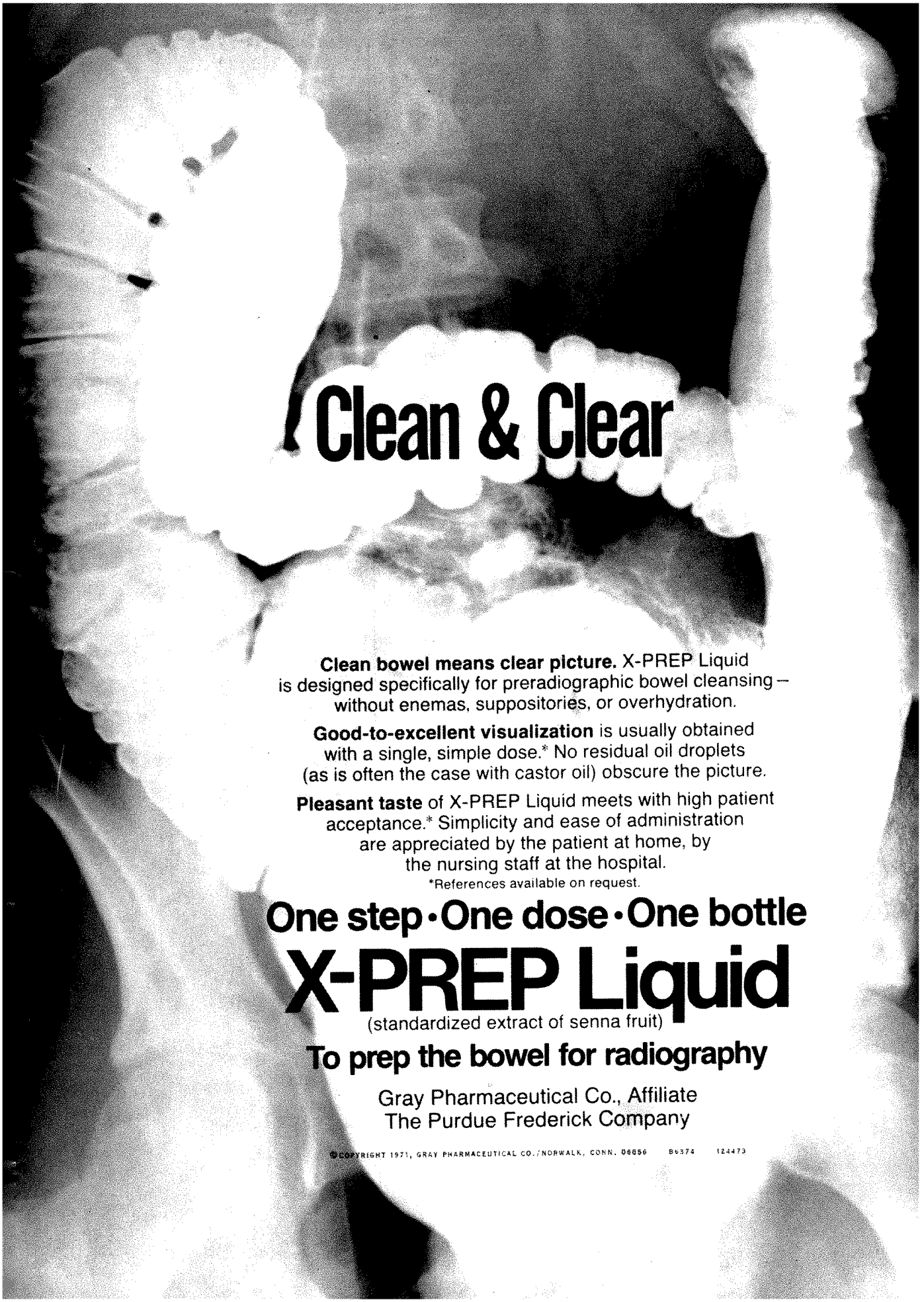
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By GILBERT H. FLETCHER, M.D., Head of Department of Radiotherapy, The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston. 39 Contributors. About 816 pages, 7″ × 10″. 427 illustrations. *New (2nd) Edition. Ready Soon. 1973.* (ISBN: 0-8121-0438-2).

The text has been expanded considerably, with several new chapters added to the extensively revised, updated material that made the first edition so successful. Radiotherapists and general radiologists practicing radiotherapy will find much of interest in this book.

ECHOCARDIOGRAPHY

By HARVEY FEIGENBAUM, M.D., Indiana University-Purdue University at Indianapolis School of Medicine. xiii plus 239 pages, 7″ × 10″. 193 illustrations. *1972.* $11.00. (ISBN: 0-8121-0406-4).

MEDICAL RADIATION BIOLOGY

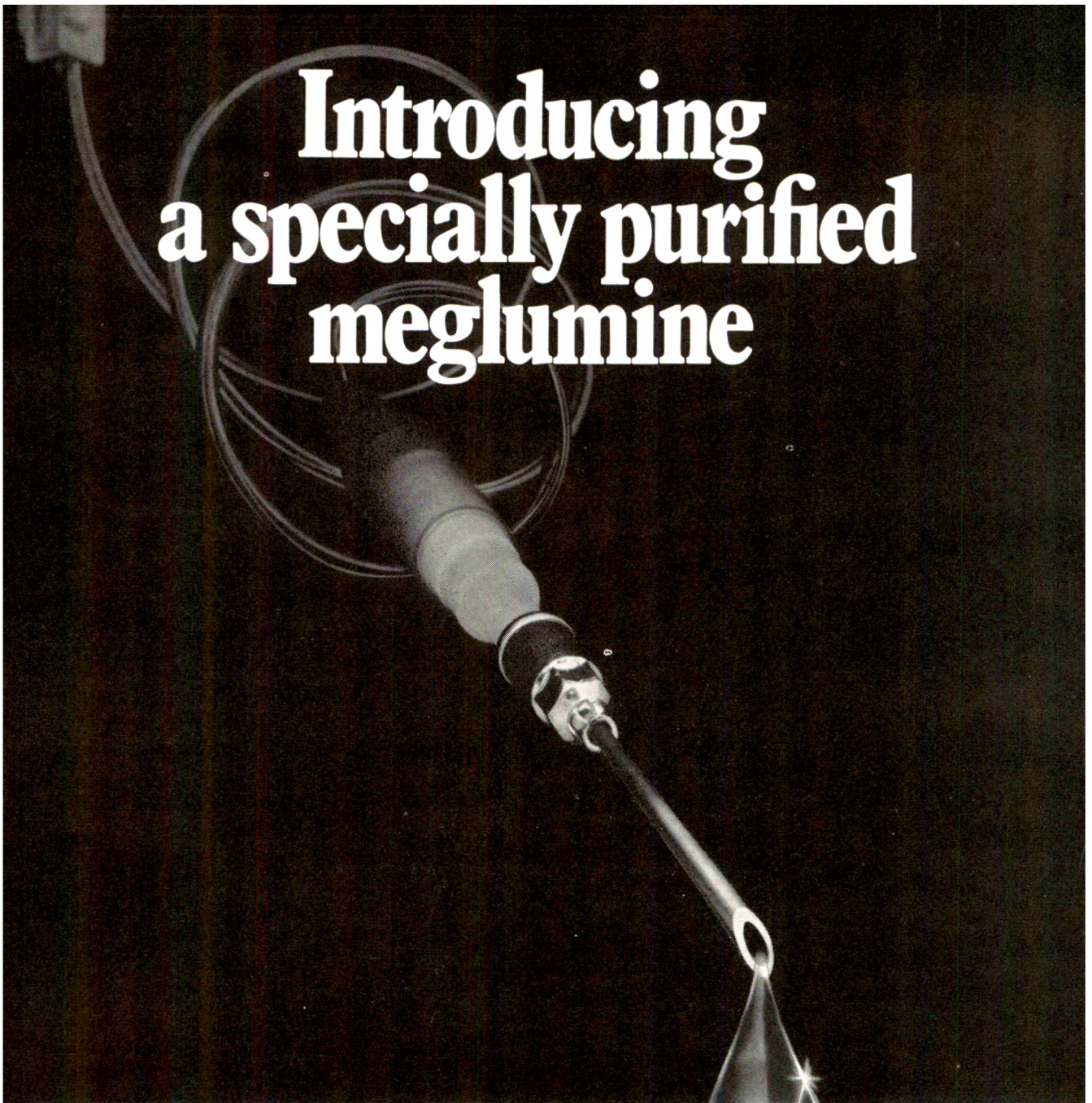
By DONALD J. PIZZARELLO, Ph.D., New York University Medical Center, New York; and RICHARD L. WITCOFSKI, Ph.D., The Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina. xi plus 111 pages, 7″ × 10″. Illustrated 1972. $9.00. (ISBN: 0-8121-0355-6).

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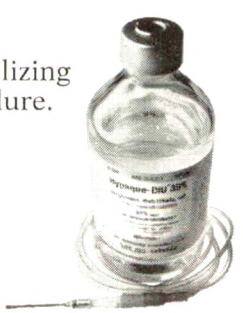
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Action: Hypaque-DIU 30 per cent is a diagnostic radiopaque medium which provides visualization of internal structures.

Indication: Hypaque-DIU 30 per cent is indicated for infusion urography.

Warnings: *Usage in Pregnancy.* No teratogenic effects attributable to a 60 per cent solution of Hypaque meglumine have been observed in reproduction studies in animals. However, before administration of Hypaque-DIU 30 per cent to women of child-bearing potential, the benefit to the patient should be carefully weighed against the possible risk to the fetus. In addition, most authorities consider elective contrast radiography of the abdomen contraindicated during pregnancy.

Serious or fatal reactions have been associated with the administration of radiopaque media. It is of utmost importance that a course of action be carefully planned in advance for the immediate treatment of serious reactions, and that adequate and appropriate facilities be readily available in case a severe reaction occurs.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravascularly.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake and direct thyroxine assays, are not affected.

Precautions: Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered. **Diphenhydramine hydrochloride (i.e., Benadryl®), however, should not be mixed with Hypaque-DIU 30 per cent since it may cause precipitation.** The intravenous injection of a test dose of 0.5 to 1 ml. of the contrast agent before infusion of the full dose has been employed in an attempt to predict severe or fatal adverse reactions. The preponderance of recent scientific literature, however, now demonstrates that this provocative test procedure is not reliably predictive of serious or fatal reactions. Severe reactions and fatalities have occurred with the test dose alone, with the full dose after a nonreactive test dose, and with or without a history of allergy. No conclusive relationship between severe or fatal reactions and antigen-antibody reactions or other manifestations of allergy has been established. A history of allergy may be useful in predicting reactions of a mild or intermediate nature. Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations, or of sensitivity to iodine.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure, it is essential to omit preparatory dehydration, to administer fluids, and attempt to alkalinize the urine.

Immediate adverse reactions to infusion urography have not been reported to occur at a higher frequency or with greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Infusion urography imposes a sudden osmotic load. This should be considered in patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, there is a possibility of the development of congestive heart failure hours after the procedure.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating infusion urography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion urography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

Adverse Reactions: Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

The following have been reported after administration of diatrizoates and other iodinated contrast media. *Cardiovascular reactions:* vein cramp and thrombophlebitis following intravenous injection, rare cases of cardiac arrhythmias, hypertension, hypotension and shock, and cardiac arrest. *Allergic reactions:* asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria, and rarely, anaphylactic reactions. Signs and symptoms related to the *respiratory system:* pulmonary or laryngeal edema, bronchospasm; or to the *nervous system:* restlessness, convulsions. *Other reactions:* flushing, pain, warmth, nausea, vomiting, anxiety, headache, dizziness, hematoma, and ecchymosis. Occasionally, transient proteinuria, and rarely, oliguria or anuria. Rarely, immediate or delayed rigors can occur, sometimes accompanied by hyperpyrexia. Infrequently, "iodism" (salivary gland swelling) from organic iodinated compounds appears two days after exposure and subsides by the sixth day.

Dosage and Administration: The recommended dose for infusion urography is calculated on the basis of 2 ml. of Hypaque-DIU 30 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimal results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

The solution is infused over a period of three to ten minutes. Radiographs are usually taken at 10, 20, and 30 minutes from the beginning of the infusion. Earlier films may be obtained for the evaluation of hypertension. Nephrograms are usually obtained at the end of the infusion, and voiding cystourethrograms are usually obtained 30 minutes after the beginning of the infusion.

How Supplied: Calibrated bottles of 300 ml., rubber stoppered, packed with and without sterile disposable intravenous infusion sets.

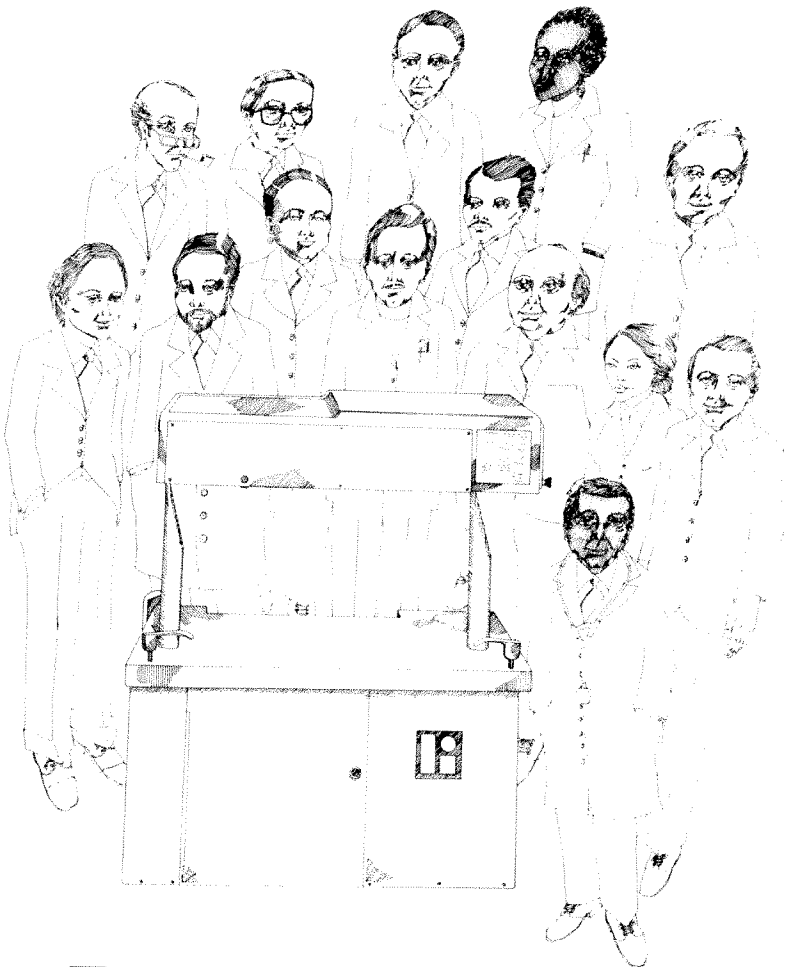
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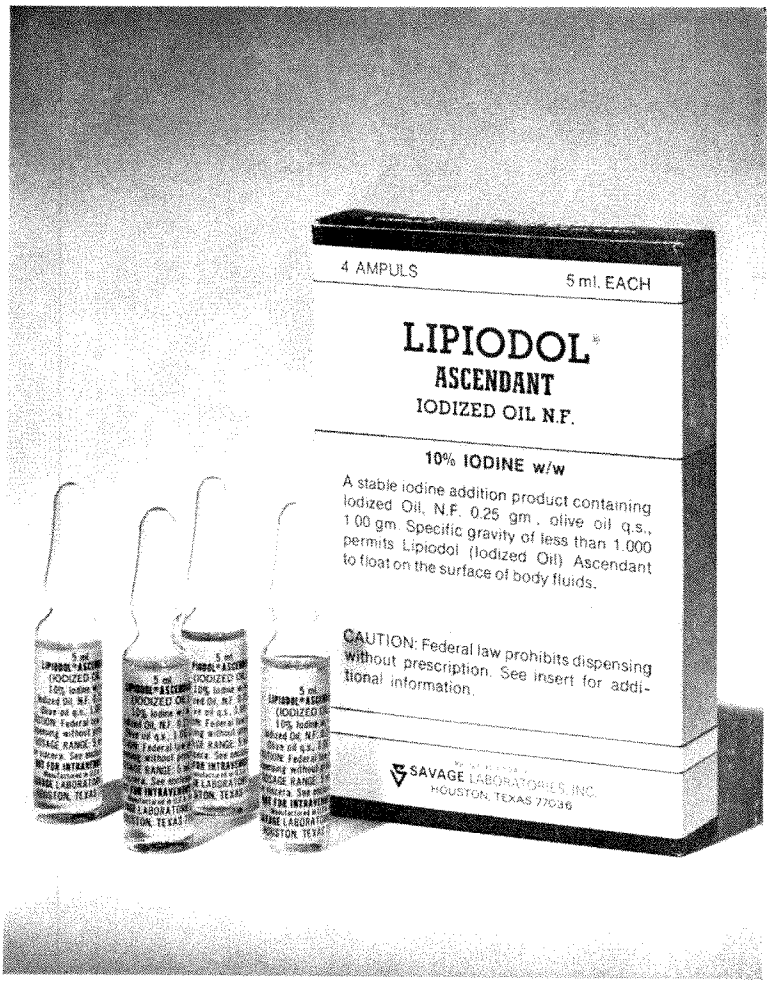
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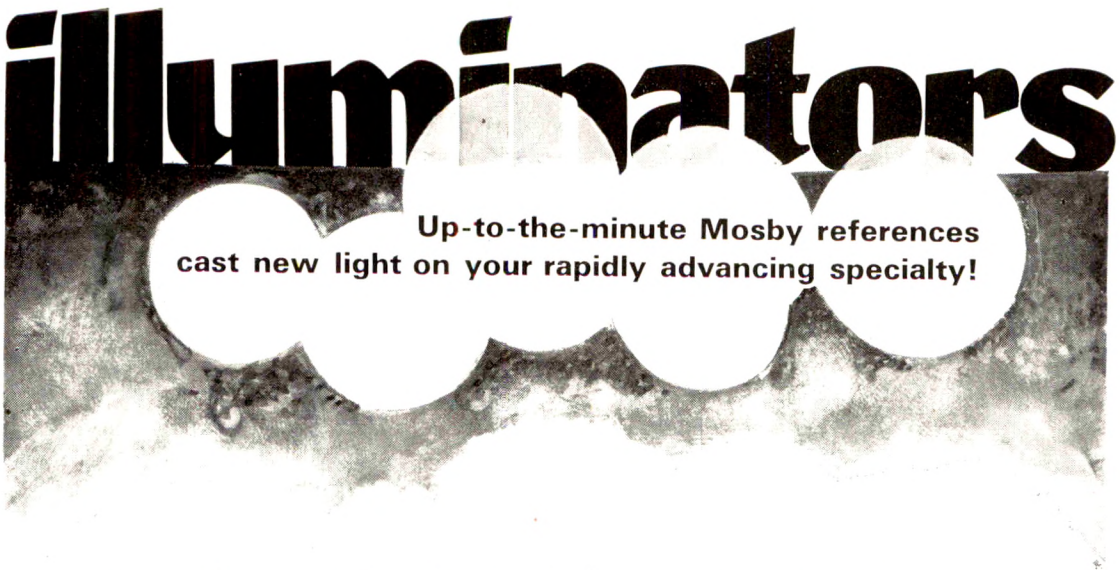
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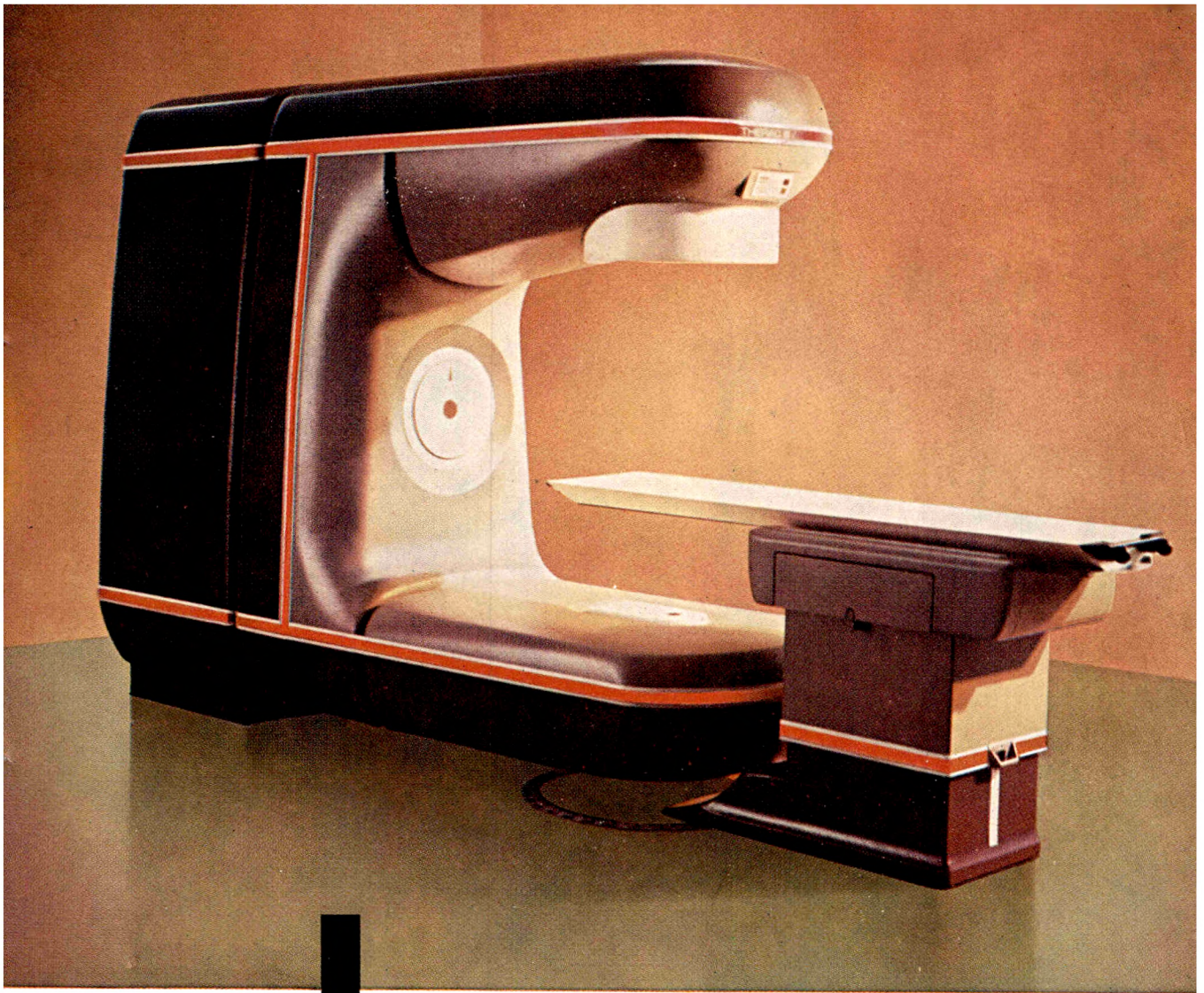
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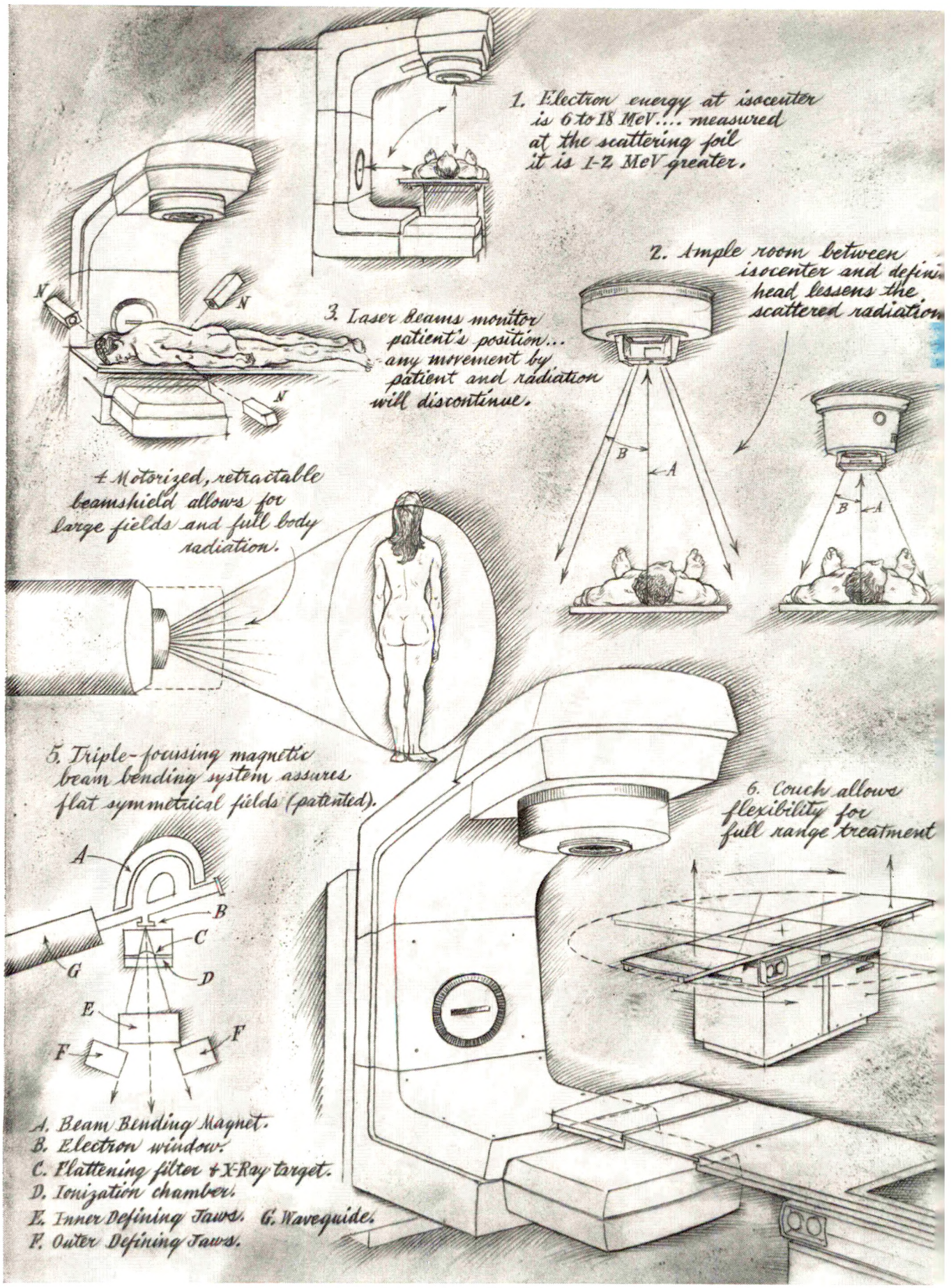
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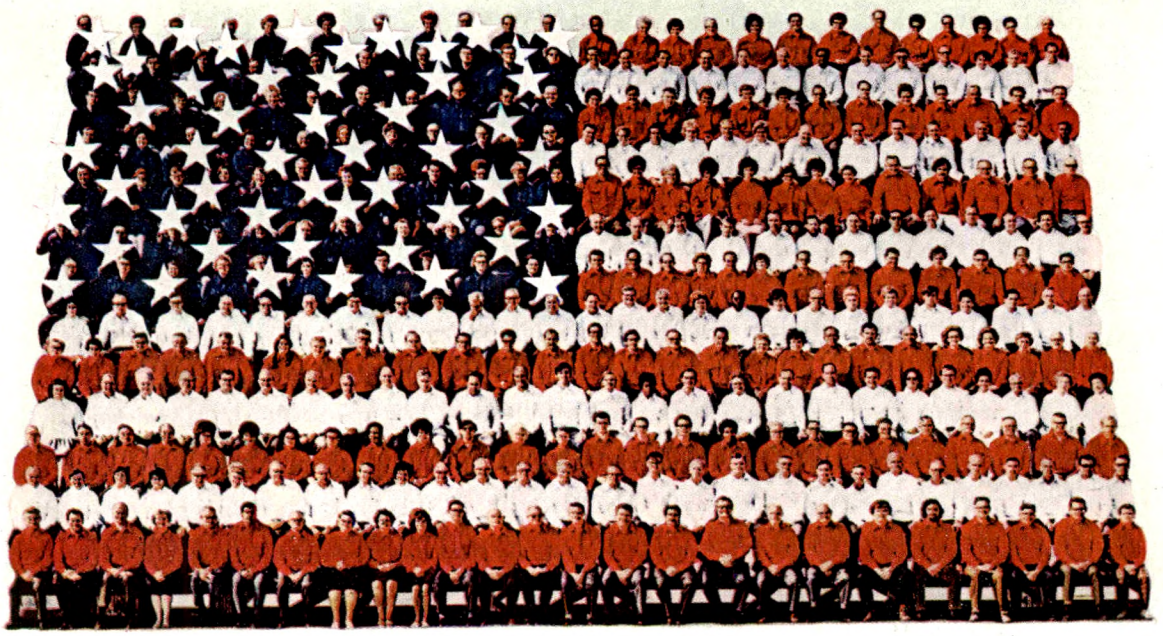
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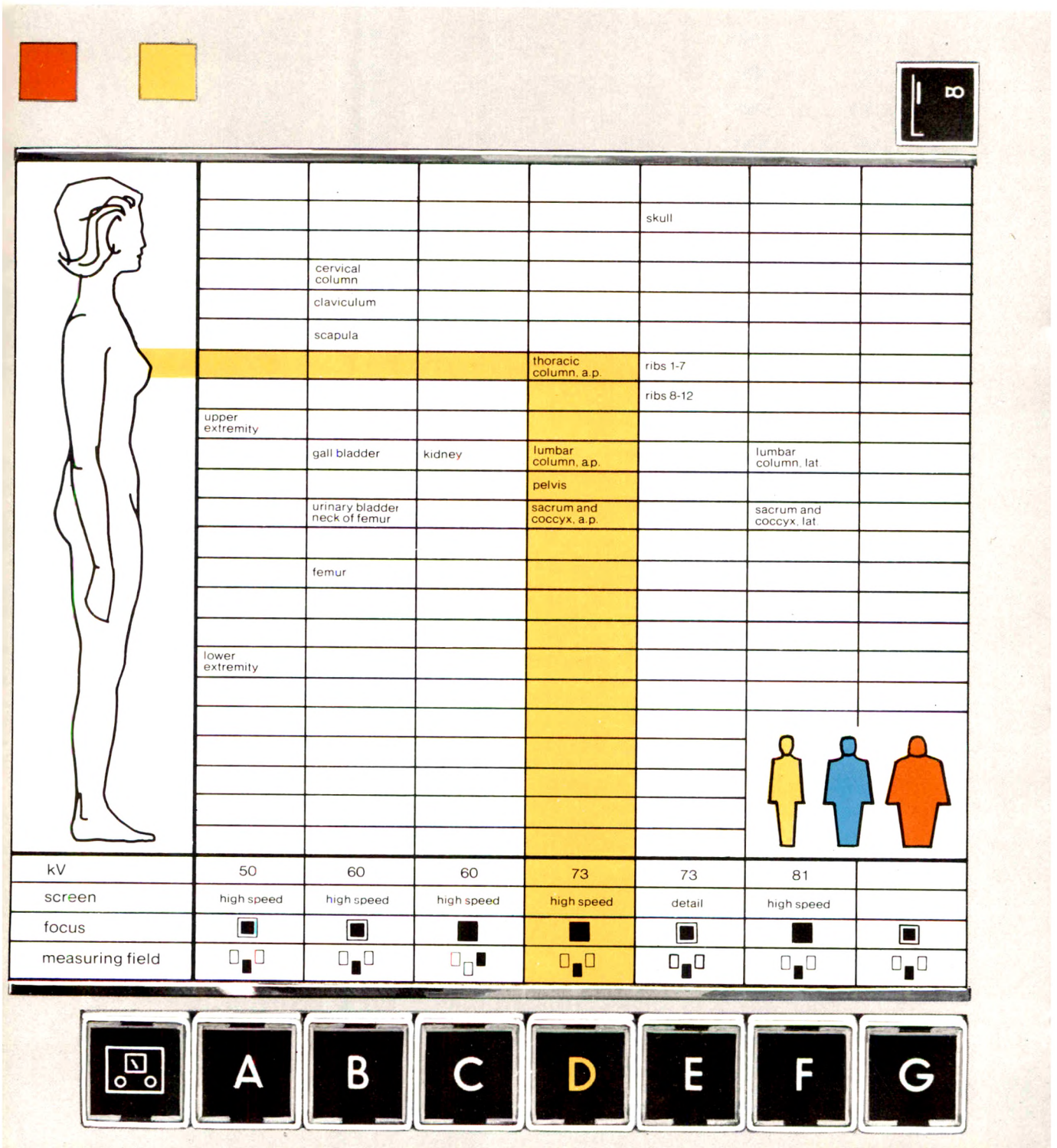
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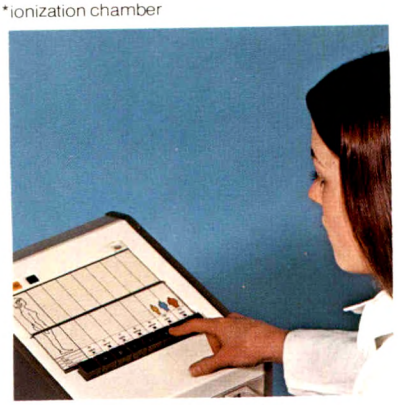
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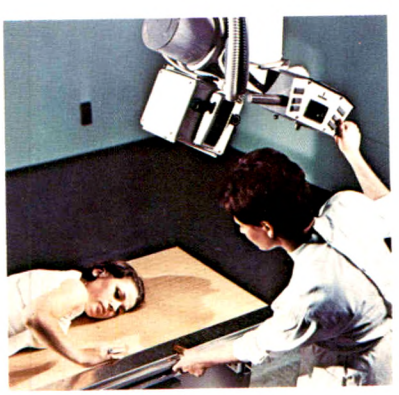
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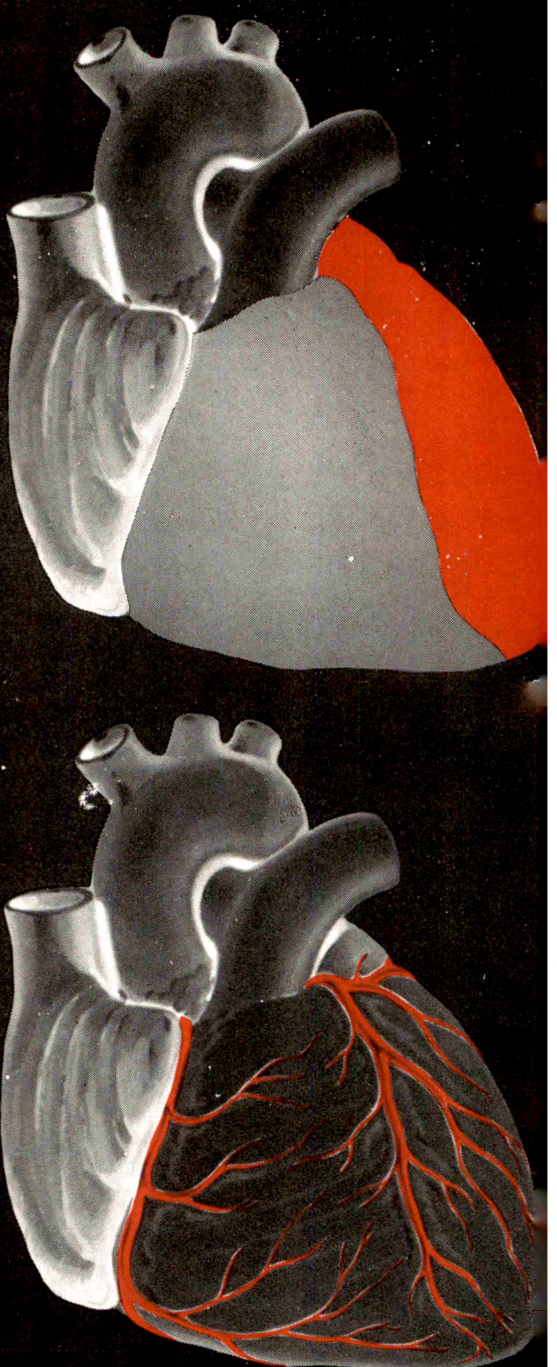
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In *excretion urography*, adequate visualiza- tion may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be haz- ardous; this also applies to *pediatric angio- cardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography*, hypotension or moderate de- creases in blood pressure seem to occur fre- quently with intra-arterial (brachial) injections; this is transient and usually requires no treat- ment. Monitor blood pressure during the im- mediate 10 minutes after injection. It is recom- mended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and ade- quate facilities for immediate resuscitation and cardioversion.

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Adverse reactions as a consequence of *ex- cretion urography* include cardiac arrest, ven- tricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to gen- eralized vasodilation. Risks of *aortography* pro- cedures include injury to aorta and neighbor- ing organs, pleural puncture, renal damage (including infarction and acute tubular necrosis with oliguria and anuria), accidental selective filling of right renal artery during translumbar procedure in presence of preexistent renal dis- ease, retroperitoneal hemorrhage from trans- lumbar approach, spinal cord injury and pathology associated with syndrome of trans- verse myelitis, generalized petechiae, and death following hypotension, arrhythmia, and anaphylactoid reactions. In *pediatric angio- cardiography*, arrhythmia and death have oc- curred. During *peripheral arteriography*, hemor- rhage from puncture site, thrombosis of the vessel, and brachial plexus palsy (following axillary artery injection) have occurred. During *selective coronary arteriography* and *selective coronary arteriography with left ventriculog- raphy*, transient ECG changes (most patients); transient arrhythmias (infrequent); ventricular fibrillation (from manipulation of catheter or administration of medium); hypotension; chest pain; myocardial infarction; transient elevation of creatinine phosphokinase (occurred in about 30% of patients tested); fatalities have been reported; hemorrhage, thrombosis, pseudo- aneurysms at puncture site, dislogment of arteriosclerotic plaques, dissection of coronary vessels, and transient sinus arrest have oc- curred due to the procedure.

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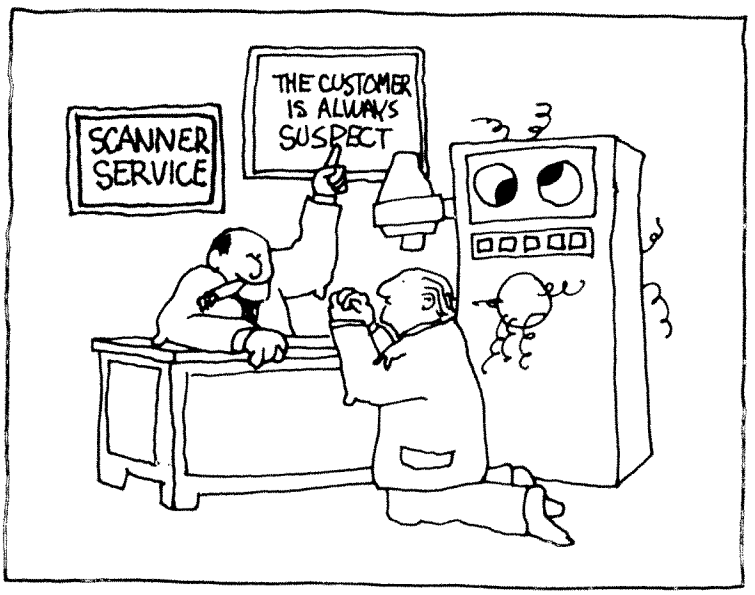
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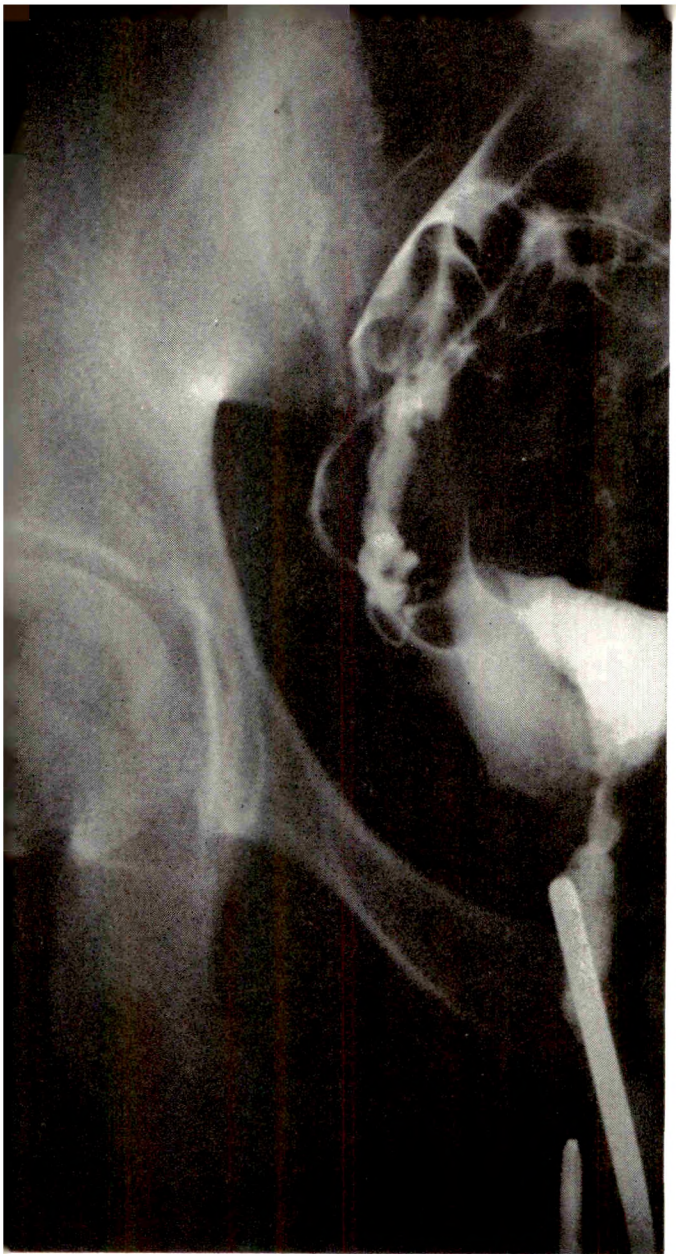
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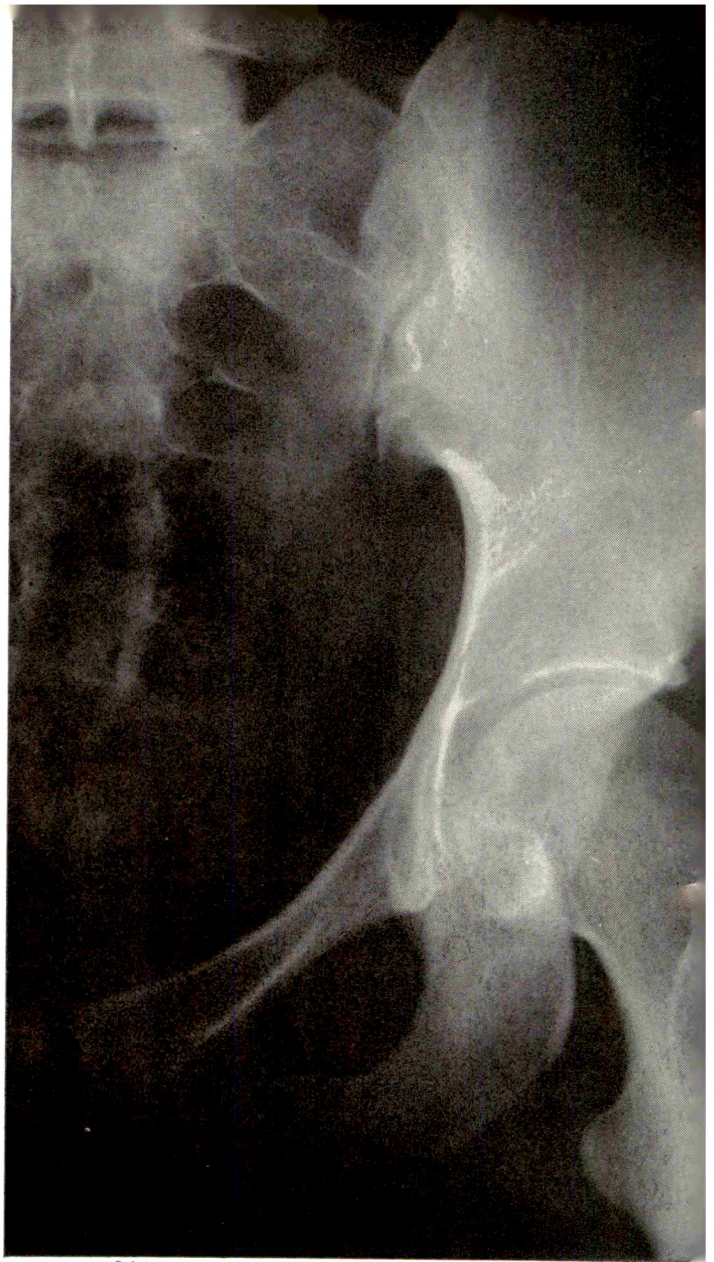
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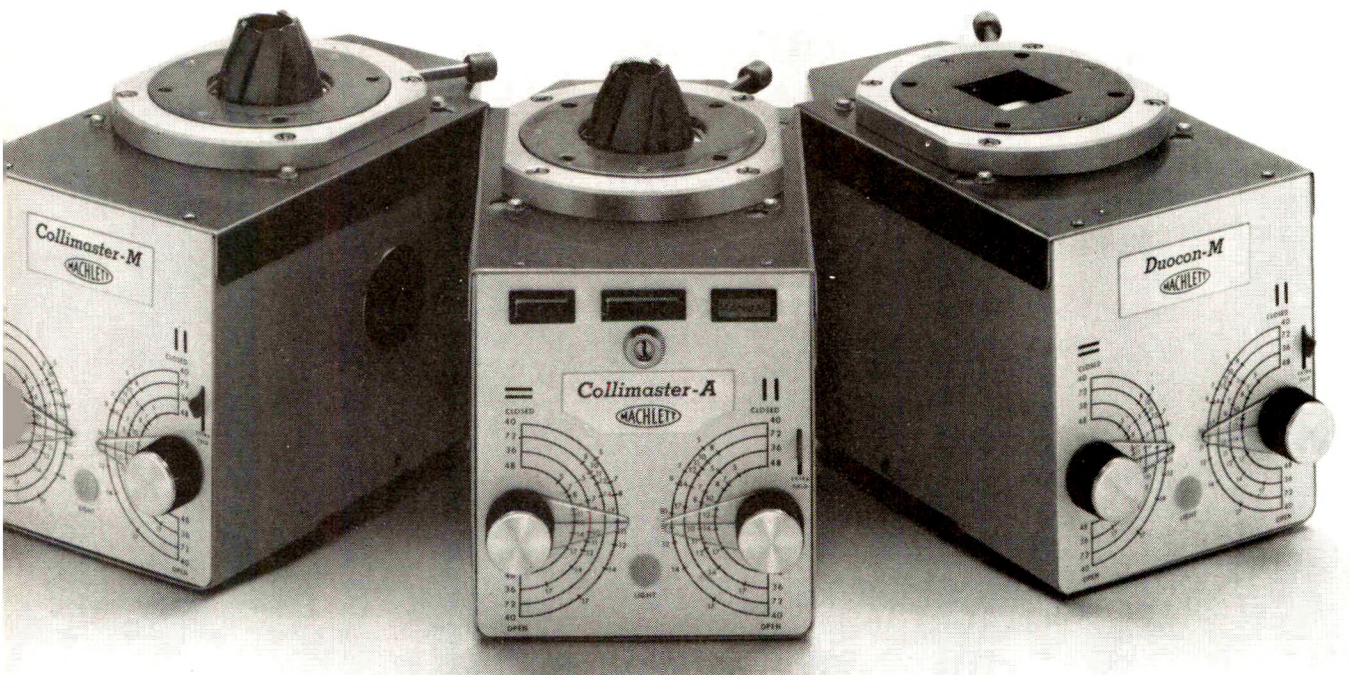
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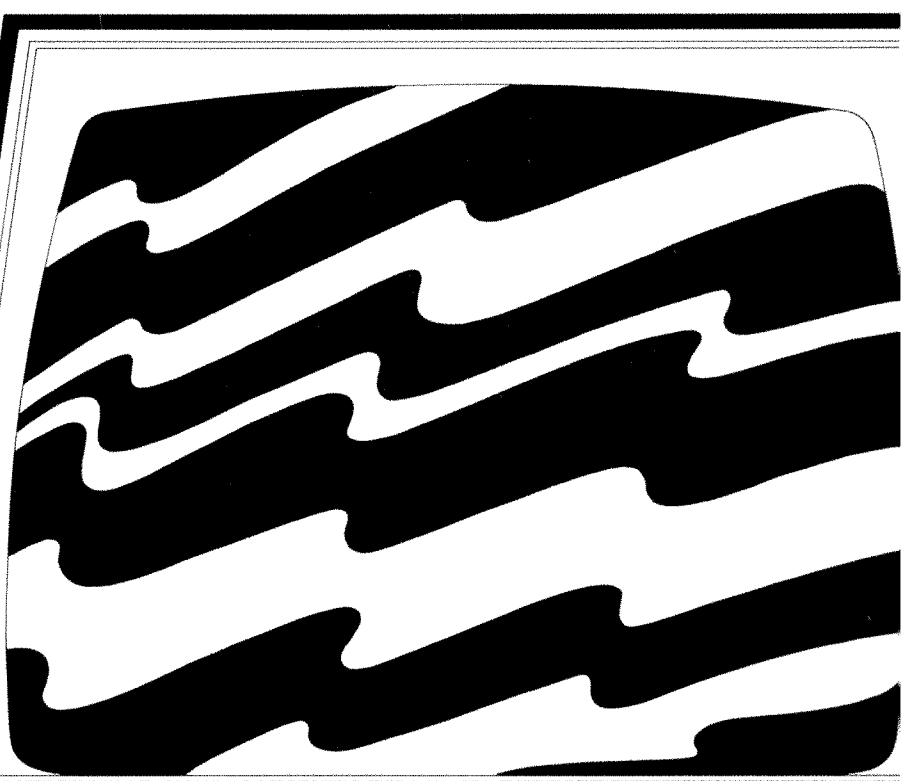
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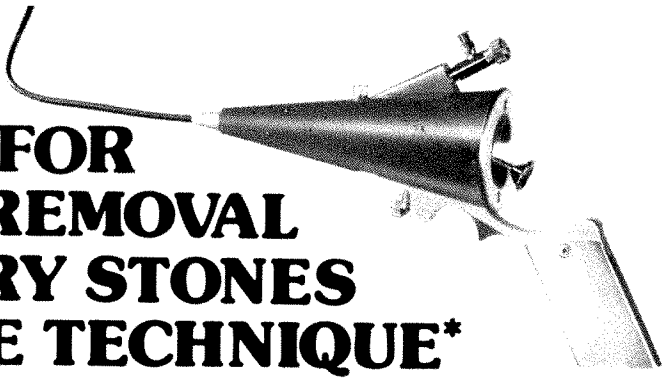
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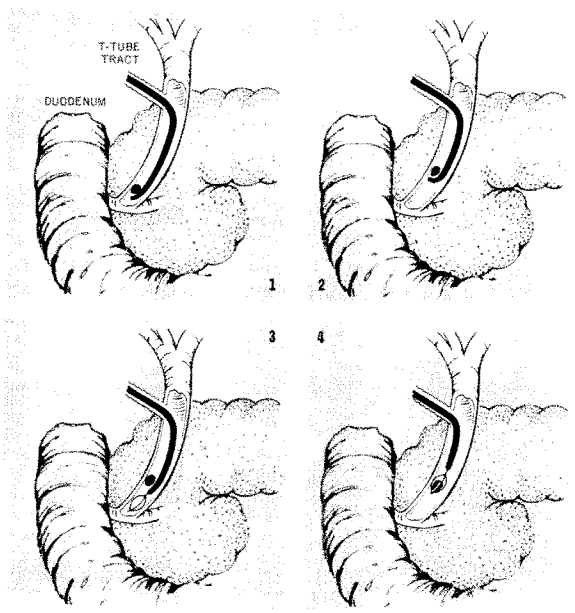
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*REFERENCE

Burhenne, H. J., Non-operative Retained Biliary Tract Stone Extraction — A New Roentgenologic Technique, AMER. J. ROENTGENOLOGY, Vol. CXVII, No. 2, pp. 388-399, February 1973.

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CORONARY COLLATERAL CIRCULATION*

By DAVID C. LEVIN, M.D.,† MICHAEL KAUFF, M.D.,‡ *and* HAROLD A. BALTAXE, M.D.
NEW YORK, NEW YORK

CORONARY arteriography in patients with severe obstructing coronary atherosclerosis will frequently demonstrate the presence of varying degrees of collateral circulation. Although there is some controversy over the clinical significance of this collateral circulation,[6] our experience suggests that it plays a key role in preserving myocardial viability.

Collateral arteries within the heart probably do not develop *de novo*. They are in actuality anastomotic channels which are present in virtually all normal hearts,[2,7] but which are not utilized until the need arises. These anastomotic channels have been carefully studied in pathologic specimens by Baroldi and Scomazzoni,[2] who describe both "homocoronary" anastomoses, connecting various branches of the same coronary artery, and "intercoronary" anastomoses, connecting branches of the 3 major coronary arteries. These anastomoses are generally less than 200 micra in diameter, but may occasionally be as large as 350 micra. In spite of this considerable size, however, they possess only a thin endothelial lining. In healthy hearts, they cannot be demonstrated arteriographically because they are nonfunctional.[5] However, if an obstructing lesion develops in a major coronary artery, a pressure gradient is created between the distal segment of that artery and either the proximal segment of the same vessel or a nearby unobstructed vessel. Blood will then begin to flow from the high pressure segments to the low pressure segment through these pre-existing anastomotic channels. The latter, because of their thin walls, become elongated and tortuous and may dilate up to 1 and even 2 mm. in diameter. At this stage, they have become collaterals which are readily visualized on coronary arteriograms. This sequence of events generally does not take place unless severe coronary artery narrowing is present with at least a 90 per cent reduction in diameter of the lumen.[5,12] It is very rare to be able to arteriographically demonstrate collateral circulation when the degree of stenosis is less severe. In addition to the hemodynamic factors discussed above, there may also be metabolic stimuli

* An exhibit based upon this material was shown at the Seventy-third Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 3–6, 1972.

From the Department of Radiology, The New York Hospital-Cornell Medical Center, New York, New York.
† Current Address: Department of Radiology, State University of New York, Downstate Medical Center, Brooklyn, New York.
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to the development of collateral circulation,[3] but these are not well understood at present.

Coronary collaterals do not meander randomly through the myocardium, as do those of the thigh and knee in arterial occlusions of the lower extremities. Instead, a number of definable pathways exist. Thorough knowledge of these pathways is of more than purely academic interest. First of all, one must obviously be able to differentiate normal from pathologic circulation. Second, because of the variability of coronary artery anatomy, obstructions at the origins of small branches often cannot be detected except by the demonstration of collateral flow into the distal portions of these vessels. Finally, the presence or absence of collateral flow into distal segments of obstructed major coronary arteries may affect the advisability of surgical therapy or the nature of the operation to be performed.[10]

Previous publications have only partially catalogued the various collateral pathways.[5,11] The purpose of this paper is to briefly describe all of the important pathways seen in a large group of patients with coronary artery disease.

MATERIAL AND METHOD

Selective coronary arteriograms of 200 patients with significant coronary artery disease comprise this series. All patients had at least 50 per cent stenosis of at least 1 major coronary artery. Patients with insignificant coronary lesions were not included, nor were those with valvular heart disease. The studies were performed utilizing the percutaneous femoral approach and the catheters and techniques described by Judkins,[8] and Amplatz *et al.*[1] All studies were recorded on 35 mm. cine film and most were also recorded on serial roentgenograms exposed at 2 films per second for 5 seconds.

The arteriograms were carefully analyzed for the presence of collateral circulation. In the following sections, the major collateral pathways are briefly described. The number in parentheses after each listing indicates the number of cases in the series in which that particular pathway was visualized. In many cases 2 or even 3 collateral pathways were present for a given obstructive lesion. Each listed pathway is accompanied by a correspondingly lettered diagram in Figure 1, and many are also illustrated angiographically in Figures 2–14. For the sake of simplicity, the diagrams assume right coronary dominance. In this anatomic arrangement, present in 90 per cent of all patients, the right coronary artery gives rise to both the posterior descending and posterior left ventricular arteries. The following abbreviations are used: RC = right coronary artery; LC = left coronary artery; AM = acute marginal branch of the right coronary artery; OM = obtuse marginal branch of the left circumflex artery; PD = posterior descending branch of the right coronary artery; PLV = posterior left ventricular branch of the right coronary artery; LAD = left anterior descending artery.

COLLATERAL PATHWAYS IN OBSTRUCTIONS OF THE RIGHT CORONARY ARTERY

Among the 200 patients in this series, 33 were found to have moderate stenoses (between 50–90 per cent) of the RC artery. Collaterals were not present with any of these lesions; 105 patients had severe stenoses (greater than 90 per cent) or complete obstructions of the RC artery. Collateral circulation was seen circumventing the lesions in 74 of these patients.

1. HOMOCORONARY COLLATERALS

A. Either high acute marginal branch or conus branch to low acute marginal branch (9 cases). The conus branch arises either from the first part of the RC artery or directly from a separate aortic ostium and passes forward to supply the infundibular portion of the right ventricle. The AM branch or branches arise slightly further down the RC artery and pass forward to supply the free wall of the right ventricle. When the RC is obstructed between the

origins of any of these branches, collateral connections between them may serve to bypass the obstruction (Fig. 1*A*; and 2).

B. Kugel's artery (9 cases). This artery, first described in 1927 by Kugel,[9] originates from the proximal RC artery (less commonly from the proximal LC artery), then passes down the anterior margin of the atrial septum to connect with the AV node artery. The latter in turn passes cephalad from the distal RC artery near the crux of the heart (the junction of the

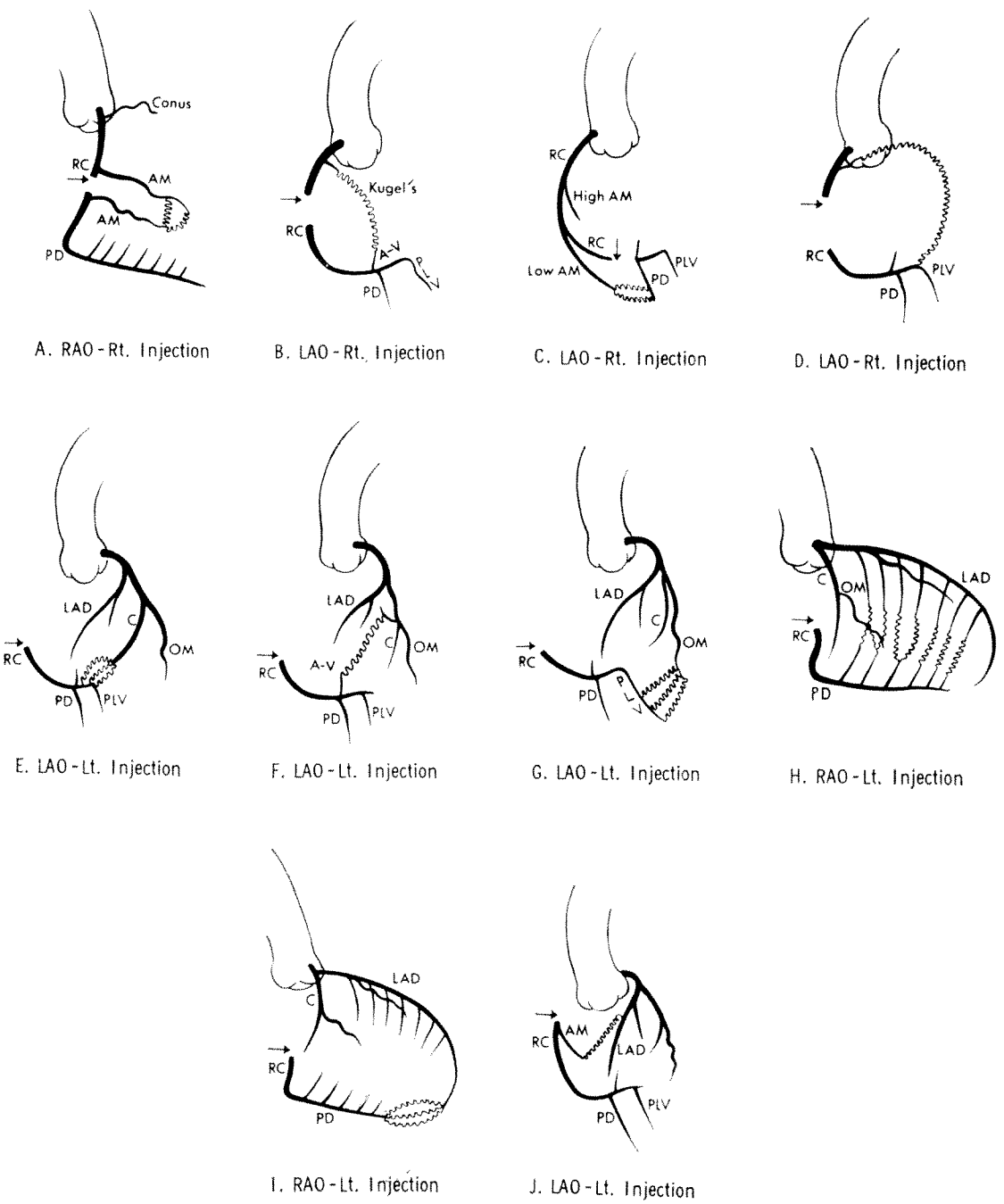


FIG. 1. (*A–J*) Collateral pathways in right coronary obstruction.

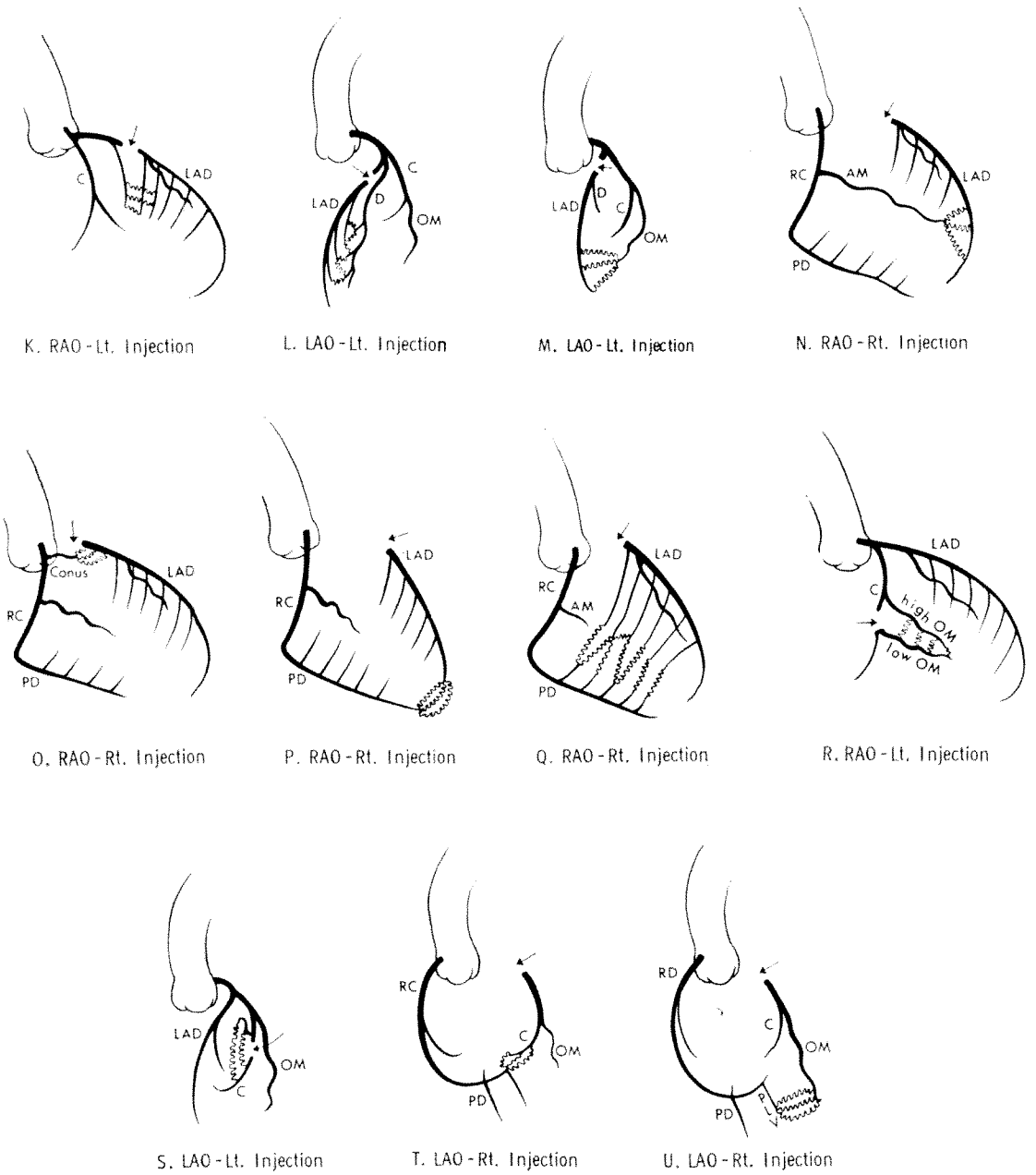


FIG. 1. (*K–Q*) Collateral pathways in left anterior descending obstruction. (*R–U*) Collateral pathways in left circumflex obstruction.

Small arrows indicate the point of obstruction of the coronary artery. RAO = right anterior oblique projection. LAO = left anterior oblique projection. RC = right coronary artery. AM = acute marginal branch. PD = posterior descending branch. PLV = posterior left ventricular branch. AV = branch to the AV node. LAD = left anterior descending artery. D = diagonal branch. C = circumflex artery. OM = obtuse marginal branch.

right and left atrioventricular sulci and the posterior interventricular sulcus). Kugel's artery may thus bypass a RC occlusion anywhere between the proximal segment and the crux (Fig. 1*B*; and 3).

C. Distal acute marginal branch to pos-

terior descending branch (6 cases). Low AM branches or posterior right ventricular branches frequently arise from the RC artery and pass along the diaphragmatic surface of the right ventricle. If the RC artery becomes occluded distal to these branches, collateral circulation may pass beneath the right ventricle from the AM branch to the PD branch (Fig. 1*C*).

D. SA node artery to left atrial branch to distal right coronary artery (2 cases). RC obstruction may be circumvented by this long collateral pathway in which flow passes from a right atrial branch of the proximal RC artery (usually the artery to the SA node) to a left atrial branch of the circumflex artery. This in turn connects with the terminal segment of the RC artery (Fig. 1*D*; and 4).

II. INTERCORONARY COLLATERALS FROM THE LEFT CIRCUMFLEX ARTERY

E. Either distal left circumflex or left atrial circumflex artery to distal right coronary artery (24 cases). Dominant RC arteries terminate by giving rise to PLV branches which supply the posterior or diaphragmatic surface of the left ventricle. The left circumflex artery terminates along the left atrioventricular sulcus, at or near the diaphragmatic surface of the left ventricle. In the presence of RC obstruc-

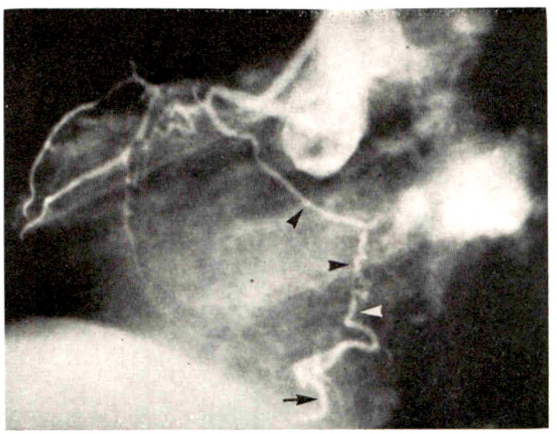


Fig. 3. Right coronary arteriogram, left anterior oblique projection. The right coronary artery is obstructed. There is collateral flow through Kugel's artery (black arrowheads) to the AV node artery (white arrowhead) and ultimately to the posterior descending artery (black arrow).

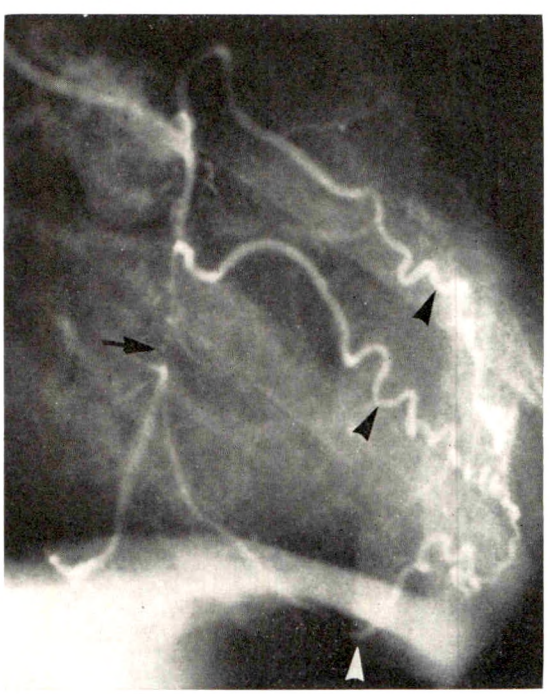


Fig. 2. Right coronary arteriogram, right anterior oblique projection. Collaterals pass from high acute marginal branches (black arrowheads) to a low acute marginal branch (white arrowhead). Note severe right coronary narrowing (black arrow).

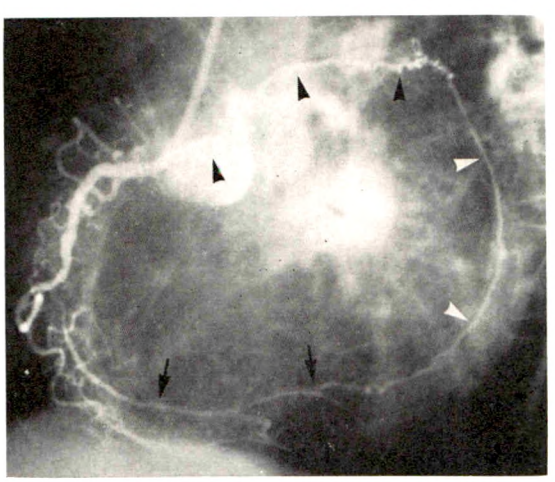


Fig. 4. Right coronary arteriogram, left anterior oblique projection. Collateral flow passes from the SA node artery (black arrowheads) to a left atrial branch (white arrowheads) and then to the distal portion (black arrows) of the obstructed right coronary artery.

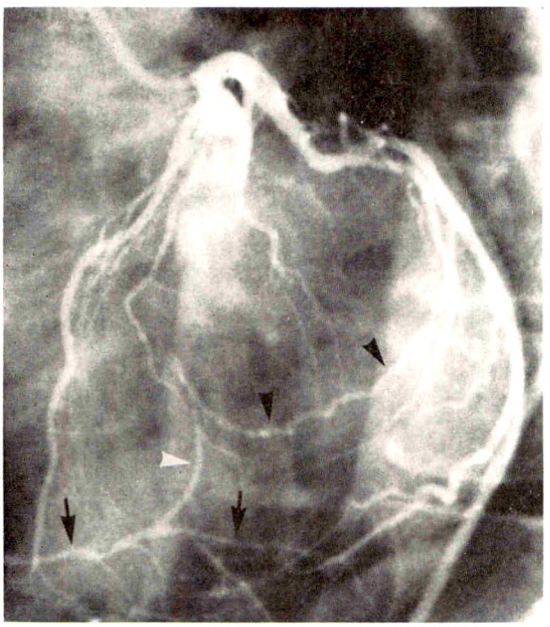


FIG. 5. Left coronary arteriogram, left anterior oblique projection. There is collateral circulation from the atrial circumflex branch (black arrowheads) to the AV node artery (white arrowhead) with subsequent opacification of the entire distal right coronary artery (black arrows).

tion, intercoronary collaterals may pass from the distal circumflex artery or its left atrial circumflex branch to the nearby distal RC artery (Fig. 1*E*).

F. Either distal left circumflex or left atrial circumflex artery to AV node artery (6 cases). This is quite similar to the preceding pathway. Here the collateral flow passes from the distal circumflex or left atrial circumflex arteries indirectly to the distal RC artery by way of the AV node artery (Fig. 1*F*; and 5).

G. Obtuse marginal or diagonal artery to posterior left ventricular artery (17 cases). Virtually every patient has at least 1 large OM branch arising from the circumflex artery and/or 1 large diagonal branch arising from the LAD artery. These OM and diagonal branches supply the anterolateral aspect of the free wall of the left ventricle. Their terminal ramifications lie close to those of the PVL branches of the RC artery. Collateral flow from the OM or, less commonly, the diagonal branches may opacify the distal tree of a blocked RC artery *via* its PLV branches (Fig. 1*G*; and 6).

III. Intercoronary Collaterals from the Left Anterior Descending Artery

H. Left anterior descending artery to posterior descending artery through the interventricular septum (28 cases). The septum contains numerous anastomotic connections between the septal branches of the LAD and PD arteries. These constitute the most common intercoronary collateral pathway to the PD artery in cases of RC obstruction (Fig. 1*H*; and 7).

I. Left anterior descending artery to posterior descending artery *via* the cardiac apex (9 cases). The LAD artery generally passes completely around the apex of the heart and terminates at or near the posterior interventricular sulcus. The PD artery passes along this sulcus from the crux to its termination near the apex. If the RC

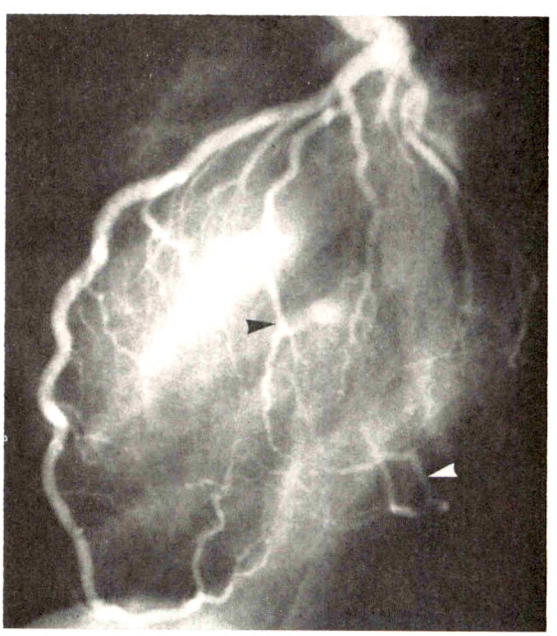


FIG. 6. Left coronary arteriogram, left anterior oblique projection. Collateral flow passes from the diagonal branch (black arrowhead) of the left anterior descending artery to a posterior left ventricular branch (white arrowhead) of an obstructed right coronary artery.

artery is obstructed, collateral flow may pass from the distal LAD artery through anastomotic connections at the cardiac apex to the PD artery (Fig. 1*I*; and 8).

J. Left anterior descending artery to acute marginal artery (2 cases). Small muscular branches from the LAD artery can occasionally be seen passing over the anterior right ventricular wall. One of the rare collateral pathways in proximal RV obstruction is the connection between these branches of the LAD artery and the AM branch of the RC artery (Fig. 1*J*).

COLLATERAL PATHWAYS IN OBSTRUCTION OF THE LEFT ANTERIOR DESCENDING ARTERY

Forty-six patients in this series had moderate stenoses of the LAD artery; no collateral circulation was seen in this group. One hundred and twenty-seven patients had severe stenoses or complete obstructions of this vessel, and 70 of them showed collateral circulation.

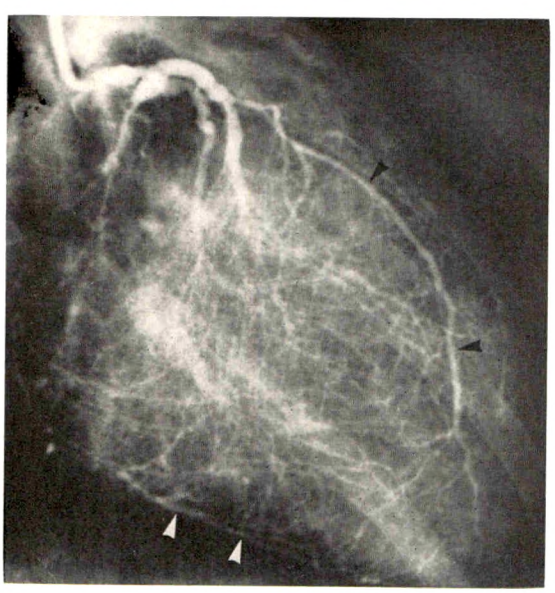


Fig. 7. Left coronary arteriogram, right anterior oblique projection. There is an extensive collateral network within the interventricular septum connecting the left anterior descending artery (black arrowheads) with the posterior descending branch (white arrowheads) of an obstructed right coronary artery.

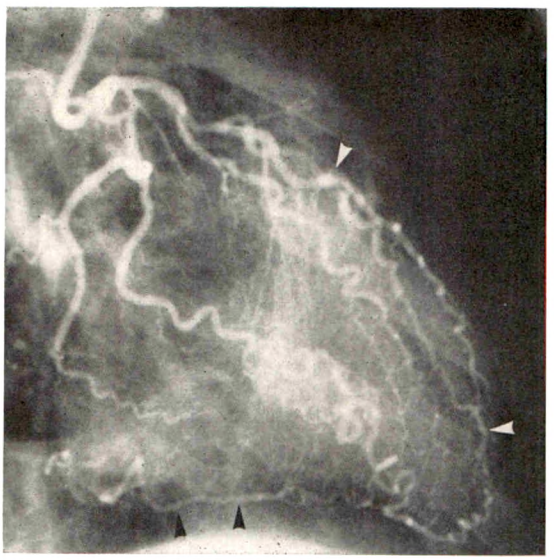


Fig. 8. Left coronary arteriogram, right anterior oblique projection. Collateral flow passes around the cardiac apex from the left anterior descending artery (white arrowheads) to the posterior descending branch (black arrowheads) of an obstructed right coronary artery.

1. HOMOCORONARY COLLATERALS

K. Interventricular septal collaterals bypassing left anterior descending artery obstructions (27 cases). The proximal septal branches of the LAD artery are often large and LAD obstructions frequently occur beyond their origin. In this situation, blood may pass down these proximal septal branches, through a network of collaterals in the septum and then into more distal septal branches which arise beyond the lesion. Retrograde flow within these distal septal branches in turn perfuses the distal LAD artery (Fig. 1*K*; and 9).

L. Diagonal branch to left anterior descending artery (6 cases). As mentioned earlier, there is generally at least 1 large diagonal branch which helps to supply the anterolateral aspect of the left ventricle. This vessel originates either from the LAD artery or directly from the LC artery bifurcation and often parallels the course of the LAD artery. If the LAD artery is obstructed beyond the origin of its diagonal branch, collateral circulation may pass

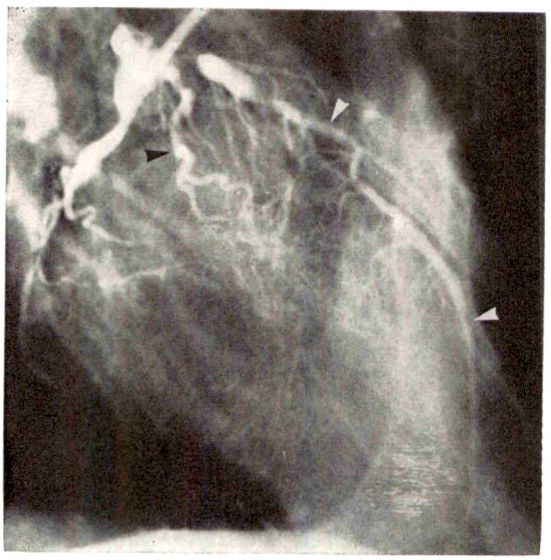


Fig. 9. Left coronary arteriogram, right anterior oblique projection. Note complete obstruction of the proximal left anterior descending artery. Collateral flow passes down the large first septal branch (black arrowhead), through a dense network of vessels in the interventricular septum and then fills the distal left anterior descending artery (white arrowheads).

from the latter to the former across the anterior surface of the left ventricle (Fig. 1*L*).

II. Intercoronary Collaterals from the Left Circumflex Artery

M. Obtuse marginal branch to distal left anterior descending artery (17 cases). This is the only important intercoronary collateral pathway from the circumflex artery to the LAD artery. Flow passes from the terminal branches of the OM artery around the anterolateral aspect of the left ventricle to the distal LAD artery or, occasionally, to 1 of its diagonal branches (Fig. 1*M*; and 10).

III. Intercoronary Collaterals from the Right Coronary Artery

N. Acute marginal artery to the left anterior descending artery (28 cases). Flow passes from the AM branch around the anterior aspect of the right ventricle to the distal LAD artery. As Paulin[11] has pointed out, there is also a closely related pathway from the AM branch through the moderator band to a septal branch of the LAD artery (Fig. 1*N*; and 11).

O. Conus branch to left anterior descending artery (15 cases). This collateral pathway was first noted in 1706 by Vieussens.[13] In roughly half of all human hearts, the conus artery arises from the proximal RC artery. In the remainder, it arises directly from a separate aortic ostium located above the right coronary cusp. It passes over the pulmonary outflow tract to terminate near the LAD artery and serves as an important collateral route in proximal LAD obstruction (Fig. 1*O*; and 12).

P. Posterior descending artery to left anterior descending artery *via* the cardiac apex (3 cases). This is the reverse of the pathway described previously in Section I but is seen less frequently. Here flow passes from the distal end of the PD artery through anastomotic connections around the apex of the heart to fill the distal segment of an obstructed LAD artery (Fig. 1*P*; and 13).

Q. Posterior descending artery to left anterior descending artery through the

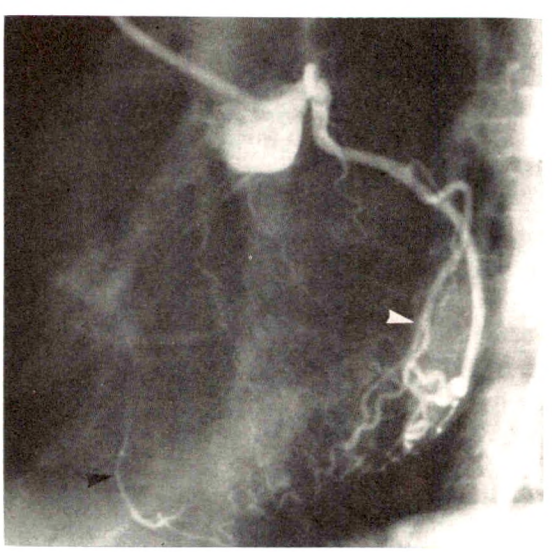


Fig. 10. Left coronary arteriogram, left anterior oblique projection. Collateral flow passes from the obtuse marginal branch (white arrowhead) of the circumflex artery to the distal segment of the obstructed left anterior descending artery (black arrowhead).

interventricular septum (3 cases). This is the reverse of the pathway described in Section H but it too is seen less frequently. The numerous anastomoses between the septal branches of the PD and LAD arteries provide a collateral route through which flow passes from the PD artery to the distal segment of an obstructed LAD artery (Fig. 12; and 13).

COLLATERAL PATHWAYS IN OBSTRUCTIONS OF THE LEFT CIRCUMFLEX ARTERY

Fifty-three patients in this series had moderate stenoses of the circumflex artery; no collaterals were present in this group. Forty-eight patients had severe stenoses or complete obstructions of this vessel, and 21 of them had evidence of collateral circulation.

I. HOMOCORONARY COLLATERALS

R. Either high obtuse marginal or diagonal branch to low obtuse marginal branch (11 cases). The circumflex artery may give rise to several OM branches supplying the anterolateral aspect of the left ventricle. Collaterals may pass from one of these branches to the other when the circumflex artery is obstructed between their origins. Less commonly, the proximal limb of this

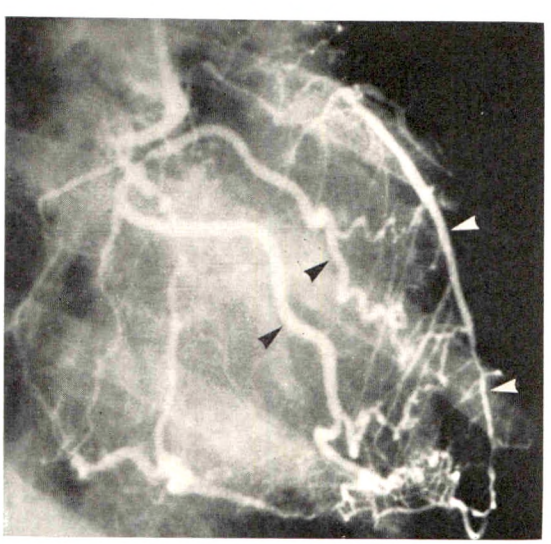


FIG. 11. Right coronary arteriogram, right anterior oblique projection. There is extensive collateral circulation from acute marginal branches (black arrowheads) of the right coronary artery to the left anterior descending artery (white arrowheads).

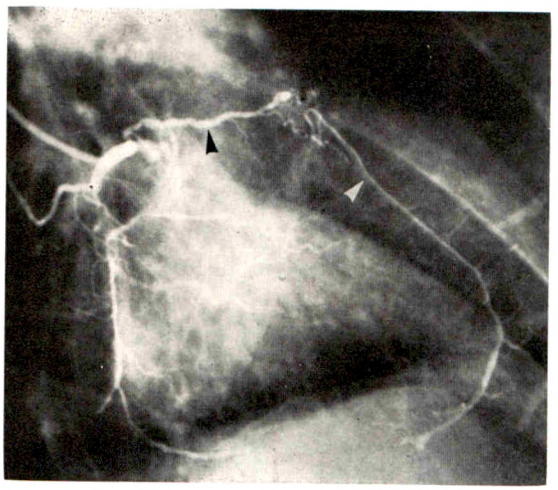


FIG. 12. Right coronary arteriogram, right anterior oblique projection. There is collateral flow from the conus branch (black arrowhead) to the distal segment of a severely stenotic left anterior descending artery (white arrowhead).

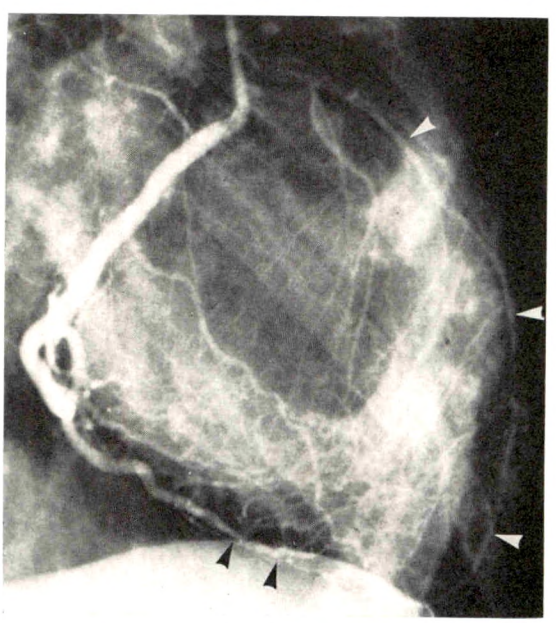


FIG. 13. Right coronary arteriogram, right anterior oblique projection. The patient has severe stenosis of the left main coronary artery. The left anterior descending artery (white arrowheads) fills from the posterior descending artery (black arrowheads) *via* collaterals passing both up through the interventricular septum and around the cardiac apex.

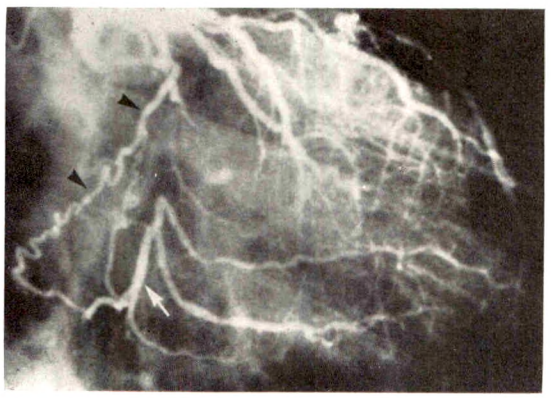


FIG. 14. Left coronary arteriogram, right anterior oblique projection. The circumflex artery is obstructed. Collateral flow passes from its left atrial circumflex branch (black arrowheads) to the distal circumflex artery (white arrow).

collateral pathway is the diagonal branch of the LAD artery (Fig. 1*R*).

S. Left atrial circumflex branch to distal circumflex artery (7 cases). If the circumflex artery is occluded beyond the origin of its left atrial circumflex branch, collaterals may pass from the atrial vessel to the distal circumflex artery (Fig. 1*S*; and 14).

II. INTERCORONARY COLLATERALS FROM THE RIGHT CORONARY ARTERY

T. Distal right coronary artery to distal circumflex artery (2 cases). This is the reverse of the pathway described above in Section E but is seen only infrequently. Flow passes from the distal portion of the dominant RC artery to the nearby distal segment of the obstructed circumflex artery (Fig. 1*T*).

U. Posterior left ventricular branch to obtuse marginal branch (2 cases). This is the reverse of the pathway described in Section G, and is also seen only infrequently. Here collaterals connect the PLV branch of the RC artery with the nearby OM branch of an obstructed circumflex artery (Fig. 1*U*).

OTHER TYPES OF CORONARY AUTO-REVASCULARIZATION

Collateral arterial circulation is not the only mechanism by which blood flow can be maintained in ischemic areas of myocardium. Recanalization of coronary artery thrombi also plays an extremely important part in this process of coronary autorevascularization. Angiographically, a recanalized thrombus has the same appearance as a very severe localized stenosis and it is virtually impossible to differentiate the 2.

Another phenomenon which may resemble recanalization angiographically but which is different pathophysiologically, is the development of a network of tiny bridging vessels which bypass segmental occlusions. James[7] believes that these are not true collateral arteries, but are instead enlarged vasa vasorum in the wall of the coronary artery.

Finally, anatomic studies have suggested that collateralization might occur through Thebesian vessels, arterioluminal connections within the heart and retrocardiac connections between bronchial or mediastinal arteries and the coronary arteries.[4] There has been no convincing clinical or angiographic evidence, however, that these pathways are of any significance in human coronary artery disease.

SUMMARY

In the presence of obstructing coronary atherosclerosis, collateral circulation may develop through pre-existing anastomotic channels which are present, though nonfunctional, in normal hearts. These anastomoses connect various branches of the same coronary artery or different coronary arteries. The common collateral pathways found among a group of 200 patients with significant coronary artery disease are described. Collateral circulation is only visible angiographically when severe narrowing (greater than 90 per cent) or total occlusion of the coronary artery is present.

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CORONARY COLLATERAL CIRCULATION AND DISTAL CORONARY RUNOFF: THE KEY FACTORS IN PRESERVING MYOCARDIAL CONTRACTILITY IN PATIENTS WITH CORONARY ARTERY DISEASE*

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CONSIDERABLE controversy exists over the role of collateral circulation in patients with obstructing coronary atherosclerosis. Some authors feel that it exerts a beneficial effect upon the course of the disease. Rees[12] has stated that collaterals may preserve normal myocardial function despite multiple arterial obstructions. Franklin[4] believes that adequate collateral circulation originating from arteries which are themselves non-stenotic can significantly ameliorate symptoms and improve prognosis. Adams *et al.*[1] feel that myocardial ischemia is reduced and Mason *et al.*[11] suggest somewhat cautiously that collaterals may protect against more severe forms of myocardial dysfunction. Other authors,[3,7,8] however, are of the opinion that collateral circulation does not benefit either cardiac function, patient symptomatology or prognosis.

The largest previous angiographic study of the effect of collateral circulation upon function of the left ventricle (LV) in patients with coronary artery disease (CAD) is that of Helfant *et al.*[7] These investigators compared ventricular contractility in patients having single or multiple stenoses greater than 75 per cent *without* collateral circulation and that in patients having single or multiple stenoses greater than 75 per cent *with* collateral circulation. Left ventriculography performed in the right anterior oblique projection in these 2 groups revealed a slightly higher incidence of contraction abnormalities among the group *with* collaterals. They concluded that collateral circulation offers no protection against development of an abnormal ventricular contraction pattern.

This conclusion, however, appears to be based upon a fallacious statistical grouping of subjects. Previous authors[5,13] have stated, and our own experience has confirmed, that collateral circulation can only be visualized angiographically when severe stenosis (90 per cent or more) or complete occlusion of a coronary artery has occurred. Moderate stenoses (75–89 per cent) undoubtedly also stimulate development of collateral circulation, but at this earlier stage, flow is so minimal as to be imperceptible angiographically. It follows that the Helfant *et al.*[7] group "with collaterals" would consist exclusively of patients with severe stenoses (over 90 per cent) or complete occlusions. The group "without collaterals" would contain some patients with severe stenoses or complete occlusions but would also contain all those patients having only moderate stenoses (75–89 per cent); *i.e.*, it would be heavily weighted with patients having less severe disease. Since the patients "with collaterals" have more advanced CAD as a group, it is hardly surprising that they exhibit ventricular dysfunction more commonly.

The purpose of this paper is a re-evaluation of the role of collateral circulation in preservation of regional myocardial con-

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tractility in CAD patients. In particular, an effort has been made to categorize patients carefully according to the severity of the obstructing lesion, the adequacy of collateral circulation and distal arterial runoff flow and the degree of ventricular contraction abnormality.

METHOD AND RESULTS

Two hundred consecutive patients with significant CAD were included in this study. All had at least 75 per cent stenosis of at least 1 major coronary artery supplying the anterior and/or diaphragmatic surfaces of the LV. Coronary arteriography was performed by the percutaneous femoral approach, utilizing catheters of the Judkins[10] or Amplatz *et al.*[2] design. Both cine filming and rapid serial roentgenograms were obtained. Cine left ventriculography was performed in the right anterior oblique projection, providing optimal observation of contractility of the anterior and diaphragmatic surfaces. Lesions of the left circumflex artery were excluded from consideration when it was a relatively small vessel perfusing only a portion of the lateral wall of the LV. This area is not well seen on right anterior oblique ventriculograms. In such cases, only lesions of the left anterior descending and right coronary arteries were taken into account, since these supply the anterior and diaphragmatic surfaces of the LV, respectively. Lesions of the circumflex artery were included only when it was the "dominant" vessel and provided significant blood flow to the diaphragmatic aspect of the ventricle.

Individual coronary arteries were first graded according to degree of obstruction. Three different grades were recognized, as follows: (1) 75 to 89 per cent—these lesions are hemodynamically of only moderate degree and are virtually never accompanied by angiographically visible collaterals; (2) 90 to 99 per cent—these lesions are hemodynamically severe and can stimulate the development of angiographically visible collaterals; and (3) total occlusion.

The same vessels were then graded according to runoff flow distal to the obstructing lesion. Although there is no way to directly measure this runoff flow in the intact patient, a good approximation is obtained by noting the caliber of the opacified distal segment. Where the distal segment had a measured diameter of over 1 mm. after correction for magnification, runoff flow was considered adequate. Where the distal segment diameter was 1 mm. or less or a complete lack of distal opacification occurred, runoff flow was considered inadequate.

Finally, in every instance in which a significant obstruction of a coronary artery was seen, careful ventriculographic evaluation was made of the contractility of the affected portion of myocardium in the resting state. Function was considered normal if a uniform, forceful, nearly concentric contraction of the endocardial surface occurred.[9] Mild regional dysfunction was manifested by hypokinesia; *i.e.*, contractility was still present but diminished in degree. Severe regional dysfunction was manifested by either a total lack of contractility (akinesia) or paradoxical bulging (dyskinesia) during ventricular systole. Akinesia or dyskinesia usually indicates advanced myocardial scarring or fibrous aneurysm formation, although it has been pointed out that on rare occasions they may occur in areas of myocardium which are ischemic but still viable.[6]

The results of the study are summarized in the accompanying tables. Table I examines the effect of severity of coronary artery obstruction upon regional left ventricular contractility. Table II examines the effect of adequacy of distal coronary artery runoff (regardless of the degree of obstruction) upon regional left ventricular contractility. Table III combines these elements by subdividing each grade of arterial obstruction according to whether or not adequate distal runoff is present. The combined effect of these factors upon regional contractility is then assessed.

TABLE I

CORRELATION OF REGIONAL LV CONTRACTILITY WITH DEGREE OF CORONARY ARTERY OBSTRUCTION

Degree of Coronary Artery Obstruction	No. of Affected Vessels	Regional LV Contractility		
		Normal	Hypokinesia	Akinesia-Dyskinesia
75–89%	51	74%	16%	10%
90–99%	81	60%	26%	14%
Complete Obstruction	166	28%	35%	37%

DISCUSSION

Table I indicates that incomplete coronary obstructions (75–89 per cent, and 90–99 per cent groups) are in the vast majority of instances accompanied by normal myocardial contractility or regional hypokinesia. The more severe forms of ventricular dysfunction, akinesia or dyskinesia, occur only rarely with such lesions. With complete arterial occlusion, akinesia or dyskinesia was seen somewhat more frequently, occurring 37 per cent of the time. However, it is noteworthy that 35 per cent of these completely occluded arteries were distributed to hypokinetic myocardium and 28 per cent were distributed to areas exhibiting entirely normal contractility—virtually a random distribution of results! If degree of obstruction were the primary determinant of contractility, one would not expect as many as 28 per cent of complete arterial occlusions to be accompanied by perfectly normal contractility. Conversely, one would expect more than 37 per cent of such lesions to result in areas of akinesia or dyskinesia. It seems obvious that some additional factor must act as the principal determinant of left ventricular function in these patients.

Table II shows how regional myocardial contractility is affected by the adequacy of distal coronary artery runoff and suggests that the latter is this key factor. Among all significantly diseased coronary arteries with adequate distal runoff, normal regional contractility occurred in 60 per cent, hypokinesia in 35 per cent and akinesia or dyskinesia in only 5 per cent. Where distal runoff was inadequate, however, only 11 per cent had normal contractility, 18 per cent had hypokinesia and 71 per cent had akinesia or dyskinesia. Blood may, of course, reach the distal segment of a diseased coronary artery either in antegrade fashion past an incomplete obstruction or *via* collateral circulation where obstruction is total or nearly so.

Table III carries this correlation one step further by analyzing the effect of runoff upon contractility within each of the three

TABLE II

CORRELATION OF REGIONAL LV CONTRACTILITY WITH ADEQUACY OF DISTAL CORONARY ARTERY RUNOFF FLOW

Distal Coronary Artery Runoff	No. of Affected Vessels	Regional LV Contractility		
		Normal	Hypokinesia	Akinesia-Dyskinesia
Adequate (distal vessel diameter > 1 mm.)	202	60%	35%	5%
Inadequate (distal vessel diameter 1 mm. or less)	96	11%	18%	71%

Table III

CORRELATION OF REGIONAL LV CONTRACTILITY WITH BOTH DEGREE OF CORONARY ARTERY OBSTRUCTION AND ADEQUACY OF DISTAL RUNOFF

Degree of Coronary Artery Obstruction	Distal Runoff	No. of Affected Vessels	Regional LV Contractility		
			Normal	Hypokinesia	Akinesia-Dyskinesia
75–89%	Adequate	48	79%	15%	6%
	Inadequate	3	0%	33%	67%
90–99%	Adequate	68	69%	27%	4%
	Inadequate	13	15%	23%	62%
Complete Obstruction	Adequate	86	43%	52%	5%
	Inadequate	80	11%	16%	73%

grades of obstruction. It shows that within each grade, regional myocardial contractility is strikingly better when distal coronary runoff is adequate than when it is inadequate.

Completely occluded coronary arteries provide the optimal isolated model for studying the effect of collateral circulation upon left ventricular contractility. Any blood flow within the distal segment of a totally occluded vessel must, by definition, arrive *via* collateral circulation. Adequate distal runoff in such an artery indicates well-developed collateral circulation emanating either from other nearby arteries or from the proximal segment of the same artery. Conversely, inadequate distal runoff indicates poor collateral circulation, diffuse disease of the distal segment or both. Table III shows that among 86 completely occluded arteries with adequate distal flow *via* collateral circulation, the affected

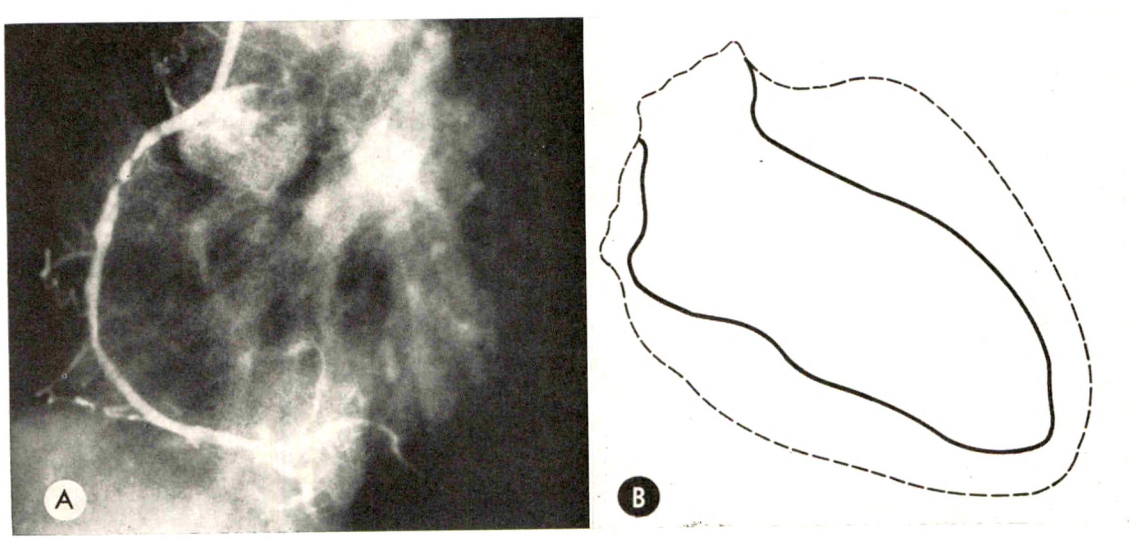


Fig. 1. (*A*) Right coronary arteriogram, left anterior oblique view. Very severe proximal stenosis is present but distal runoff flow in the antegrade direction is quite adequate in spite of diffuse atherosclerotic plaque formation. (*B*) Line tracing of the patient's left ventriculogram in the right anterior oblique projection. The contour of the LV is indicated at end-systole by the solid line and at end-diastole by the broken line. Note normal contractility of the diaphragmatic surface, which is the area affected by the right coronary stenosis.

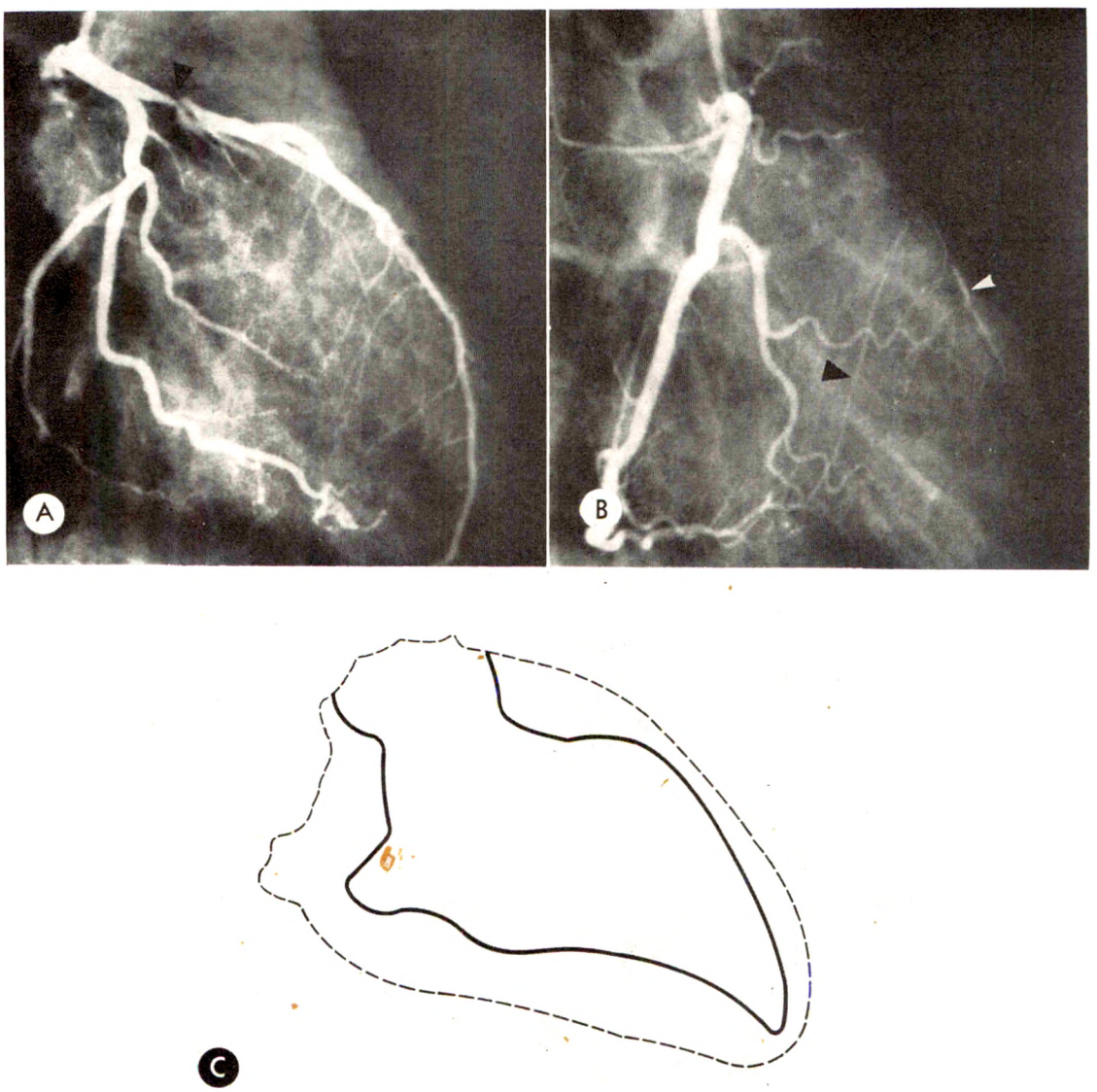


FIG. 2. (*A*) Left coronary arteriogram, right anterior oblique projection. There is severe stenosis (black arrowhead) of the proximal left anterior descending artery with adequate antegrade runoff in the distal segment. (*B*) Right coronary arteriogram, right anterior oblique view. Developing collateral circulation is seen from the posterior descending artery through a large septal branch (black arrowhead) to the distal left anterior descending artery (white arrowhead). (*C*) Ventriculography shows that the corresponding regions of the LV, the anterior wall and apex, are hypokinetic but that some degree of contractility has been preserved.

myocardium contracted normally in 43 per cent of cases and was hypokinetic in 52 per cent. Akinesia or dyskinesia occurred in only 5 per cent. On the other hand, among 80 completely occluded arteries having inadequate distal flow, normal regional contraction and hypokinesia occurred in only 11 per cent and 16 per cent of cases respectively, whereas 73 per cent exhibited akinesia or dyskinesia.

The relationships between the various factors discussed above are demonstrated in Figures 1–7. Figures 1 and 2 show rapid and complete antegrade filling of the distal

segments of coronary arteries having very severe proximal stenosis. This phenomenon occurs with surprising frequency. In both cases, regional left ventricular contractility is preserved, although hypokinesia is seen in Figure 2. This case also demonstrates development of early collateral circulation from the posterior descending artery through the ventricular septal branches to the left anterior descending artery in response to severe narrowing of the latter. Figures 3–5 are cases in which akinesia or dyskinesia has developed secondary to complete obstruction of a major artery with inadequate collateral circulation and distal runoff. The patient shown

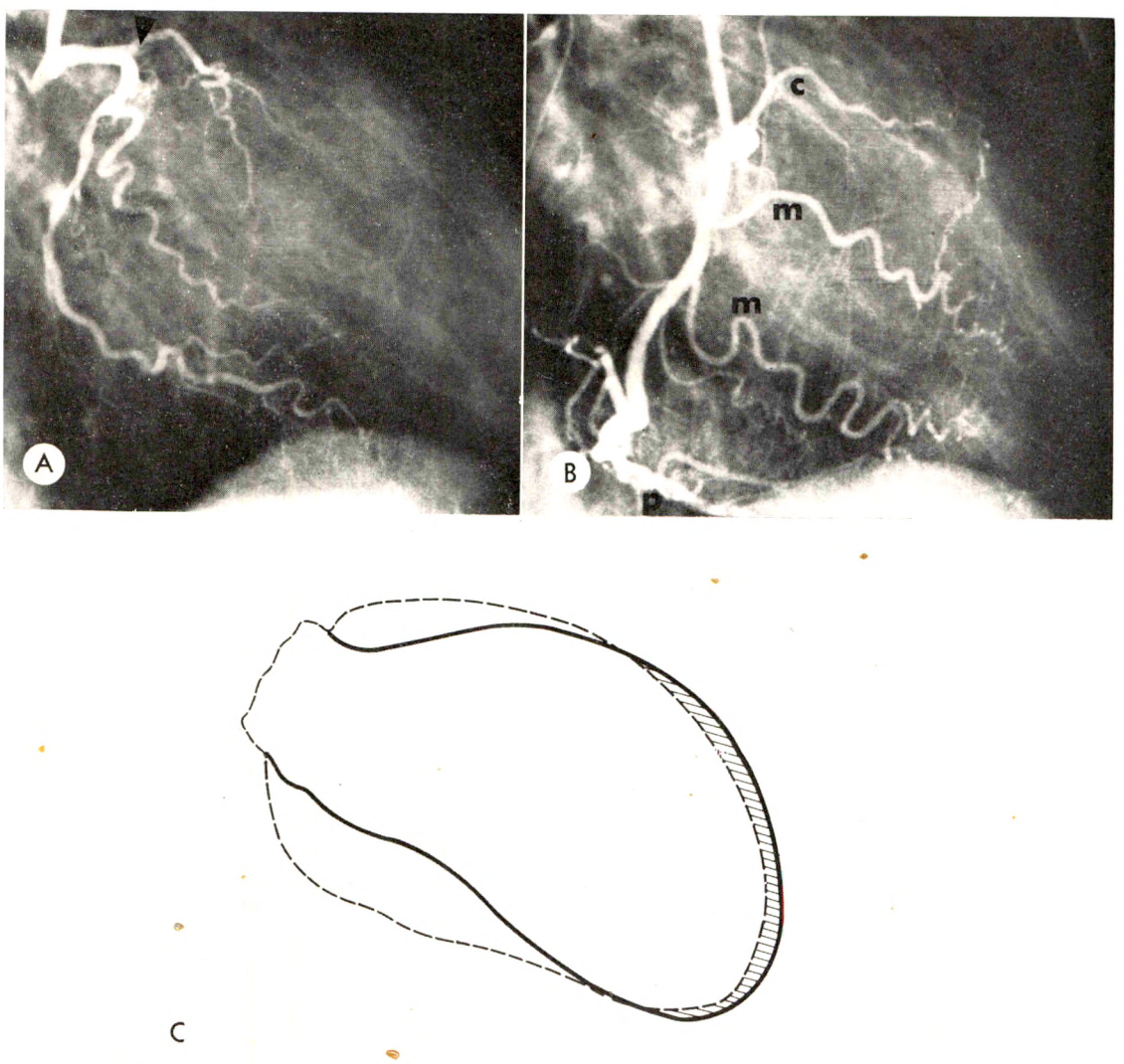


Fig. 3. (*A*) Left coronary arteriogram, right anterior oblique projection. Complete obstruction of the proximal left anterior descending artery is present (arrowhead) with no evidence of filling of the distal segment. (*B*) Right coronary arteriogram, right anterior oblique projection. Note hypertrophy and increased flow through the distal tributaries of the conus (c), marginal (m) and posterior descending (p) arteries, in an apparent attempt to supply collateral flow. In spite of this, the distal left anterior descending artery fails to opacify. Collateral circulation from both sides is thus inadequate and distal runoff is totally absent. (*C*) Ventriculography reveals a large dyskinetic segment of the corresponding anterior and apical portions of the LV.

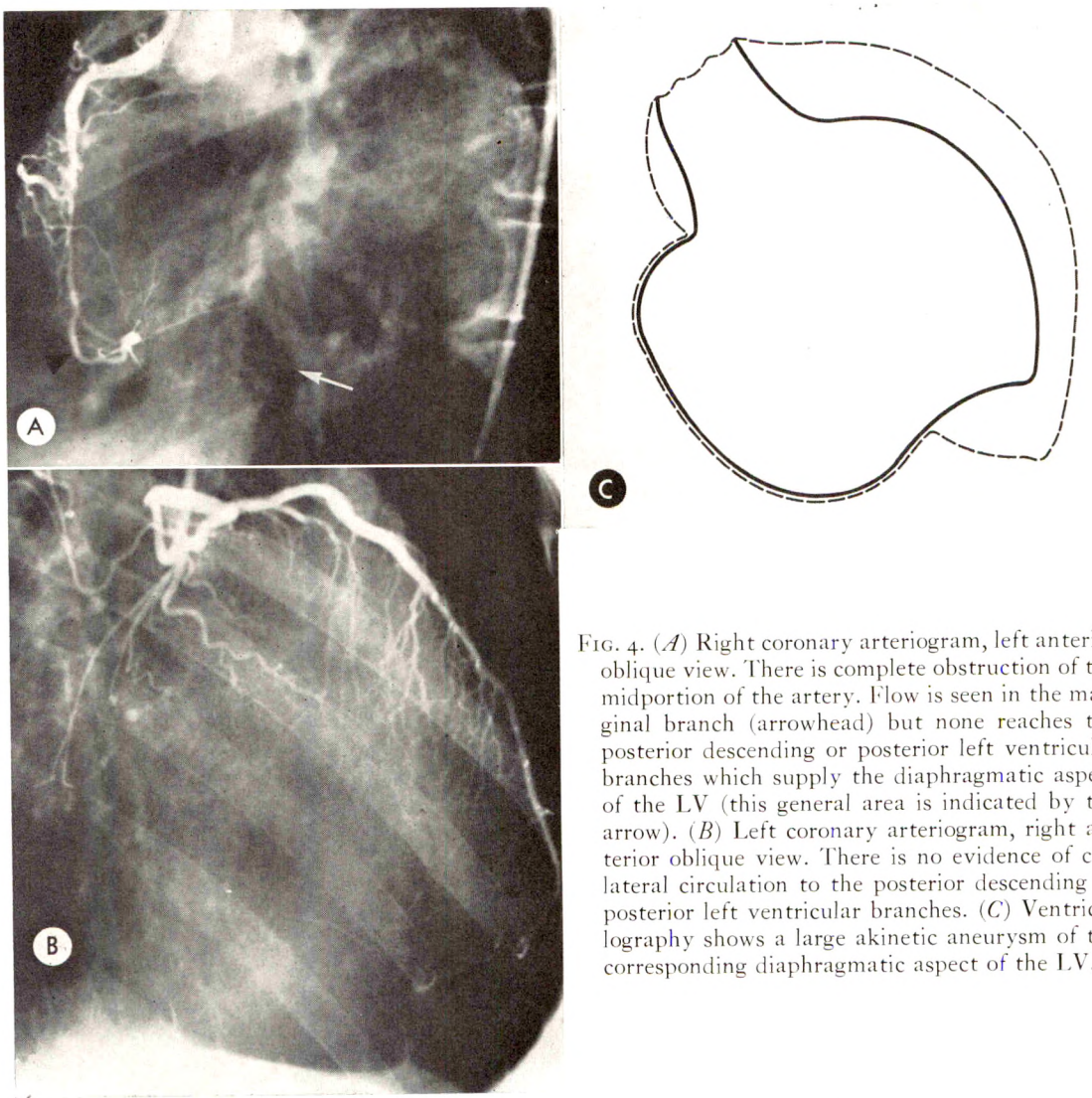


FIG. 4. (*A*) Right coronary arteriogram, left anterior oblique view. There is complete obstruction of the midportion of the artery. Flow is seen in the marginal branch (arrowhead) but none reaches the posterior descending or posterior left ventricular branches which supply the diaphragmatic aspect of the LV (this general area is indicated by the arrow). (*B*) Left coronary arteriogram, right anterior oblique view. There is no evidence of collateral circulation to the posterior descending or posterior left ventricular branches. (*C*) Ventriculography shows a large akinetic aneurysm of the corresponding diaphragmatic aspect of the LV.

in Figure 6 has complete obstruction of the right coronary artery with good runoff flow through the entire distal segment and its posterior descending and posterior left ventricular branches *via* collateral circulation. As a result, contraction of the diaphragmatic surface of the ventricle is normal. He also has a severe localized stenosis of the proximal left anterior descending artery with excellent antegrade flow in the distal segment. Contraction of the anterior wall and apex of the ventricle is nearly normal. The patient shown in Figure 7 has complete obstruction of the left anterior descending artery but has maintained normal contractility of the anterior wall of the ventricle because of good collateral circulation and good distal runoff in the artery.

It thus appears that with stenoses of less than 90 per cent, distal runoff almost always is adequate and regional myocardial contractility is likely to be normal. Collateral circulation is almost never visible angiographically in this group.

When the degree of stenosis exceeds 90 per cent, blood reaches the distal segment either in antegrade fashion past the stenotic lesion or *via* angiographically visible collateral circulation or both. In any event, if distal runoff flow is adequate, regional myocardial contractility will be normal in most cases and mildly impaired in some

others. Only rarely will severe impairment be noted. Where distal runoff is not adequate, however, most cases will show severe impairment of contractility.

Of those coronary arteries which are completely occluded, approximately half will demonstrate adequate distal runoff *via* collateral circulation. In the vast majority of these instances, regional contractility will be either mildly impaired or normal. The other half will demonstrate inadequate collateral circulation and distal runoff, and the vast majority of these lesions will result in severe impairment of contraction. The reasons for failure of development of good collateral circulation in some CAD patients are as yet poorly understood. A variety of factors is probably responsible, including compromised flow in the donor vessel and clotting or diffuse atherosclerosis of the distal segment of the recipient vessel.

The logical conclusion to be drawn from this study is that the key determinants of left ventricular contractility in the resting state in patients with CAD are collateral circulation and distal coronary artery runoff. When they are adequate, myocardial function is significantly better than when they are inadequate, regardless of the severity of the obstruction of the vessel. As indicated earlier, there remains some contention as to whether these factors can ameliorate symptoms or improve prognosis. Further studies of a prospective nature are required to resolve these important questions.

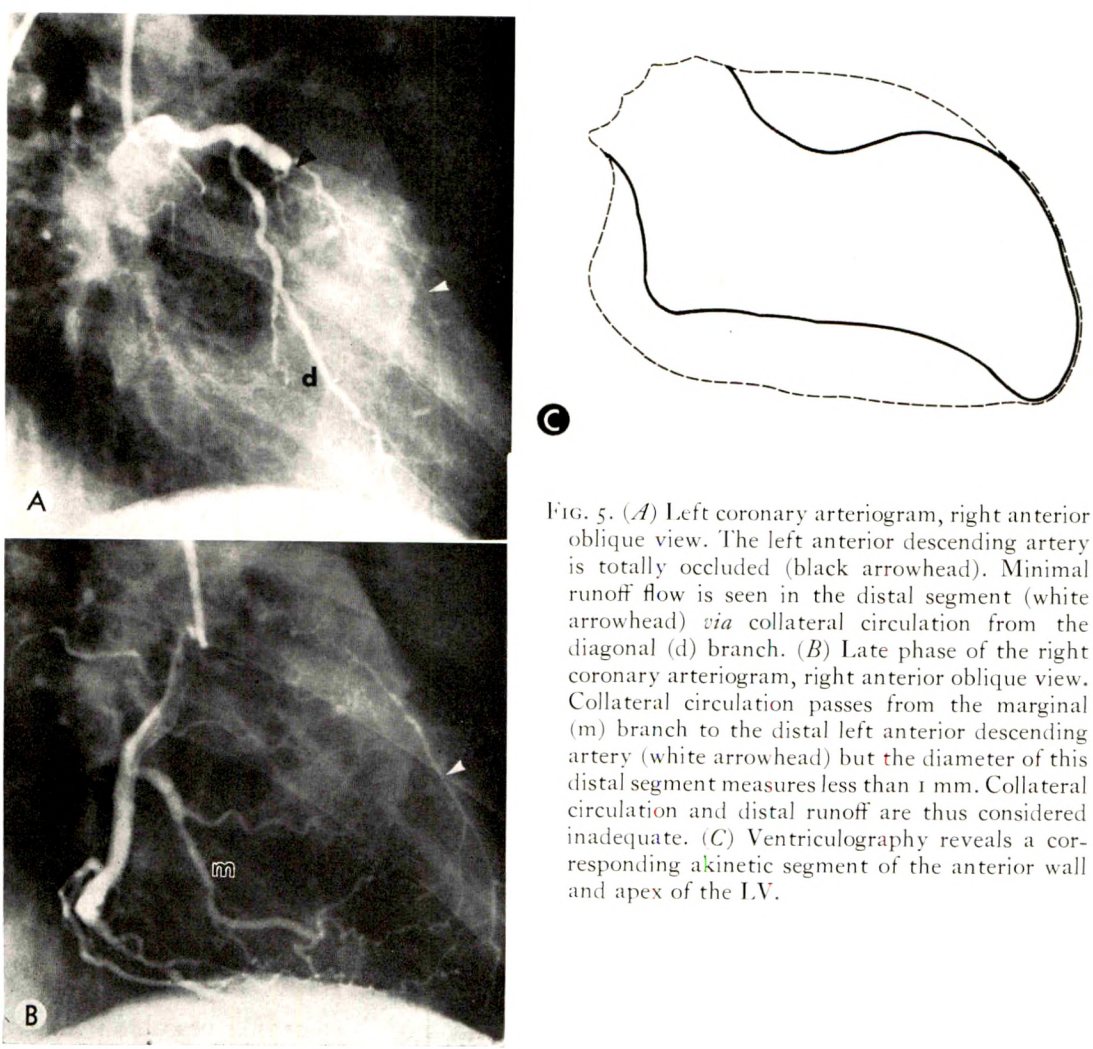


FIG. 5. (*A*) Left coronary arteriogram, right anterior oblique view. The left anterior descending artery is totally occluded (black arrowhead). Minimal runoff flow is seen in the distal segment (white arrowhead) *via* collateral circulation from the diagonal (d) branch. (*B*) Late phase of the right coronary arteriogram, right anterior oblique view. Collateral circulation passes from the marginal (m) branch to the distal left anterior descending artery (white arrowhead) but the diameter of this distal segment measures less than 1 mm. Collateral circulation and distal runoff are thus considered inadequate. (*C*) Ventriculography reveals a corresponding akinetic segment of the anterior wall and apex of the LV.

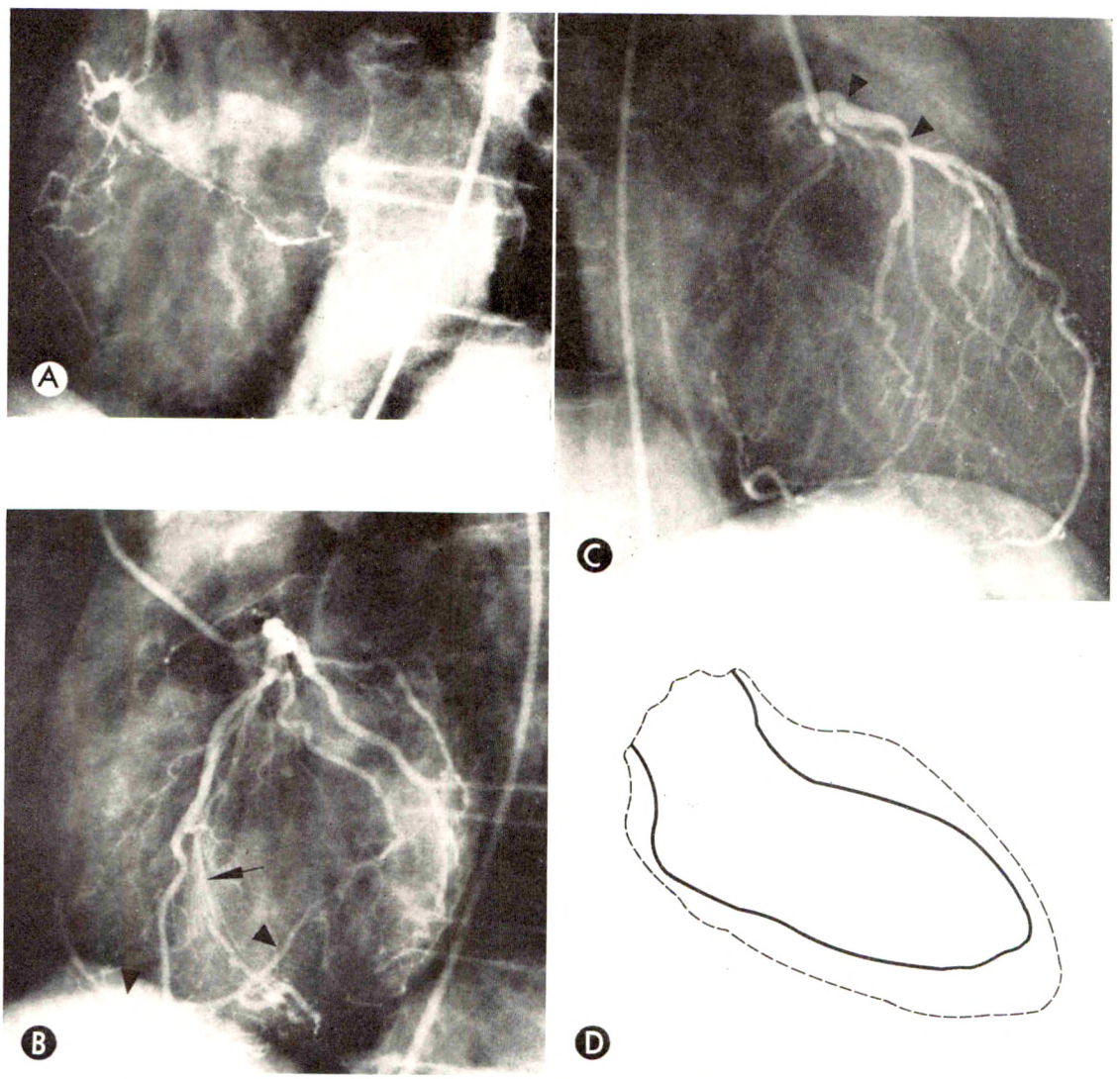


FIG. 6. (*A*) Right coronary arteriogram, left anterior oblique view. The right coronary is completely occluded with no distal flow seen. (*B*) Left coronary arteriogram, left anterior oblique view. The entire distal right coronary tree (arrowheads), including the posterior descending and posterior left ventricular branches, is well-filled *via* collateral circulation. A large septal branch (arrow) of the left anterior descending artery is the principal collateral route. (*C*) Left coronary arteriogram, right anterior oblique view. Two stenotic lesions of the left anterior descending artery are seen (arrowheads), the more proximal of which is quite localized but severe. The patient thus has complete right coronary occlusion with adequate distal runoff *via* collateralization, and severe left anterior descending stenosis with adequate antegrade distal runoff. (*D*) Ventriculography shows nearly normal contractility of the LV except for mild hypokinesia of the lower portion of the anterior wall.

SUMMARY

The effects of obstructing coronary artery lesions upon regional left ventricular contractility were studied angiographically in 200 patients with significant coronary artery disease. The arteries were graded according to degree of obstruction and adequacy of distal runoff flow beyond the lesion. The corresponding regional myocardial contractility was evaluated by ventriculography.

Left ventricular contractility was found to be determined not primarily by the degree of arterial obstruction but rather by the adequacy of runoff flow in the distal segment of the diseased vessel. In cases of

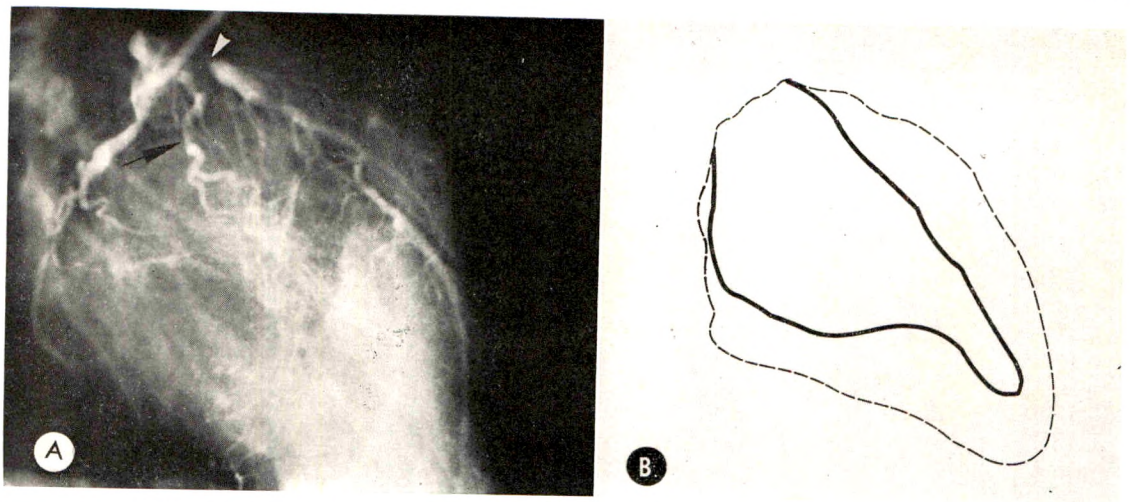


FIG. 7. (*A*) Left coronary arteriogram, right anterior oblique view. The left anterior descending artery is totally occluded proximally (arrowhead). Collateral circulation reaches the distal segment after originating from the first septal branch (arrow) and passing to more distal septal branches. Runoff flow in the distal left anterior descending artery appears adequate. (*B*) Ventriculography shows normal contractility of the corresponding anterior portion of the LV.

complete occlusion, the adequacy of distal runoff flow is determined directly by the degree of development of collateral circulation. Normal or mildly impaired contractility was present in most instances where distal runoff was adequate; regional akinesia or dyskinesia occurred in most instances where distal runoff was inadequate.

Collateral circulation and distal coronary runoff are thus felt to be the key factors in preserving myocardial contractility in patients with coronary artery disease.

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ANGIOGRAPHIC DEMONSTRATION OF COMPLICATIONS RELATED TO THE SAPHENOUS AORTO-CORONARY BY-PASS PROCEDURE*

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IN the last few years, the surgical treatment of coronary artery disease has met with great enthusiasm. The saphenous-venous by-pass procedure is considered the operation of choice when one seeks aggressive therapy of atherosclerotic obstructive coronary artery disease.[4,5] Of late, more and more papers have described the results of the procedure.[2,3,8,9] Most reports discuss the patency rate of the by-pass and its role in clinical symptomatology. In addition to the presence or lack of patency we have, however, found a variety of significant post-operative complications with which both angiographers and clinicians must be familiar. Some of these complications may explain the deterioration of left ventricular function which sometimes occurs during the postoperative period.

The purpose of this paper is to describe these complications and to discuss their effect upon myocardial function.

METHOD OF INVESTIGATION

Patients who are candidates for the saphenous-venous by-pass procedure are studied preoperatively in our laboratory by coronary angiography and left ventriculography. Ventricular volumes and ejection fractions are calculated utilizing the left ventriculogram in the right anterior oblique projection and Dodge's formula adapted to a single plane method.[5] Intraventricular pressures are always recorded. In an attempt to assess the value of the procedure, a certain number of patients, whether symptomatic or not, were restudied (if they accepted the procedure) between 2 weeks and 18 months after surgery. Venous by-pass angiography and left ventriculography were done in each case. Coronary angiography was not repeated routinely; it was only performed in those cases where the graft was occluded or unidirectional flow[7] was found in the coronary artery to which the vein by-pass had been connected. As of this date, 50 patients have been re-examined. The complications which will be described have been found in this group of patients.

DISCUSSION OF THE MATERIAL

For the sake of simplicity, the complications are divided into 2 groups: 1. complications affecting the veins; and 2. complications affecting the coronary arteries.

1. COMPLICATIONS AFFECTING THE VEINS

(A) *Clotting.* The most obvious complication is clotting of the vein by-pass. Early clotting (from 2 weeks to 4 months after surgery) has occurred in 14 per cent of our cases, whereas late occlusions (from 4 months to 18 months postoperatively) occurred in 30 per cent of our cases.[2] Angiographically, one can usually demonstrate a remnant of the vein (Fig. 1) when occlusion occurs, but sometimes the clot extends all the way back to the orifice of the vein and no stump can be seen. This has happened only twice in our experience.

Proximal clotting of the vein, however, does not necessarily mean that the entire vein is involved. It is most important to obtain a coronary angiogram when a vein by-pass is occluded. At times, one may demonstrate reflux of contrast medium from the coronary artery into the distal still patent portion of the vein (Fig. 2, *A* and *B*; and 3). When such patency is deter-

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mined, it simplifies the task of the surgeon when the patient is re-operated upon. The distal patent segment of the vein is then connected to a new vein which is in turn attached to the aorta. Obviously, re-operation is simplified because it can be done without stopping the heart or requiring cardiopulmonary by-pass.

Clotting of the vein is not necessarily accompanied by myocardial infarction. Following such an event, myocardial perfusion may revert to its preoperative status.

(B) *Intimal hyperplasia.* To a certain extent, thickening of the intima of the vein caused by fibroblastic cells lying in a basophilic mucoid matrix occurs in all cases.[11] This process has been called intimal fibrous proliferation by Vlodaver and Edwards,[11] who object to the term arteriolization of the vein, since they did not notice any changes in the media. Angiographically, this process is manifested by reduction of the inner caliber of the vein (Fig. 4, *A* and *B*). Sometimes the intimal hyperplasia becomes so severe that the flow within that vessel is markedly hindered (Fig. 5). This process might eventually lead to clotting of the vein and failure of the procedure.

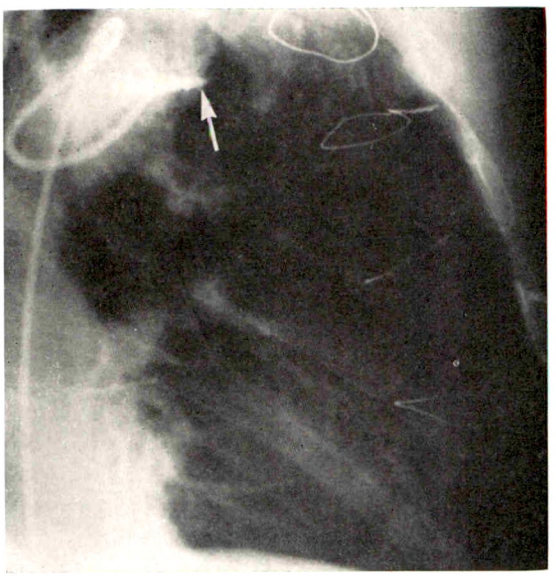


FIG. 1. Angiogram in right anterior oblique projection demonstrating a clotted graft. Note that the tip of the catheter is in a stump (arrow). This angiogram belongs to the patient demonstrated in Figure 10, *A–C*.

(C) *False aneurysms.* Aneurysmal dilatation of the vein is an unusual complication

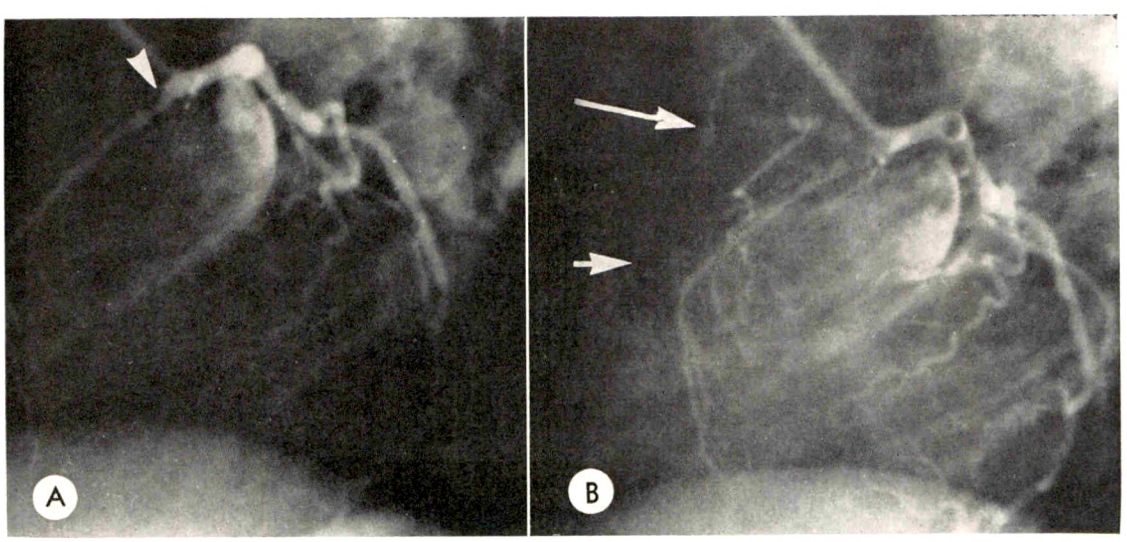


FIG. 2. (*A* and *B*) Selective left coronary arteriograms in left anterior oblique projection, which belong to a patient whose graft to the left anterior descending (LAD) artery is occluded. (*A*) The LAD is occluded (arrowhead points to a stump). (*B*) (Obtained at a later sequence.) The LAD (short white arrow) fills through collaterals and opacifies partially the vein graft (long white arrow).

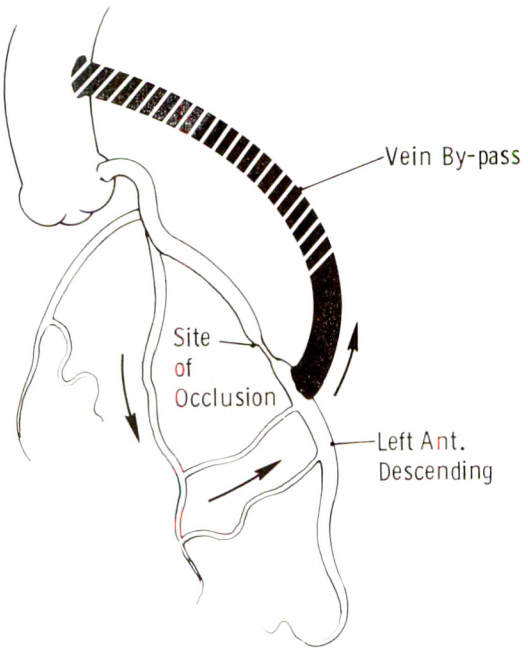


FIG. 3. Schematic representation of a partially occluded vein by-pass. The crosshatched lines represent the portion of the graft which is occluded.

which we have not found in our material. However, we have seen 2 cases of false aneurysms forming at the site of anastomosis of the vein to the aorta. Figure 6, *A* and *B*, demonstrates such a case. Interestingly, when this patient was studied 2 weeks after surgery, the vein by-pass was functioning normally and no evidence of false aneurysm was found (Fig. 6*A*). During a re-study 8 months after surgery, it was noted that the vein was occluded. The occlusion was caused by a false aneurysm of the anastomotic site pressing upon the by-pass (Fig. 6*B*). It is evident that such an aneurysm might be life-threatening and must be repaired.

II. COMPLICATIONS AFFECTING THE CORONARY ARTERIES

(A) *Loss of collaterals.* The opening of collateral vessels is an important process which may or may not take place during or after the occlusion of a major coronary artery. If collateralization is adequate,

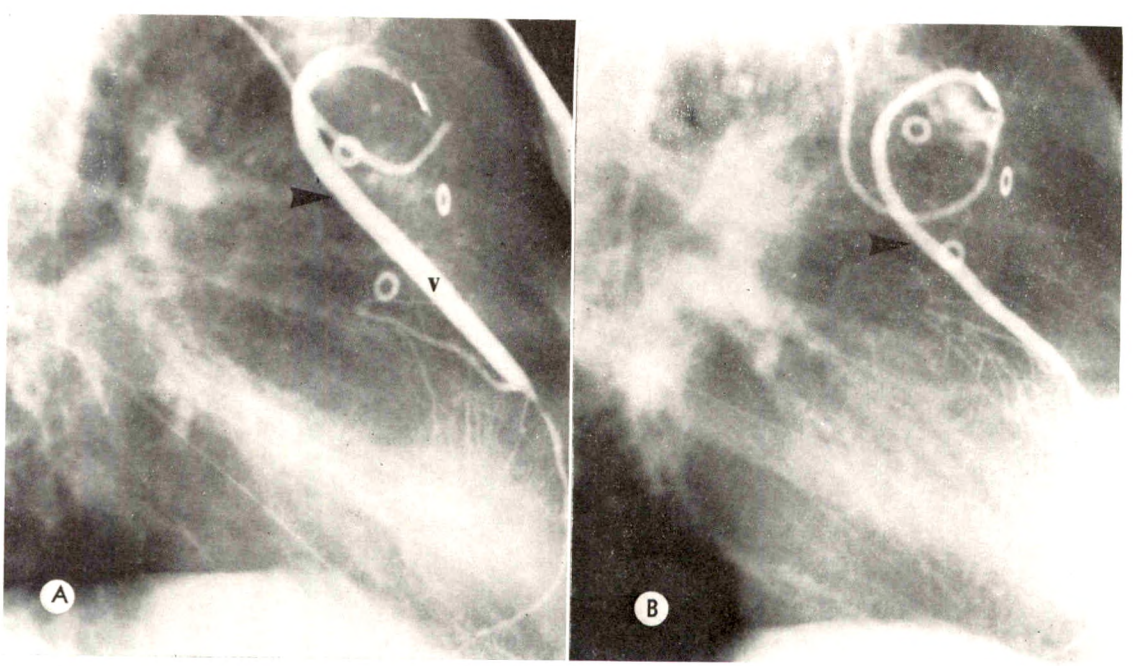


FIG. 4. (*A* and *B*) Vein by-pass (V) to the left anterior coronary artery seen in the right anterior oblique projection: (*A*) 12 days postoperatively; (*B*) 12 months postoperatively. Note the difference in caliber between *A* and *B*, probably due to intimal hyperplasia.

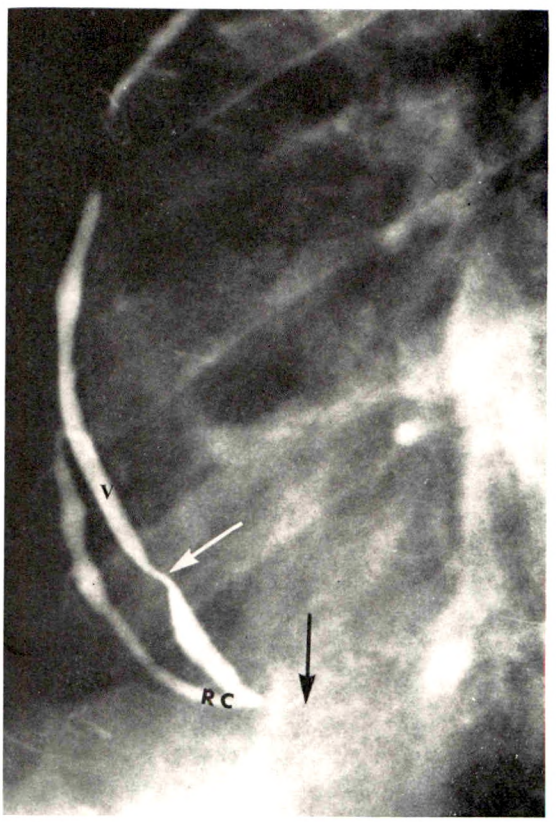


Fig. 5. Vein by-pass (V) to the distal right coronary artery (RC). The arrow points to an area of narrowing caused by intimal hyperplasia. Note absence of distal flow into the right coronary artery. Black arrow points to an area which should be opacified.

ventricular function is often preserved in the face of this occlusion. Many clinicians, however, feel that the amount of blood carried to the myocardium through these collaterals is inadequate and therefore they recommend a by-pass procedure. Such an undertaking might be hazardous to the patient, since these important collateral vessels may be lost if the vein by-pass clots. The patient illustrated in Figure 7, *A–C*, had excellent collateralization of an obstructed right coronary artery at the time of his pre-by-pass coronary angiogram. After the surgical procedure, the graft clotted, and re-examination of the right coronary artery failed to opacify these im-

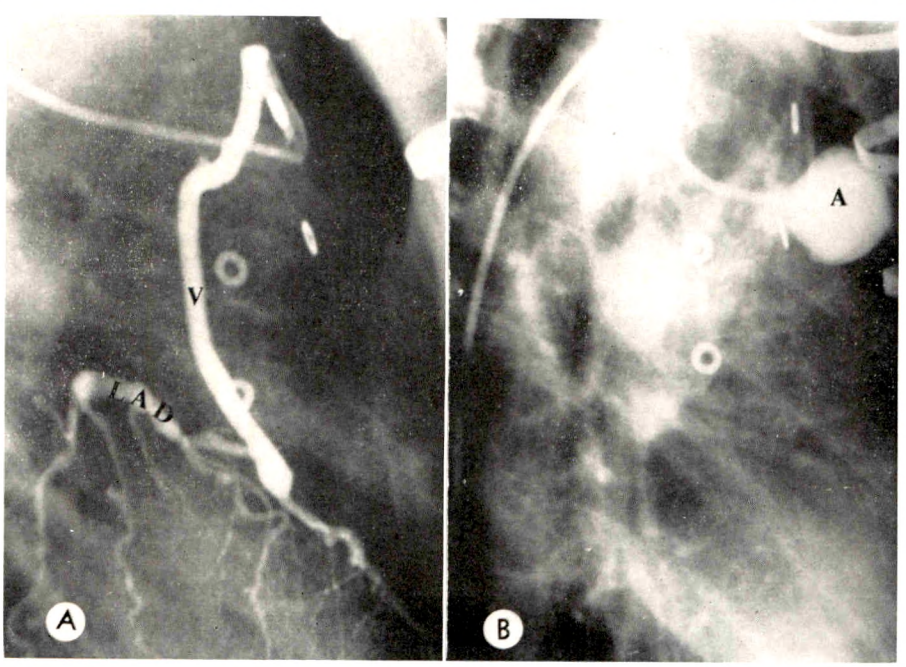
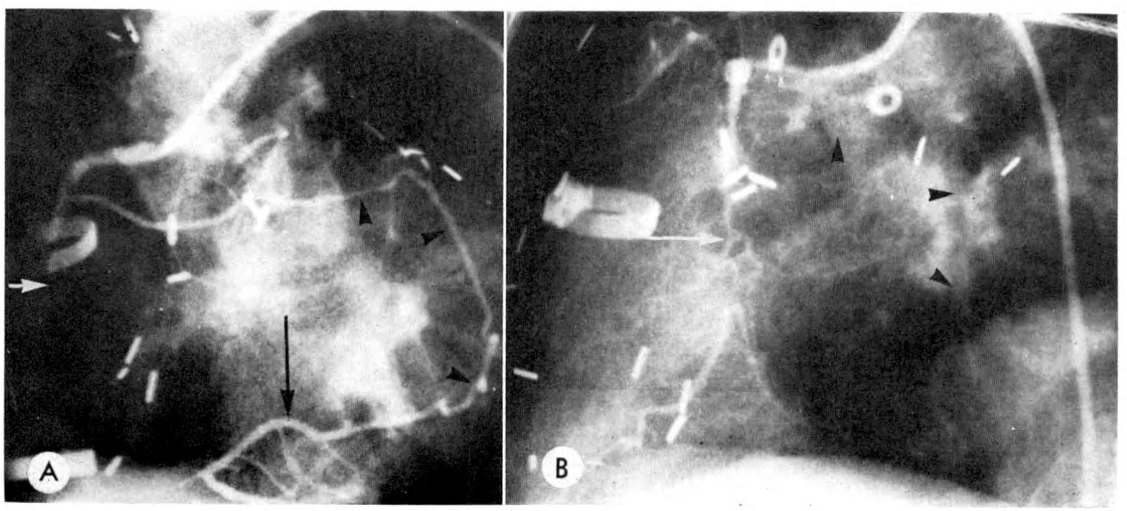


Fig. 6. (*A*) Angiogram in right anterior oblique projection of a by-pass (V) to (LAD) 16 days postoperatively; and (*B*) 16 months postoperatively. The vein is occluded and the catheter opacifies a false aneurysm (A).



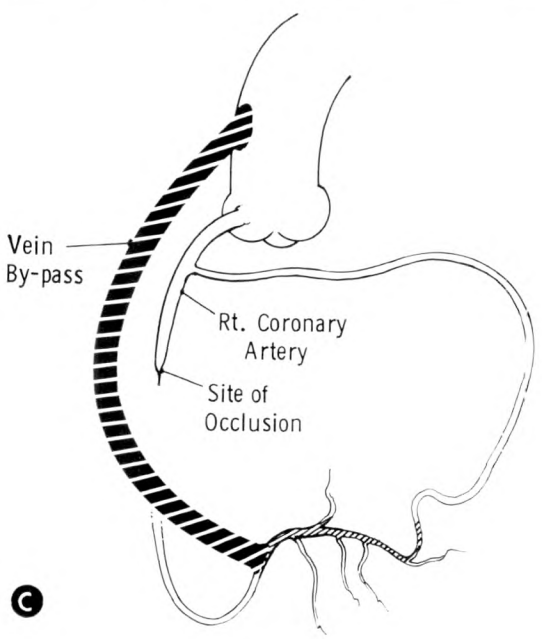


FIG. 7. (*A*) Selective right coronary arteriogram in left anterior oblique projection. The white arrow points to the occlusion of the right coronary artery. The arrowheads point to the collateral pathway bridging the proximal and distal (black arrow) right coronary artery. (*B*) Repeat right coronary angiogram obtained after vein by-pass. The graft is occluded and the collateral pathway (arrowheads point to where the collaterals should be) is not opacified. (*C*) Schematic explanation of *A* and *B*. The vein by-pass is occluded. The clot in the vein extended into the distal right coronary artery and destroyed the distal "runoff." The crosshatched lines represent clotting.

portant collaterals which contributed to the perfusion of the base of the heart. It is likely that the clot in the graft extended into the branch of the distal right coronary artery which was supplied by these collaterals. As a result, there was no more distal "runoff" and the collaterals ceased to function (Fig. 7*C*). In essence, as a result of the procedure, the patient lost the natural by-pass he had developed by himself.

(B) *Clotting of a major coronary artery proximal to the by-pass.* It is not unusual to see clotting of a coronary artery from the point of severe stenosis to the point of anastomosis of the vein to the coronary artery (Fig. 10*C*). This complication has already been reported by others.[1] If the atherosclerotic process reduces enough of the lumen of the coronary artery to produce symptoms, there is usually a pressure gradient between the proximal and distal portions of this vessel. As a result of this gradient, antegrade flow in the distal segment of the coronary artery is maintained. Once the vein has been inserted into the

distal portion of the coronary artery, the ascending aortic pressure is transmitted through the vein to the distal portion of the coronary artery. At that point, there is no more gradient between these 2 segments and the flow through the stenosis may no longer take place. The coronary artery from the point of stenosis to the graft is perfused retrograde from the vein. This flow, however, is turbulent because the anastomosis of the vein to the coronary artery is at an angle which favors forward flow (Fig. 8). The turbulence and lack of antegrade flow through the stenosis often lead to clotting. If the vein has been anastomosed to the coronary artery far enough beyond the stenosis, this proximal clotting may involve a large portion of the coronary artery and compromise important branches arising from it. Thus, in the patient demonstrated in Figure 9, *A–C*, a large portion of the right coronary artery and important muscular branches arising from it were lost. Ventricular function deteriorated in this patient; the left ventricular ejection fraction was 0.76 pre- and immediately postoperatively, but decreased to 0.56 after the clotting of the proximal portion of the right coronary artery. In this case, the repeat right coronary angiogram explained why myocardial function deteriorated even though the graft was patent.

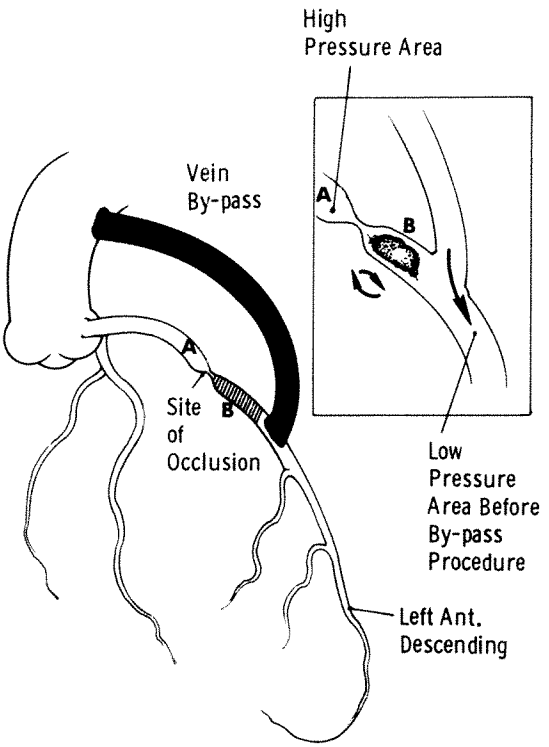


Fig. 8. Schematic explanation of why clotting occurs between the area of stenosis of the coronary artery and the insertion of the graft. The vein by-pass is patent and a clot (crosshatched lines) formed because after graft insertion there is no more gradient between points A and B. The arrows in the inserted drawing represent forces of flow.

(C) *Clotting of a major coronary artery distal to the by-pass.* In one case, we saw clotting of the coronary artery distal to the insertion of the graft with retrograde flow into the proximal segment (Fig. 5). This could be explained either by surgical trauma or the creation of an acute angle between the graft and the distal coronary artery.

(D) *Myocardial infarction as a result of clotting of both the coronary artery and the graft.* This complication may occur in 2 steps as demonstrated in Figure 10, *A–C*. First, there may be progression of an incomplete obstruction of the proximal coronary artery into a total obstruction, as described in Section 2B (Fig. 10*C*). Subsequently, if the graft happens to clot, there is no more flow into the distal portion of that coronary artery and a massive infarction follows.

DISCUSSION

It is important to diagnose the complications which may result from the saphenous-venous by-pass. For an accurate postoperative assessment of the coronary arteries and graft, the angiographic examination must be carried out in proper fashion. The importance of bidirectional flow in the coronary artery[7] cannot be stressed enough. If unidirectional flow is present or if the vein is obstructed, the coronary arterio-

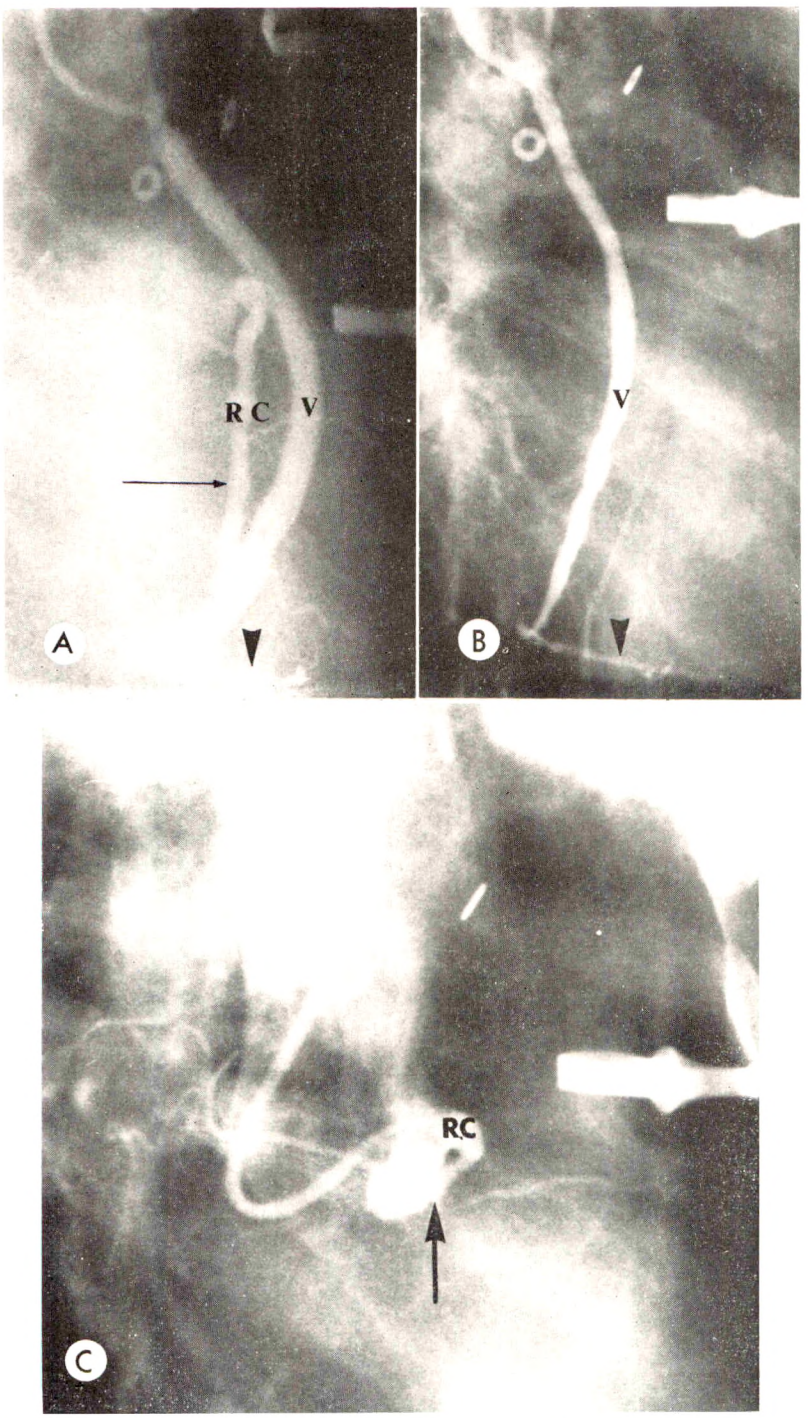


FIG. 9. (*A*) Vein by-pass (V) opacified 2 weeks after surgery. Note that the right coronary artery (RC) fills distally (arrowhead) and proximally (thin black arrow). (*B*) Re-examination 15 months after surgery. The vein (V) is patent but proximal flow into RC is now absent. (*C*) Selective right coronary arteriogram in right anterior oblique projection obtained at the same sitting as *B*. The black arrow points to the occlusion of the right coronary artery (RC). Note that RC was completely patent in *A*.

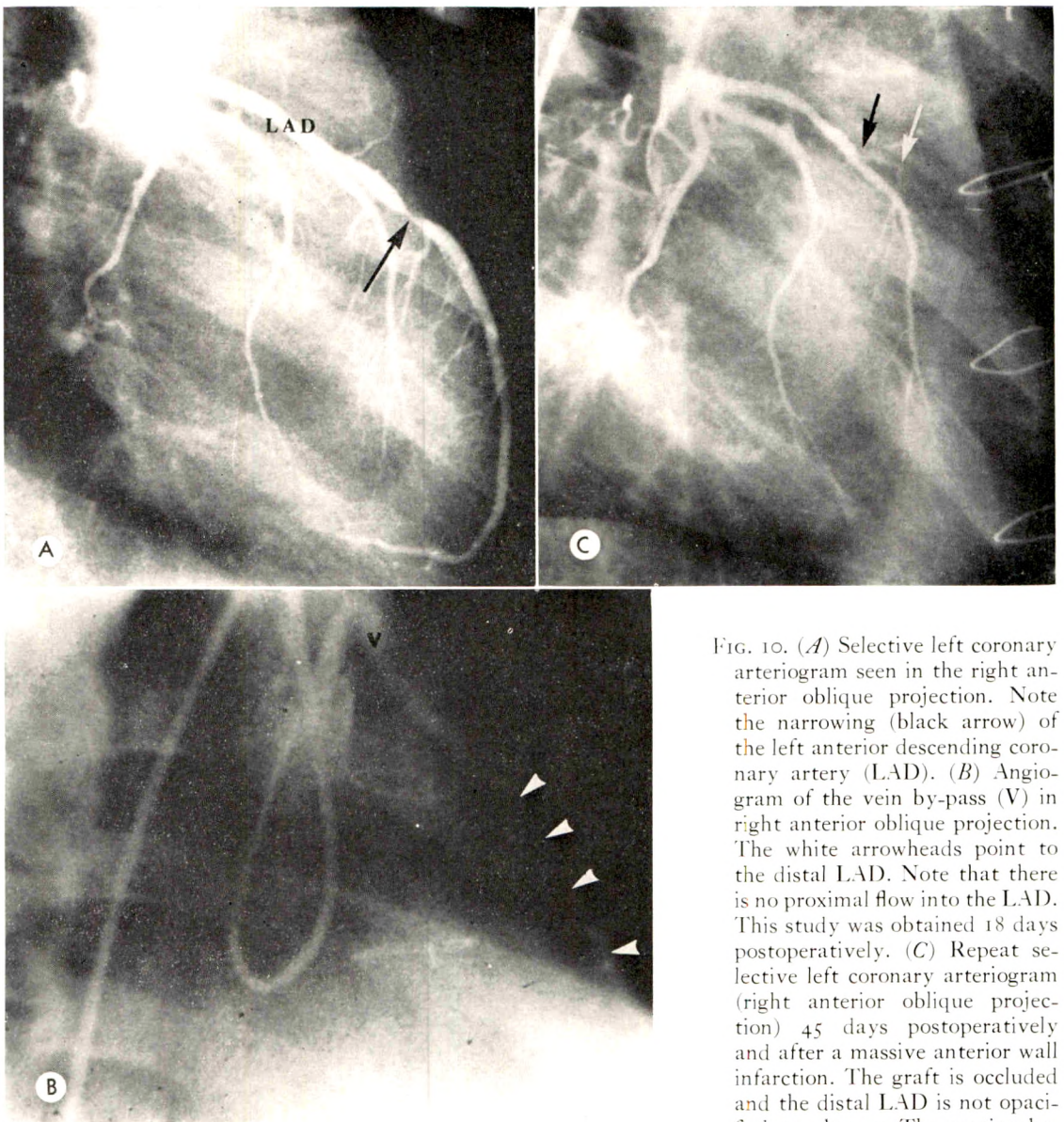


Fig. 10. (*A*) Selective left coronary arteriogram seen in the right anterior oblique projection. Note the narrowing (black arrow) of the left anterior descending coronary artery (LAD). (*B*) Angiogram of the vein by-pass (V) in right anterior oblique projection. The white arrowheads point to the distal LAD. Note that there is no proximal flow into the LAD. This study was obtained 18 days postoperatively. (*C*) Repeat selective left coronary arteriogram (right anterior oblique projection) 45 days postoperatively and after a massive anterior wall infarction. The graft is occluded and the distal LAD is not opacified any longer. The portion between the black and the white arrow is where the proximal clot in the LAD was seen 18 days postoperatively. (This case is also shown in Figure 8.)

gram must be repeated in order to locate the level of obstruction and determine whether the distal coronary segment is still patent.

Deterioration of myocardial function following the by-pass procedure can be due to infarction during the surgical procedure (which happens rather frequently), to progression of the disease, but also to complications related directly to the procedure. Some of these complications can be remedied by surgery; for instance, a clotted graft can be replaced. False aneurysms should be resected. Occlusion of a major coronary artery and loss of collaterals could presumably be treated by a Vineberg procedure.[10]

As a final consideration, the potential occurrence of these complications and their frequency should be considered when a

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patient is submitted to the saphenous vein by-pass procedure.

SUMMARY

Our experience with the postoperative study of 50 saphenous-venous by-pass procedures has shown that a number of postoperative complications might affect either the veins or the coronary arteries.

It is pointed out that the angiographic study must be tailored to properly diagnose and evaluate these complications. Among them one may find: occlusion of the graft; intimal hyperplasia; formation of false aneurysms; loss of coronary collaterals; occlusion of major coronary arteries; or myocardial infarctions.

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ROENTGENOGRAPHIC DETECTION OF CARDIOMEGALY IN EMPLOYEES WITH NORMAL ELECTROCARDIOGRAMS*

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A VOLUNTARY health screening program was begun in September 1968, and during the first year about 26,500 employees above the age of 40 years were examined. The group consisted of 88 per cent males and 12 per cent females, with the majority in the 41 to 50 year age range (Table 1).

TABLE I

AGE DISTRIBUTION; HEALTH SCREENING PROGRAM

Age (yr.)	Males (per cent)	Females (per cent)
41–45	44	34
46–50	27.6	30.8
51–55	15.5	19.4
56–60	8.9	12.3
61–65	4.1	3.3

We performed a battery of tests including: a self administered, machine processable questionnaire; measurement of height, weight and blood pressure; a recording of the nurses' observations; an electrocardiogram; seven blood chemical determinations; and a standard roentgenogram of the chest made at 6 feet. The film was phototimed and exposed as the subject stopped breathing in full, but not necessarily maximum, inspiration.

The electrocardiograms were recorded by the standard 12 lead (classic) system for all persons and also, in about two-thirds of the cases, by the 3 lead Frank (vector) system. The cardiologist read those cases where only the classic leads were available. The vector leads were analyzed by computer using the Electrocardiographic Analysis Program for the IBM 1800 Data Acquisition and Control System, July 1968 version. (This program was developed in a joint effort of the Advanced Systems Development Division of IBM and Dr. Ralph E. Smith of the Mayo Clinic in Rochester, Minnesota.) If the computer interpreted the electrocardiograms as either borderline or abnormal, they were reviewed by the cardiologist and his interpretation overrode the computer's. In addition, the cardiologist reviewed about 25 per cent of the computer normals. Of the 26,500 examined, 25,831 had electrocardiograms; 87 per cent were within normal limits; 6 per cent were borderline; and 7 per cent were abnormal.

Because we had planned to determine the contribution the standard chest film makes toward the detection and elucidation of cardiovascular disorders, the radiologist carefully searched for and noted all evidences of these conditions. This report deals only with the radiologic detection of heart enlargement in individuals having normal electrocardiograms.

The roentgenographic sieve catching instances of cardiomegaly was the eye of the examiner reinforced, when needed, by a determination of the cardiothoracic ratio. We used a percentage equal to or greater than 50 as a broad, yet practical reference. We then gathered all cases detected by this approach through the use of a computer word search (Fig. 1) and selected for analysis only those having a normal electrocardiogram. This yielded a group of 476 individuals, most according to the initial

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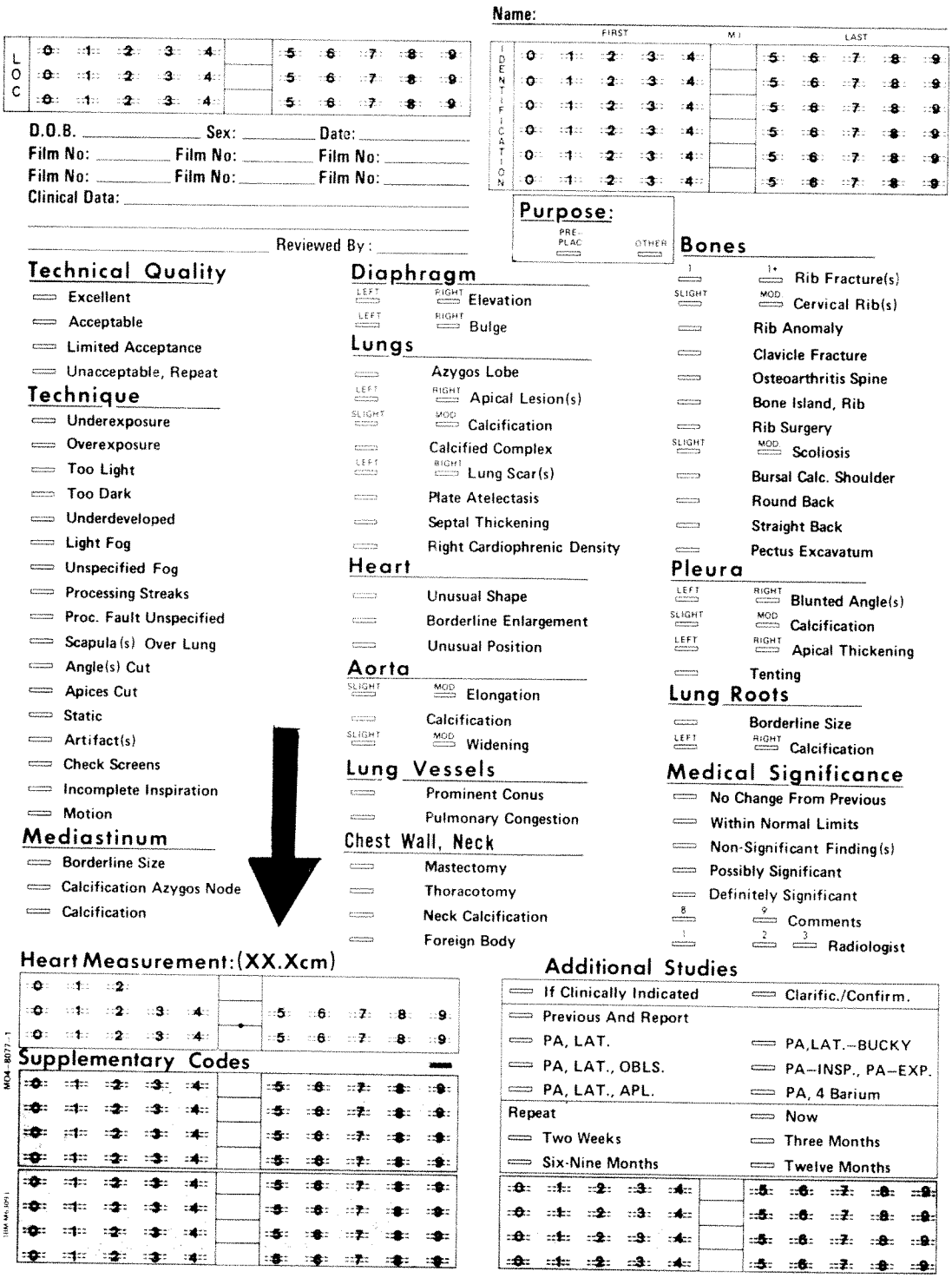


FIG. 1. IBM chest roentgenographic report. Arrow designates the Section of Heart Measurement.

Computer words used in search for cardiac enlargement are: *heart enlargement; cardiac enlargement; heart size; increased size; upper normal; borderline enlargement; borderline size; diameter heart; prominence ventricle;* and *pulmonary congestion.*

dictated report with possibly enlarged hearts, but also including a special group of 80 whose hearts seemed much more likely to be enlarged. We ascertained as a by-product of this word search that approximately twice this number (950 of 3,437 individuals with borderline or abnormal electrocardiographic readings) had film evidence of some degree of cardiomegaly as judged by the radiologist's original interpretation along with an electrocardiographic finding. An inability to regain all films loaned to physicians and to capture all necessary data needed for further, more critical analysis as well as certain inadequacies of the height-weight chart reduced the total number of cases from 476 to a determinate 382.

As the first step in investigating heart size more thoroughly, the radiologist and the cardiologist independently measured the transverse cardiac diameter on each film of the 382 employees. Then the measurement was matched to the appropriate figure in the Ungerleider-Clark, height-weight chart of predicted diameter.[6,7] We could then assign each case to a category; no enlargement, 5, 10, 15, 20, 25 per cent above normal. If the measurement was closer by 1 mm. to one listing in the chart than another, then we placed the case in that slot. When the figure fell equally between two categories we always put the case in the lower degree of enlargement.

We combined the individuals falling into the 5 per cent and the 10 per cent groups into a probable enlargement cluster which totalled 175. The 82 comprising the 15, 20, 25 per cent categories were considered to be definitely enlarged.[2] The ones remaining, 125, were not confirmed as having enlarged hearts. Table II shows these results.

About 1 year after the evaluation made on 61 of the original group of 80 (referred to previously as those thought most likely to have heart enlargement on the basis of the radiologist's initial report), we reinvestigated those among the 61 whom we found by the height-weight method to have possibly (5 to 10 per cent) and definitely (15, 20, 25 per cent) enlarged hearts. Another chest roentgenogram was obtained; it was measured as before and then we compared the results with the previous finding. We determined that in this special re-examined group numbering 46, the hearts of 16 were larger, 5 were the same and 25 were smaller. The greatest individual increase was 12 mm. and the greatest decrease was 17 mm. We did not reweigh or re-determine the height of these employees at the time of the second roentgenogram, but used the figures for weight and height recorded about 1 year previously.

TABLE II

TABULATION OF THE RESULTS OF DETERMINING THE PREDICTED HEART SIZE FROM HEIGHT-WEIGHT CHART AND THE TRANSVERSE DIAMETER MEASURED FROM THE SIX FOOT STANDARD CHEST ROENTGENOGRAM

Normal	5%	10%	15%	20%	25%
125	89	86	56	18	8
Total cases 15% or greater				82	
Total cases 10% or greater				168	

DISCUSSION

To the degree that the height-weight technique of determining heart size from the 6 foot posteroanterior chest roentgenogram is to be relied upon, the data obtained by this method validate the conclusion that the standard chest roentgenogram reveals a significant number of employees with cardiomegaly who escape detection by electrocardiographic screening. The additional contribution of the chest roentgenogram in documenting certain structural features of an existing cardiovascular disorder already brought to the physician's attention requires no emphasis. Of the screening tests commonly used, only the chest roentgenogram, the electrocardiogram, and the physical examination provide direct, positive evidence of anatomic and physiologic abnormalities of cardiovascular nature, offering vital information as to the nature and extent of the disorder.

Although direct visual identification of most of the moderately and all of the definitely enlarged hearts causes no problems for the radiologist, a clear and easy differentiation between the normal and the slightly enlarged organ creates constant difficulty. Many standard texts and classic articles[1,3,6,7] have dealt thoroughly with cardiac mensuration. It does seem generally agreed that the transverse diameter of the heart on the 6 foot standard chest roentgenogram exposed at the end of moderate inspiration and compared to the predicted diameter from the Ungerleider-Clark height-weight chart remains the simplest, most practical approach as well as one of the more reliable. Any result obtained by this method falling into the 15 per cent and higher categories indicates true cardiomegaly.[2] Some authors also state that any percentage of 10 or more above the predicted normal should be considered enlarged, since very few subjects with normal hearts exceed this figure.[6,7]

Gammill *et al.*[3] demonstrated the effect of systole and diastole upon cardiac diameter. When measurements in each phase were compared, 52 per cent of the subjects showed differences of .3 cm. or less, 41 per cent had variations of .4 cm. to .9 cm. and only 7 per cent revealed discrepancies amounting to 1 to 1.7 cm. A number of years ago, one of us investigated the influence of various technical factors of the chest roentgenogram upon the transverse diameter of the heart. Of all, the most significant was found to be the degree of inspiration. Particular attention should be paid to this point before measurements become fully reliable. In selected instances, care must also be taken that the true heart apex be identified accurately when this area makes up the outermost left heart border.

Cardiologists agree that the truly enlarged heart means a diseased organ.[2,7] Cardiac enlargement offers contributory evidence for the identification of the coronary prone individual, and in asymptomatic persons of this category the onset of myocardial involvement may be heralded by this event.[4] The detection of cardiomegaly warrants referral of the asymptomatic worker for further investigation and possibly for admission to a prophylactic routine. The long range significance of roentgenologically detected heart enlargement when other survey procedures including the electrocardiogram are normal remains undetermined. Nevertheless, these individuals certainly harbor a potentially significant disorder, be it coronary, valvular or myocardial in origin.

Recognizing the potential of the routine standard chest roentgenogram as a means of contributing to the detection and elucidation of cardiovascular disease, we strongly felt the need to improve on the customary method (cardiothoracic ratio) of roentgenographic heart measurement. At the completion of this study a "mark sense requisition and reporting sheet," optically read by machine was devised[5] (Fig. 1). We anticipate that a major innovation of this system will enable the computer to derive the percentage deviation from the predicted normal by using an algorithm based upon the employee's height and weight. Whenever the radiologist suspects the possibility of cardiomegaly, he measures the actual transverse diameter of the heart and records this figure on the mark sense sheet. Since height and weight are available to the computer, this act will cause it to perform the necessary calculations and subsequently to print the percentage deviation from the normal predicted cardiac diameter on the report. With high volume work, the radiologist can save time by merely indicating right and left outermost cardiac borders by wax pencil on the film and a clerk can then measure and record the cardiac diameter on the mark sense sheet.

SUMMARY

Among 22,394 employees all over 40 years of age with normal electrocardiograms, we discovered 82 (approximately 3

per 1,000) with cardiomegaly as determined from life size chest roentgenograms. We used the predicted cardiac diameter as based upon height and weight as our standard. If our cut-off point for establishing enlargement was dropped from the 15 per cent level to 10 per cent then the yield would have been twice this figure.

A recently devised mark sense requisition reporting sheet will enable the radiologist to obtain the deviation from the expected normal predicted cardiac diameter from the computer simply by recording the actual measurement upon the mark sense sheet.

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We acknowledge with gratitude the help of the staff of the IBM Medical Data Center.

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AORTICOPULMONARY TUNNEL: ANGIOGRAPHIC RECOGNITION OF PULMONARY ATRESIA AND CORONARY ARTERY-TO-PULMONARY ARTERY FISTULA*

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THE pulmonary blood supply in pulmonary atresia is usually through a patent ductus arteriosus or multiple bronchial collaterals. Rarely,[1] the pulmonary blood supply is through a coronary artery-to-pulmonary artery fistula. We are aware of 2 cases of pulmonary atresia and large coronary artery-to-pulmonary artery fistula previously reported in which cardiac catheterization including angiography was done.[2,3] In 1 of these 2 cases the proper diagnosis was reportedly made by use of angiography.[3] The correct diagnosis in the second case was made at surgery.[2]

This report presents the angiographic findings in 4 cases of pulmonary atresia and coronary artery-to-pulmonary artery fistula seen at the Mayo Clinic. One of these has been previously reported.[2] In all 4 cases, there was a unique angiographic appearance typical of this anomaly.

REPORT OF CASES

Case I. Simultaneous anteroposterior and lateral views of a selective right ventricular angiogram showed early filling of the aortic root through a ventricular septal defect (Fig. 1, *A* and *B*). A dilated proximal coronary artery connecting the aortic root to the pulmonary artery was well visualized in both views. Subsequently, the pulmonary artery was visualized through this communication (Fig. 1*C*). The exact nature of the aorticopulmonary communication was not apparent from that view. The catheter was placed in the proximal dilated coronary artery, and an injection was made with the catheter tip in the fistulous communication (Fig. 1, *D* and *E*). There was selective filling of the pulmonary artery back to the pulmonary valve (Fig. 1*E*). Although most of the contrast medium was injected preferentially into the pulmonary artery, simultaneous filling of a small distal left coronary artery also was seen (Fig. 1*D*, arrow). There was a side-to-side relationship of the left coronary artery to the pulmonary artery communication (aorticopulmonary tunnel) (Fig. 1, *D* and *E*).

Case II. An anteroposterior view of a right ventricular biplane angiogram revealed an atretic right ventricular infundibulum, with complete atresia of the pulmonary valve (Fig. 2*A*, white arrow). There was filling of a right aortic arch *via* a ventricular septal defect. Contrast medium entered the pulmonary artery *via* a dilated proximal coronary artery (black arrow).

Simultaneous anteroposterior and lateral views of an aortic root injection revealed an aorticopulmonary tunnel (black arrow) connecting the ascending aorta to the pulmonary artery (Fig. 2, *B* and *C*). This communication was a dilated proximal part of the left coronary artery extending from the aortic root to the fistulous tract. The left distal coronary artery was not visualized, and the blood supply to the heart was mainly through a large right coronary artery (Fig. 2*C*, white arrow).

Case III. Simultaneous anteroposterior and lateral views of a selective right ventricular injection showed no direct communication between the right ventricle and the pulmonary artery (Fig. 3, *A* and *B*). The pulmonary artery was filled through a coronary artery-to-pulmonary artery fistula (arrow), as well as by a right subclavian-to-right pulmonary artery anastomosis (Blalock-Taussig operation). In the lateral view, the aorticopulmonary tunnel was almost visible end on (Fig. 3*B*, arrow).

Case IV. Anteroposterior and lateral views of a selective biplane aortogram revealed an

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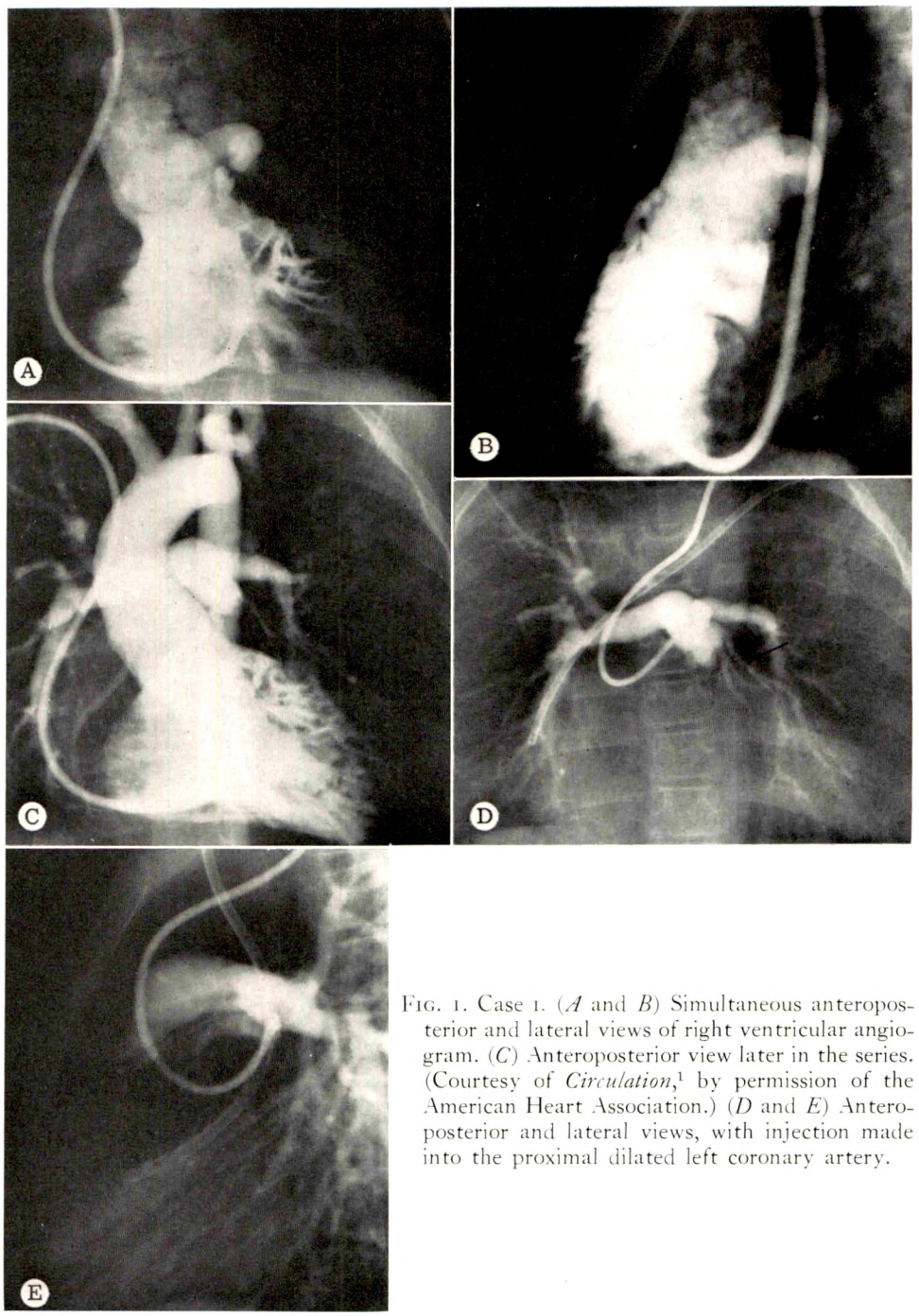


Fig. 1. Case 1. (*A* and *B*) Simultaneous anteroposterior and lateral views of right ventricular angiogram. (*C*) Anteroposterior view later in the series. (Courtesy of *Circulation*,[1] by permission of the American Heart Association.) (*D* and *E*) Anteroposterior and lateral views, with injection made into the proximal dilated left coronary artery.

aorticopulmonary tunnel (Fig. 4, *A* and *B*, arrows), although the anatomic configuration is slightly different from that in the other 3 cases. The proximal part of the left coronary artery was dilated, and its diameter was nearly the size of the hypoplastic pulmonary artery.

DISCUSSION

In all 4 cases, the diagnosis of pulmonary atresia, ventricular septal defect, and left coronary artery-to-pulmonary artery communication was confirmed at surgery.

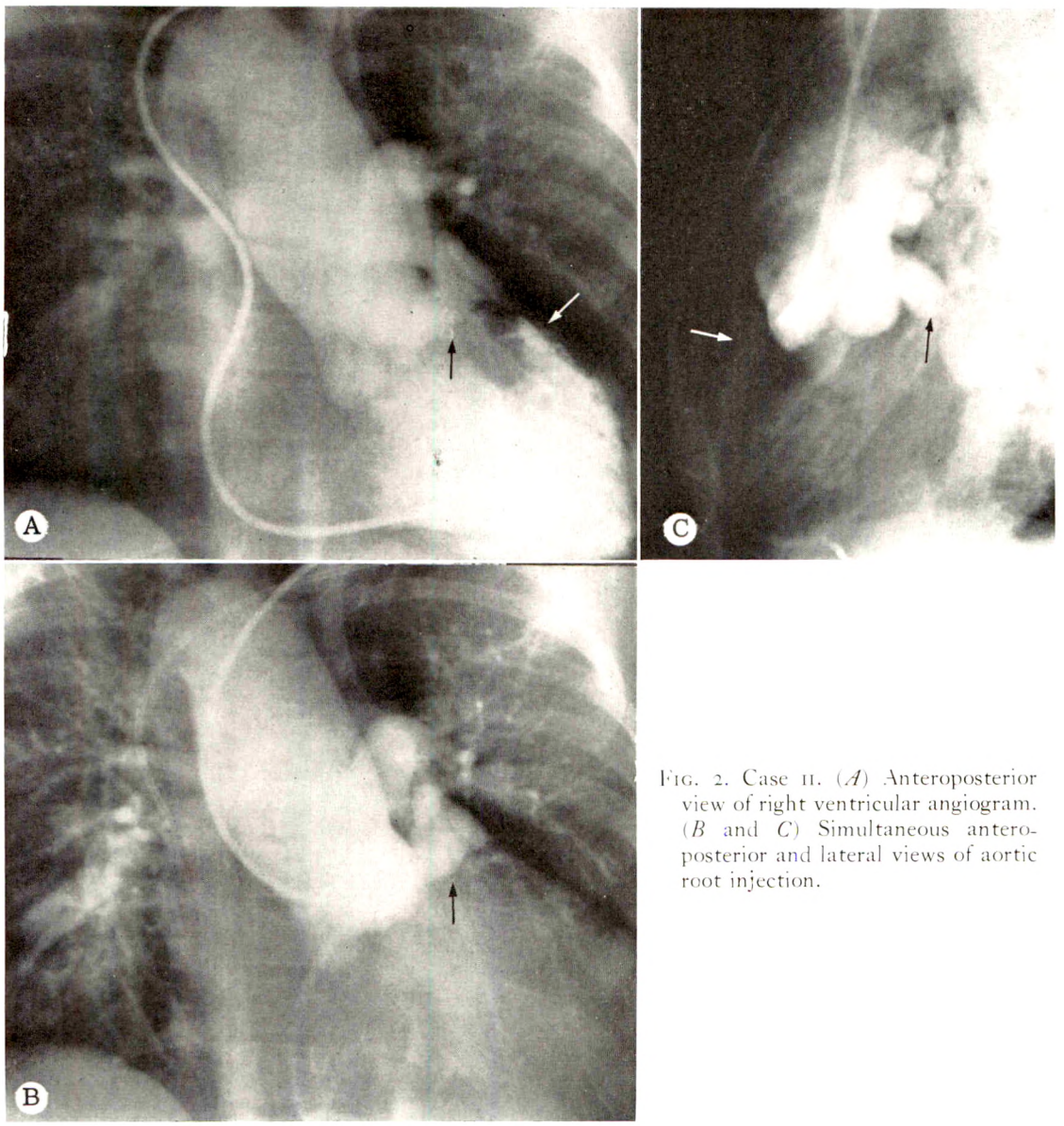


FIG. 2. Case II. (*A*) Anteroposterior view of right ventricular angiogram. (*B* and *C*) Simultaneous anteroposterior and lateral views of aortic root injection.

The pulmonary blood flow was principally provided by the side-to-side fistula between the left coronary artery and the pulmonary artery. All cases had a pathognomonic aorticopulmonary communication consisting of a wide proximal dilated left coronary artery connecting the aortic root to the pulmonary artery. We believe that this typical angiographic picture is diagnostic of pulmonary atresia and coronary artery-to-pulmonary artery fistula.

The differential diagnosis of pulmonary atresia and coronary artery-to-pulmonary artery fistula mainly concerns the 2 other types of ascending aorta-to-pulmonary communication, namely aorticopulmonary window and truncus arteriosus. In aorticopulmonary window, there is a direct communication, or window, between the ascending aorta and the pulmonary artery. The communication is often large, and there is pulmonary hypertension and dilatation of the pulmonary artery.

The pulmonary artery pressure was nor-

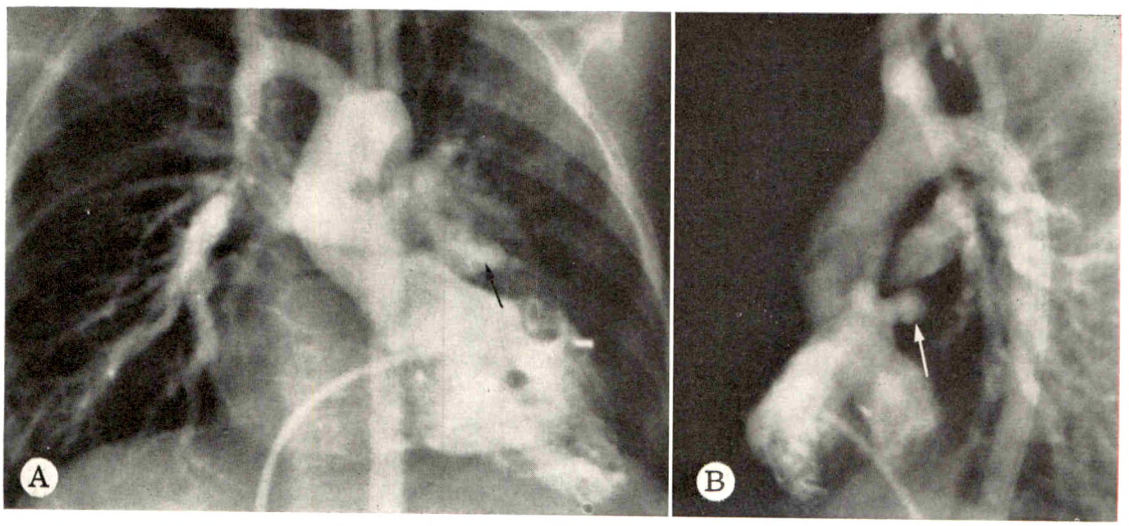


Fig. 3. Case III. (*A* and *B*) Simultaneous anteroposterior and lateral views of right ventricular angiogram.

mal-to-low in all 4 of our cases. The pulmonary artery was hypoplastic in 3 of the 4 (Cases I, III, and IV). The finding of a hypoplastic pulmonary artery is unusual in patients with aorticopulmonary window and should alert the radiologist to the possible presence of a coronary artery-to-pulmonary artery fistula. The documentation of an "aorticopulmonary tunnel" in these patients excludes the diagnosis of aorticopulmonary window.

In truncus arteriosus, there is absence of a separate main pulmonary artery, absence of a right ventricular infundibulum, and

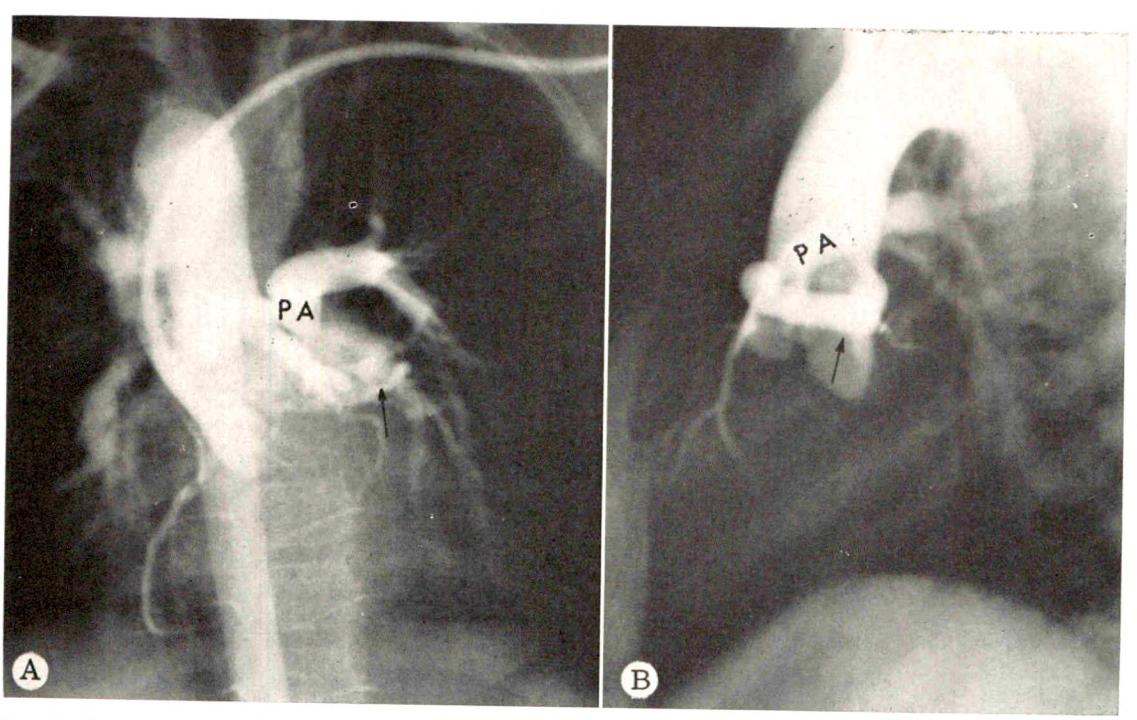


Fig. 4. Case IV. (*A* and *B*) Anteroposterior and lateral views from aortic root angiogram. Aorticopulmonary tunnel is particularly well shown in lateral film. PA = pulmonary artery.

presence of a single truncal valve. Demonstration of a separate main pulmonary artery, a hypoplastic right ventricular infundibulum, or a hypoplastic pulmonary valve excludes the diagnosis of truncus arteriosus.

The nature of the aorticopulmonary communication may not be apparent after a right ventricular injection (Fig. 1*C*). A selective injection into the aortic root or preferably into the proximal part of the left coronary artery, as done in one of these cases, allows for better definition of the aorticopulmonary tunnel in patients with pulmonary atresia and coronary artery-to-pulmonary artery fistula.

SUMMARY

The angiographic findings in 4 cases of pulmonary atresia and coronary artery-to-pulmonary artery fistula are described and their diagnostic significance discussed.

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ANGIOGRAPHIC ANATOMY OF THE KUGEL'S ARTERY*

ARTERIA ANASTOMOTICA AURICULARIS MAGNA

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KUGEL'S artery[3] is a small vessel running through the atrial septum which links the anterior coronary arteries to the posterior ventricular coronary tree.

According to the original paper of Kugel and Gross,[4] this artery presents 3 anatomic variations: In the majority of cases it originates from the circumflex artery, penetrates the left atrial wall and proceeds backward through the inferior border of the atrial septum to the crux. Occasionally, Kugel's artery originates from the sinus node artery (Fig. 1*A*). In the second variation, Kugel's artery is formed by a branch of the right coronary artery and a branch of the circumflex artery, which join near the atrioventricular node artery (Fig. 1*B*). In the third variation, which is less frequent (4 in 50 cases of Kugel's series), the artery originates from the left circumflex artery and splits into smaller branches, which connect to similar small vessels coming from the right coronary artery at the crux level (Fig. 1*C*).

To demonstrate coronary anastomoses, technical factors and the size of the vessels are of critical importance. The use of large films in selective coronary angiography has achieved the visualization of 100 μ diameter vessels. Even though the artery is usually not as large as stated by Kugel,[3] it should be demonstrable by selective coronary arteriography.

MATERIAL AND METHOD

One hundred consecutive pathologic coronary angiograms obtained by the Judkins technique using large film as well as 35 mm. cineangiography were reviewed. The arteria anastomotica auricularis magna was found in 6 cases. The coronary disease in these cases was: (a) occlusion or severe stenosis of the right coronary artery in 5 cases; (b) occlusion or severe stenosis of the circumflex artery in 6 cases; and (c) combined lesions in 5 cases.

The patency of the distal right coronary artery at the crux level was preserved in all cases. Kugel's artery originated from the right coronary artery in 3 cases and from the same source but through the sinus node artery in 3 cases (Fig. 1*B*).

The angiographic visualization of Kugel's artery has not been clearly demonstrated. Paulin[5] described a small vessel connecting the right proximal coronary artery with the distal segment of the same artery and called it a branch of the crista supraventricularis artery. In most projections Kugel's artery in the atrial septum is frequently overlapped by the right coronary and circumflex arteries as they course around the atrioventricular groove.

To clarify the roentgenologic appearance of Kugel's artery in coronary angiograms, we did a postmortem study of a heart injected with contrast medium into the coronary artery tree. We placed wires in the course of the Kugel's and the sinus node arteries. Roentgenograms were taken in lateral, left and right anterior oblique projections. The roentgenograms and the drawings of them are presented.

In the lateral projection Kugel's artery is seen anterior to the circumflex artery as a small vessel coursing to the crux (Fig. 2, *A* and *B*). The circumflex artery usually overlaps it.

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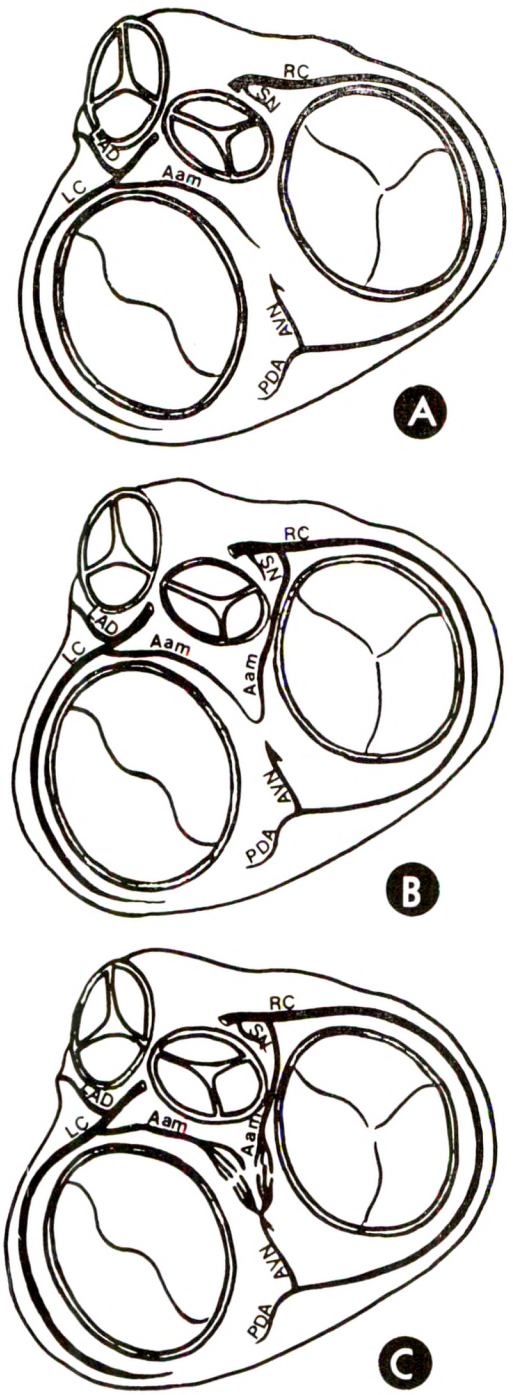


FIG. 1. (*A–C*) The 3 anatomic variations of arteria anastomotica auricularis magna modified from Kugel.

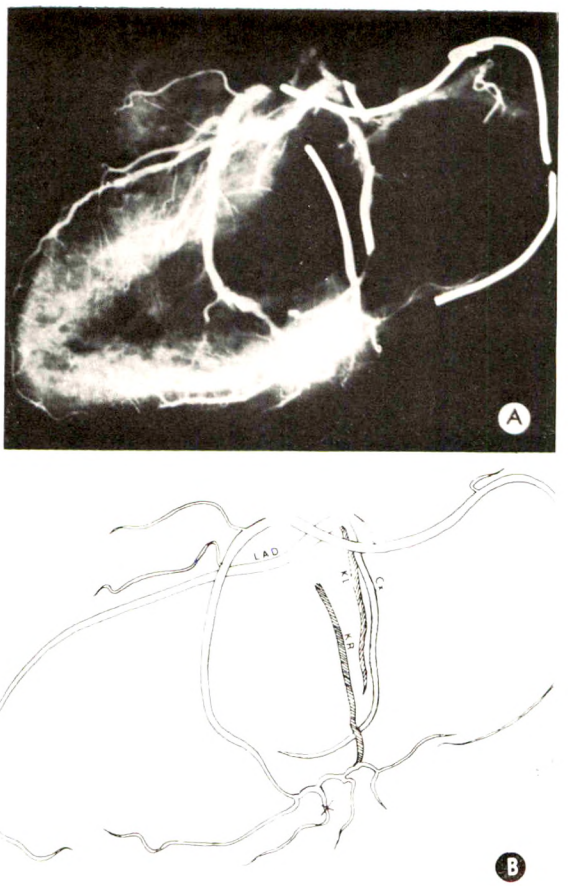


FIG. 2. (*A* and *B*) Roentgenogram and corresponding drawing in lateral projection from a fixed heart with contrast material in the coronary arterial tree. The metallic wires are placed in the position of Kugel's and sinus node arteries. Kugel's artery from the right (KR) and from the left (KL) overlap the circumflex artery (Cx). Left anterior descending artery (LAD).

In the right anterior oblique view Kugel's artery is seen overlapping the right or circumflex arteries (Fig. 3, *A* and *B*).

The left anterior oblique view shows the atrioventricular circle of the heart *en face*. Kugel's artery appears as a diameter of that circle (Fig. 4, *A* and *B*).

DISCUSSION

Due to the anatomic situation of Kugel's artery, the best roentgenologic projection is

the left anterior oblique. In this view the artery progresses downward from the anterior third of the atrioventricular plane to the crux where it often connects directly with the atrioventricular node artery (Fig. 4, *A* and *B*; and 5, *A* and *B*). This anastomosis is recognized by a flaglike rectangular termination.

The sinus node artery has a slightly higher course directed upward towards the atria. Here it usually has an open arm-appearance, where it encircles the superior vena cava (Fig. 4, *A* and *B*). When Kugel's artery comes from the left, it crosses the lower half of the atrioventricular plane and is overlapped by the marginal and diagonal branches.

In the lateral projection the atrioventricular plane is angulated towards the beam

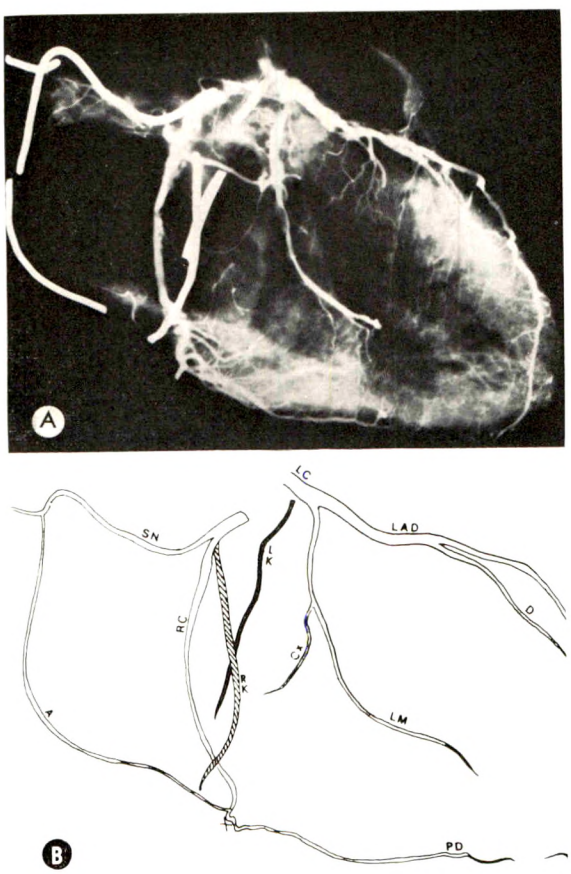


Fig. 3. (*A* and *B*) Roentgenogram and drawing in right anterior oblique (RAO) projection from the same heart as in Figure 2, showing the position of Kugel's artery (KR and KL) close to the right coronary artery (RC) and circumflex artery. The sinus node artery (SN) is projected posteriorly.

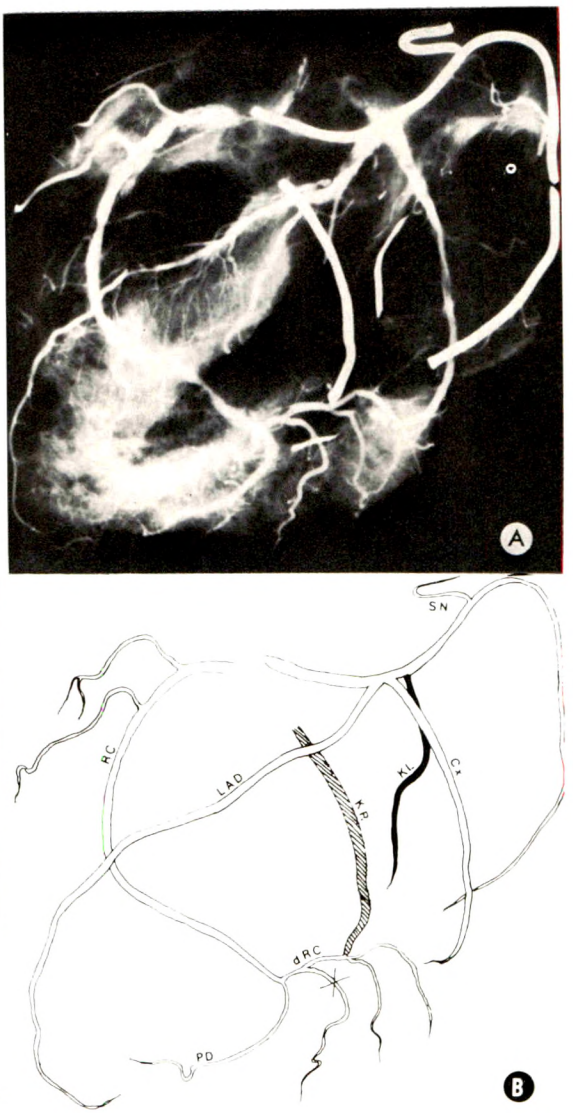


Fig. 4. (*A* and *B*) Roentgenogram and drawing in left anterior oblique (LAO) projection from the same heart showing the position of Kugel's artery free from the right coronary (RC) and circumflex arteries (Cx). The sinus node artery (SN) is projected backward.

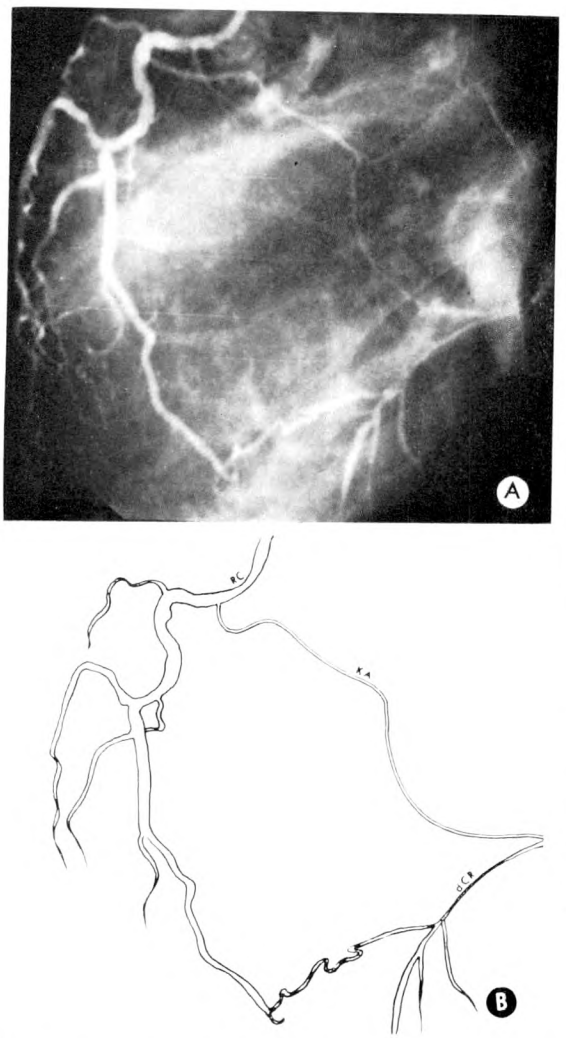


FIG. 5. (*A* and *B*) Right coronary angiogram in left anterior oblique projection. The right coronary and circumflex arteries are occluded. Kugel's artery (KA) is crossing from the proximal part of the right coronary artery to the distal segment of the right coronary system. The crux is seen later.

and appears foreshortened. Kugel's artery traverses the plane at the periphery in the upper third and turns downward to the crux. The sinus node artery again is higher, has an upward course and is projected far beyond the atrioventricular plane (Fig. 2, *A* and *B*; and 6, *A* and *B*). When the arteria anastomotica auricularis magna originates from the left, it will be projected over the left circumflex.

Because the atrioventricular plane is more foreshortened in the right anterior oblique projection, Kugel's artery is projected over the right coronary artery. When it originates from the left, Kugel's artery crosses the atrioventricular plane in the middle but it is difficult to differentiate it from other overlapping atrial vessels (Fig. 3, *A* and *B*; and 7, *A* and *B*).

All cases had a markedly reduced blood flow at the crux level due to right coronary

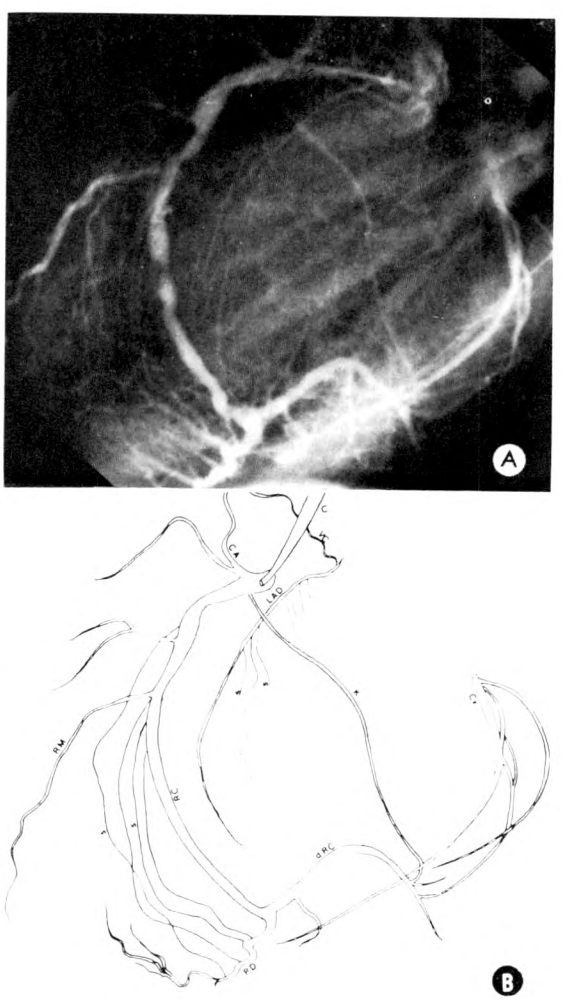


FIG. 6. (*A* and *B*) Right coronary angiogram in lateral projection. The Kugel's artery (K) is seen originating from the right coronary (RC) and courses to the crux. The occluded circumflex artery (Cx) has been opacified in retrograde direction.

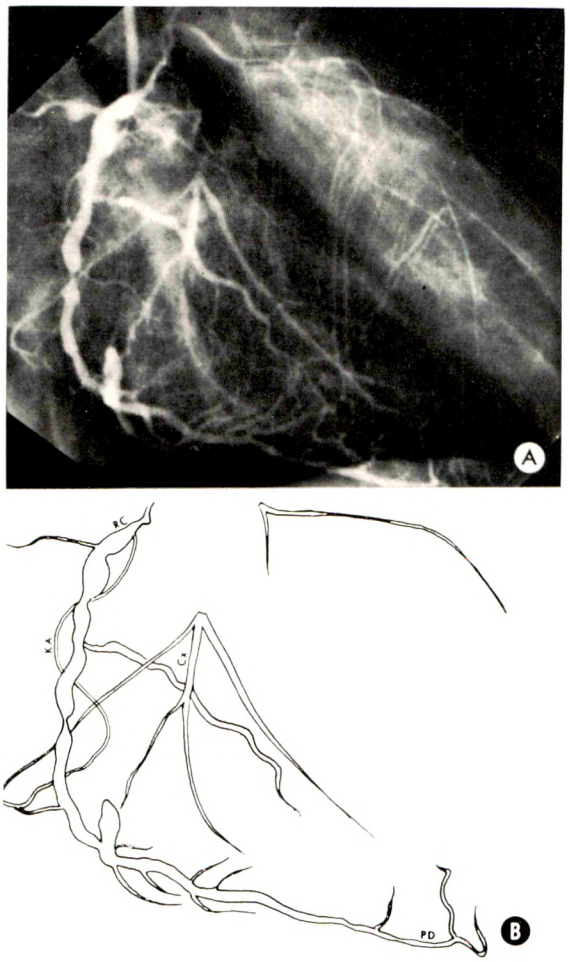


Fig. 7. (*A* and *B*) Right coronary angiogram in right anterior oblique projection. The Kugel's artery (KA) is overlapped by the right coronary artery (RC). The occluded circumflex artery (Cx) is opacified in retrograde direction.

artery and/or circumflex artery obstruction. This fact suggests that Kugel's artery may function as an appropriate collateral channel.

SUMMARY

The roentgenographic identification of the arteria anastomotica auricularis magna (Kugel's artery) is reported in 6 cases with coronary disease.

The different projections used for visualizing Kugel's artery are described and the differentiation from the sinus node artery is discussed.

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ABDOMINAL AORTOGRAPHY FROM THE AXILLARY APPROACH*

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PERCUTANEOUS axillary artery catheterization for angiography of the vertebral and carotid arteries and the aorta is a relatively easy maneuver.[1-4] However, in the older age group it is frequently difficult to pass the catheter into the descending thoracic aorta for abdominal aortography.[7] Some methods described to overcome this problem include reflection of a guidewire off the aortic valves,[3] deflector catheters,[2] double catheter techniques,[1,5] and use of curved catheters and guidewires.[4,6]

This communication describes a simple method of accomplishing catheterization of the descending thoracic aorta using regular catheters and guidewires, as well as a method of obtaining technically good aortographic studies in the occasional patient in whom the guidewire and catheter cannot be advanced distal to the aortic arch.

INDICATIONS

Angiographers use femoral, translumbar, and axillary approaches depending on the status of the pulses and objectives of the procedure, as well as their experience and personal preference. Puncture of the femoral artery is most commonly used and any arteriographic study can be performed with this technique. When the femoral pulses are weak or absent, the translumbar or axillary approaches must be utilized. Translumbar aortography can be used for abdominal aortography, whereas the axillary artery catheterization is necessary for thoracic aorta and brachiocephalic arteriography. Many angiographers, however, prefer not to use the translumbar approach for abdominal aortography, and this communique is most applicable to their practice.

METHOD

We usually utilize a Green Kifa (7.5 French) or Yellow Squibb (7 French) catheter. Two catheters are prepared. One is straight and has 4 side holes (Fig. 1*A*), and the other has a 360° or more rather tight distal curve (approximately 1 cm. radius) and also contains 4 side holes (Fig. 1*B*). A 145 cm. J-tipped removable core teflon coated guidewire is used, with the core adjusted so that approximately the distal 6 inches of the guidewire is flexible (Fig. 1*C*).

The percutaneous left axillary artery catheterization is performed by the Seldinger technique.[1-4] If the guidewire initially enters the descending aorta, the straight catheter (Fig. 1*A*) is introduced and aortography is performed. In tortuous aortas, however, the guidewire often enters the ascending aorta, and cannot be advanced into the descending aorta. In such an instance, the curved catheter is inserted. Because of the acute angle between the subclavian artery and the aortic arch, an extreme distal curvature of the catheter is necessary to assure that it can be aimed posterosuperiorly over the aortic arch. The catheter curve can be altered *in situ* by withdrawing it into the left subclavian artery (Fig. 2, *A* and *B*), until the position of the catheter will result in its being aimed over the aortic arch. The guidewire within the catheter is then advanced over the arch and well into the descending thoracic aorta (Fig. 2*C*). Its long flexible portion allows it to pass over the arch without straightening the catheter. While observing the tip of the guidewire in the distal aorta fluoroscopically, the catheter is removed, simultaneously advancing the guidewire further into the aorta so that it is not

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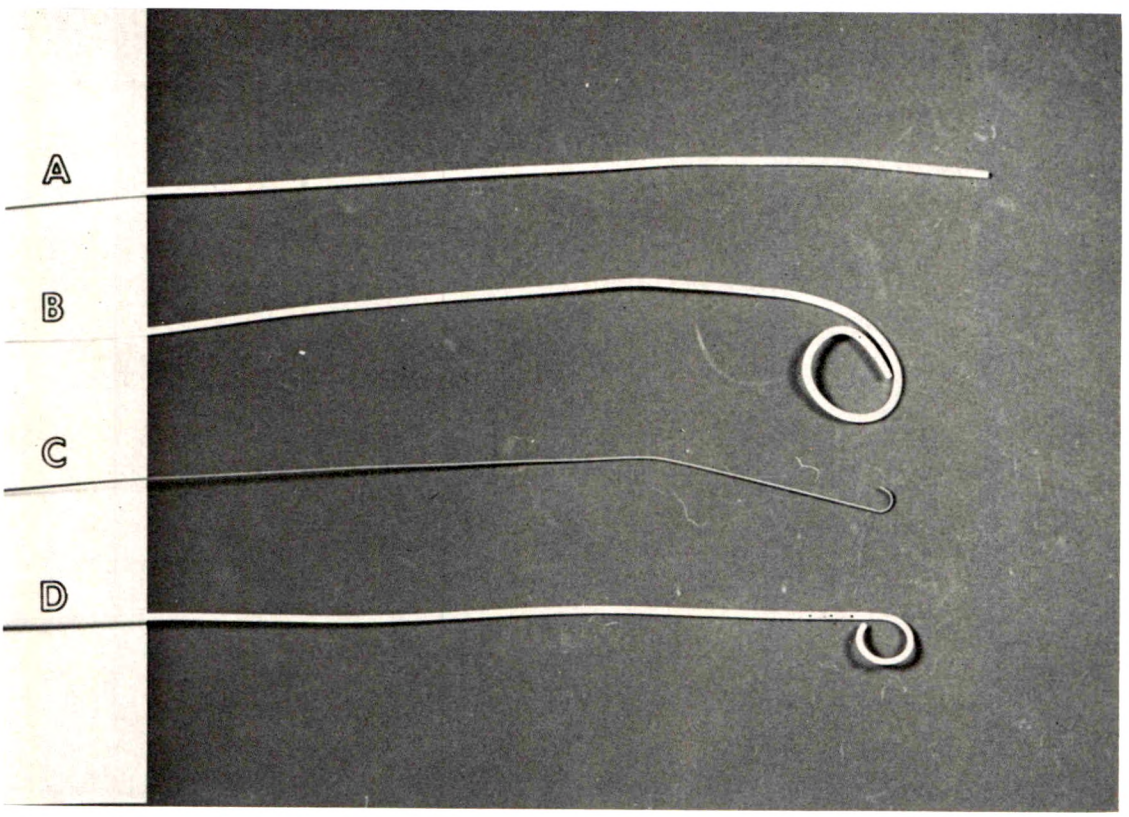


Fig. 1. (*A*) Straight 7–7.5 French catheter with 4 side holes; (*B*) same catheter with 360° end curve; (*C*) 3 mm. radius J-tipped adjustable core guidewire; (*D*) pigtail catheter.

withdrawn to the point where it slips into the ascending aorta. The straight catheter is then inserted and will follow the guidewire into the descending thoracic and abdominal aorta (Fig. 2*D*). Selective injections can subsequently be performed with the same or selective catheters.[6]

In young and middle aged patients, with only moderate elongation of the aorta, a pigtail type catheter (Fig. 1*D*—Cook or Cordis) may be used to aim the guidewire over the distal aortic arch. In this case, the pigtail catheter can then be advanced over the guidewire into the distal aorta, and aortography can be performed without having to change catheters (Fig. 2, *E* and *F*). The use of a single catheter is a significant advantage if the predicted elongation of the aorta is not great. The curve on the pigtail catheter, however, is not as great as the catheter demonstrated in Figure 1*B*, and may not negotiate the more tortuous aortic arches of older patients. In this case, the curved catheter (Fig. 1*B*) as described, should be used for this maneuver. The pigtail catheter with the side holes proximal to the curve has the same disadvantage of other ascending aortic injections for abdominal and femoral arteriography (Fig. 3*B*), in that contrast medium is unnecessarily injected into the brachiocephalic circulation, and detracts from the quality of the abdominal aortogram.

Rarely, because of advanced atherosclerotic plaques or extreme tortuosity of the arch, it may not be possible to pass the guidewire into the descending thoracic aorta. In this case, the curved catheter with 4 side holes (Fig. 1*B*) is simply left in place and the aortography is performed with this catheter. Because of its curve, most of the contrast material bolus enters

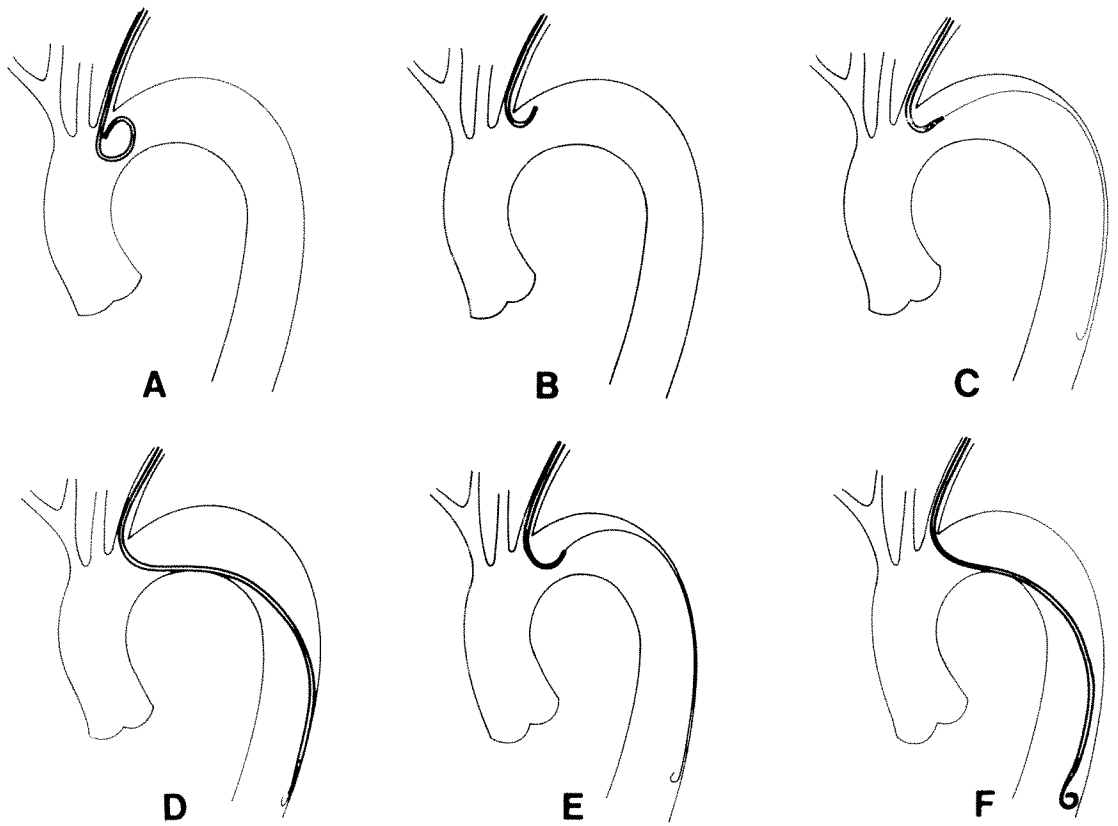


FIG. 2. (*A*) Catheter from Figure 1*B in situ*; (*B*) withdrawal of catheter will straighten the curve so the tip points in the desired direction; (*C*) the guidewire is advanced into the distal descending thoracic aorta; (*D*) a straight (Fig. 1*A*) catheter is then exchanged for the curved catheter and advanced into the descending thoracic or abdominal aorta; (*E*) a pigtail catheter may be satisfactory in the less elongated arches of middle aged patients; (*F*) the pigtail catheter can then be advanced distally and aortography safely done avoiding catheter exchange.

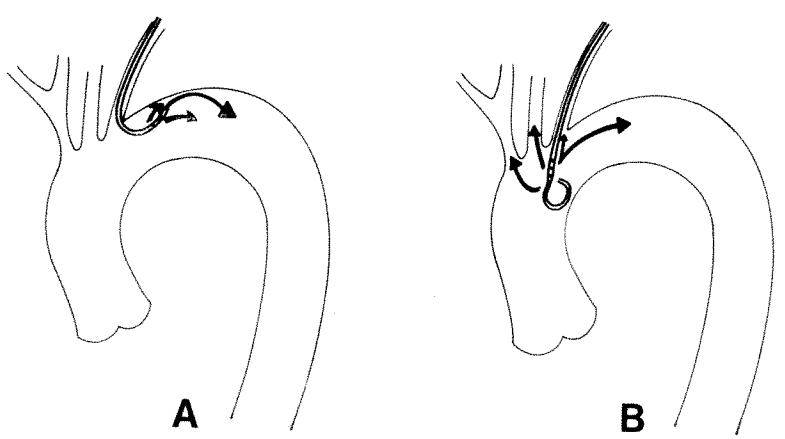


FIG. 3. (*A*) A Figure 1*B* catheter in place. If the guidewire cannot be advanced distally, aortography is done through this same catheter and most of the injected contrast medium enters the distal aorta; (*B*) a disadvantage of the pigtail catheter is that if the guidewire cannot be passed distally, abdominal aortography will be less satisfactory because some of the contrast medium enters the brachiocephalic vessels (arrows).

the distal aorta through the tip hole (Fig. 3*A*). The quality of the angiogram using this technique is considerably better than after injection into the ascending aorta with part of the bolus entering the brachiocephalic vessels. In addition, an injection of excessive amounts of contrast material into the cerebral circulation is avoided.

SUMMARY

A simple method of performing abdominal aortography from the left axillary approach is described, which is successful in most older patients with elongated, tortuous aortic arches.

No additional equipment is necessary.

A satisfactory alternative method is available on the occasional patient in whom the guidewire cannot be advanced distally over the aortic arch.

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ROENTGENOGRAPHIC DIAGNOSIS OF SINGLE VENTRICLE

ANALYSIS OF FORTY-TWO CASES

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SINGLE ventricle is a rare congenital malformation of the heart, and frequently associated anomalies of the heart and great vessels further complicate the condition. The abnormal anatomy of the malformation can be observed only by angiocardiography.

Recently, corrective surgery has been attempted at some centers and thus it seems necessary to be able to identify accurately the ventricular anatomy.

In this presentation, angiograms are analyzed in detail, and a new angiographic classification of the malformation is proposed.

DEFINITION AND CLASSIFICATION

The diagnosis of single ventricle is made according to the definition of Van Praagh *et al.*[14]: "A single or common ventricle is one ventricle, which receives both the tricuspid and mitral valves, or a common atrioventricular valve. This definition excludes tricuspid and mitral atresia."

From the results of pathologic analysis, Van Praagh *et al.* divided the single ventricle anomaly into 4 types, A, B, C and D, depending on which part of the ventricular structure was absent. This classification was not clinical, but based on the pathologic anatomy. According to Van Praagh *et al.*,[15] Type B and D cannot be differentiated by contrast examination.

Hallermann *et al.*[4] divided the malformation into two groups, corresponding to Van Praagh's Type A and Type B, C, D, because Type B, C, and D cannot be differentiated by angiocardiographic procedure. We have found, however, that

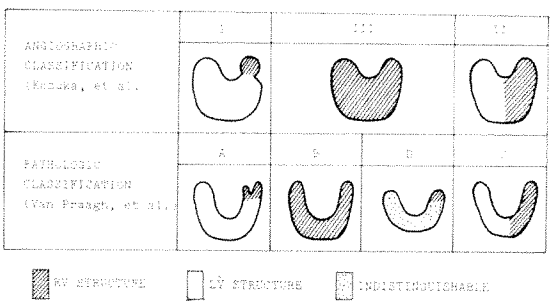


FIG. 1. Angiographic and anatomic classification of single ventricle.

Type C is distinguishable, if 2 well developed ventricular structures are shown on angiocardiograms lacking only the interventricular septum.

In this paper, we propose to differentiate single ventricle angiocardiographically into 3 types (Fig. 1):

Type I. Single left ventricle with lack of right ventricular sinus. This is identical to Van Praagh's Type A.

Type II. Undivided ventricles with missing ventricular septum. This is identical to Van Praagh's Type C.

Type III. Single right ventricle and infundibulum only. This is identical to Van Praagh's Types B and D.

Roentgenologic classification is based on the features of the inner surface of the ventricle which is demonstrated on the angiocardiograms.

According to Van Praagh *et al.* the single ventricle can furthermore be subdivided

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by the interrelationship of the great arteries. In our opinion, identification of the ventriculotruncal relationship of congenital heart disease depends not on the mutual interrelationship of the great arteries, but rather on the relationship of the great arteries to the ventricles.[9,12] Since both ventricular structures are present in Types I and II, all the types of ventriculotruncal relationships exist. In Type III, there is only one ventriculotruncal relationship possible, namely double-outlet right ventricle type, because it has no left ventricular component identifiable on angiocardiograms.

MATERIAL

Forty-two patients with single ventricle were studied by plain roentgenograms and angiocardiograms. Of the 42 patients, 28 were male (67 per cent), and 14 were female (33 per cent). The ages at the time of the examination ranged from 2 months to 32 years, and the average age was 4.2 years.

METHOD

Biplane selective ventriculography was performed on all patients except 1, where transvenous angiocardiograms were made. Anteroposterior and lateral views were simultaneously obtained. The contrast medium employed was sodium iothalamate 80 per cent or 67.5 per cent. The catheter was usually introduced through the saphenous vein. If this route was not available, the axillary vein was employed. The films were exposed at the rate of 6 per second for the initial 2 seconds, 4 per second for the following 1 second, and 2 per second for the last 2 seconds. When a nonvisualized ventricular portion seemed to remain in the cardiac silhouette of the first series of angiocardiograms, a second angiocardiography was performed by injection of the contrast medium in the presumed remaining part of the ventricle. In the cases where the anatomy concerning the atrioventricular relationship or atrioventricular valves was not determined by selective ventriculography, the contrast medium was also injected into either atrium or both.

CORRECTIVE SURGERY

Sixteen cases underwent operation at the First Department of Surgery in the Osaka University Hospital. Of these 16 cases, 11 patients underwent palliative surgery, 4 had the Blalock-Taussig operation, 4 had the Glenn operation, 1 had the Waterstone-Cooley operation, 1 had banding of the pulmonary artery, and 1 had correction of pulmonary stenosis. Corrective surgery was attempted in 5 cases, of which 1 survived. Seven cases were examined at autopsy.

ROENTGENOGRAPHIC FINDINGS

The data obtained in this study are shown in Table 1.

PLAIN ROENTGENOGRAMS OF THE CHEST (Fig. 2, *A–D*)

Almost all patients had posteroanterior roentgenograms that showed abnormal cardiovascular shadows. The cardiac silhouette was usually enlarged and not specific in shape except for some patients of Type I, where a small convexity was noted in the left border of the ventricular portion, suggesting the presence of the outflow chamber. Some patients showed a heart of wooden-shoe form quite similar to that of the tetralogy of Fallot. Dextrocardia or levocardia with peculiar shape is not uncommon in single ventricle. Pulmonary vasculature varied according to the presence of pulmonary stenosis.

Situs solitus, levocardia occurred in 7 cases of Type I, 10 cases of Type II, and 6 cases of Type III. Situs inversus, levocardia (isolated levocardia) was exclusive to Type III, and occurred in 3 of the cases. Situs solitus, dextrocardia (isolated dextrocardia) occurred in 3 cases of Type II. Situs inversus, dextrocardia was found in 1 case of Type II. Indeterminate visceral situs occurred in 11 cases and was most prevalent in Type III.

Abnormalities of the visceral situs were

TABLE I

ANGIOGRAPHIC ANALYSIS OF SINGLE VENTRICLE

Type of Single Ventricle	No. of Cases	Cardiac Position	Visceral Situs	No. of Cases	Ventriculotruncal Relationship				No. of AV Valves		No. of Atrium		Aortic Arch	
					N	TGA	DO	Uncertain	2	1	2	1	L	R
I	9 (22%)	Levocardia	solitus	7	1	4	1	1	2	5	4	3	7	
			indeterminatus	1			1			1		1		1
		Dextrocardia	indeterminatus	1			1			1		1		1
II	17 (40%)	Levocardia	solitus	10	4	2	3	1	9	1	7	3	9	1
			indeterminatus	3		1	2			3	1	2	1	2
		Dextrocardia	solitus	3	1	1		1	3		1	2	3	
			inversus	1	1					1		1	1	
III	16 (38%)	Levocardia	solitus	6			6			6	5	1	5	1
			indeterminatus	5			5			5		5	3	2
			inversus	3			3			3		3	2	1
		Dextrocardia	solitus	1			1			1		1	1	
			indeterminatus	1			1			1		1	1	
Total	42 (100%)				7 (17)	8 (19)	24 (57)	3 (7)	14 (33)	28 (67)	18 (43)	24 (57)	33 (79)	9 (21)

N: normal ventriculotruncal relationship.
TGA: transposition of great arteries.
DO: double-outlet right ventricle.
L: left-sided aortic arch. R: right-sided aortic arch.

detected by recognizing the location of the air-containing gastric fundus. Indeterminate visceral situs was implied by the symmetric hair-line of both lung fields, showing 3 pulmonary lobes on each side, and an increase of distance between the diaphragm and the gastric fundus, as well as a symmetric liver density. Of course, it was also important to identify the Howell-Jolly bodies in the blood, as well as the location of the inferior vena cava, the distribution of the hepatic vein, and the absence of the splenic artery, which were visualized on the angiocardiograms.

ANGIOCARDIOGRAPHY

By this study, Type I single ventricle was found in 9 cases (22 per cent) of these patients, Type II in 17 (40 per cent), and Type III in 16 (38 per cent).

In Type I, a large ventricle was considered as the morphologic left ventricle, since its trabeculation was rather fine in comparison with the infundibular chamber. This ventricle was connected through the bulboventricular foramen to a small triangular outflow chamber, suggesting the right ventricular structure (Fig. 3, *A* and *B*; and 4, *A* and *B*). Both ventricular structures were situated side by side in the anteroposterior view in all the cases except 2, in which they appeared in the same sagittal plane one behind the other and the anatomy was best seen in the lateral view (Fig. 5, *A–D*). The outflow chamber, which was variable in size, showed considerable contractility and no atrioventricular valve opened into it.

Both ventricular structures were angiocardiographically demonstrated in Type II. In some cases, a thick muscular ridge divided the ventricle into two separate por-

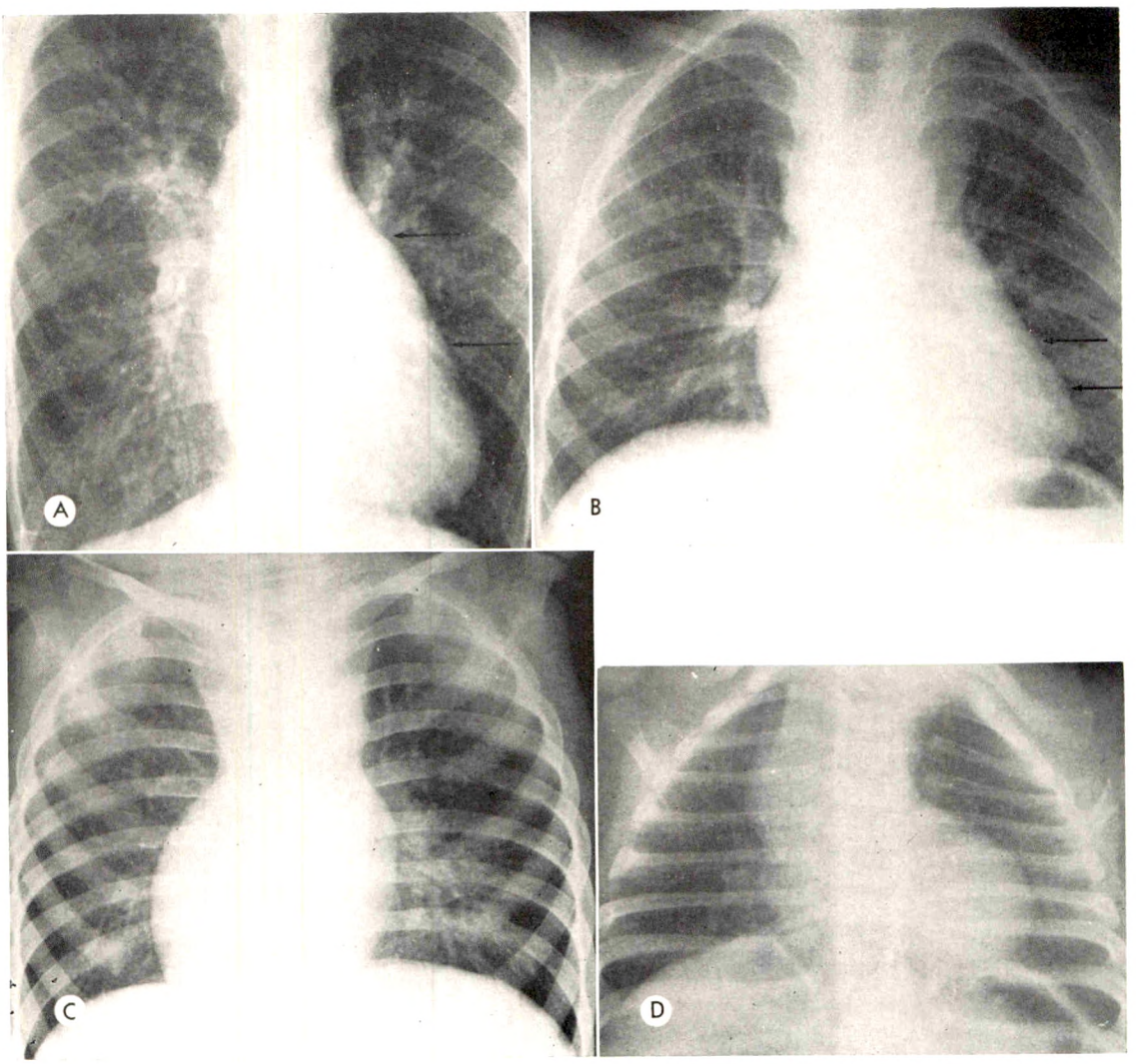


Fig. 2. (*A—D*) Plain roentgenograms of chest in 4 cases. Two cases of Type I (*A* and *B*) show a small convexity on the left cardiac border (between arrows). A case of Type II with dextrocardia and situs solitus (*C*) shows right cardiac border to consist of single gentle convexity. Wide upper central shadow represents bilateral superior vena cava. In a case of Type III (*D*), the heart shadow is similar to a "wooden-shoe" form. The aortic knob is on the right side.

tions with different trabeculation. These ventricular portions appeared to develop well and to a similar size. In some cases the initial ventricular injection demonstrated only 1 ventricular structure but when a second injection was made in a different location (atrium or other ventricle) a second ventricular portion could be seen (Fig. 6, *A–D*; 7, *A* and *B*; and 8, *A–D*).

Type III (sole ventricle with the coarse trabeculation characteristic of the right ventricle) was diagnosed only after repeated and varied injections failed to show evidence of another ventricle (or portion) (Fig. 9, *A* and *B*).

Normal ventriculotruncal relationship was found in 1 of the 9 cases (11 per cent) of Type I, and 6 of the 17 cases (35 per cent) of Type II. Transposition of the great arteries type was noted in 4 (44 per cent) of Type I, and 4 (24 per cent) of Type II. Double-outlet right ventricle

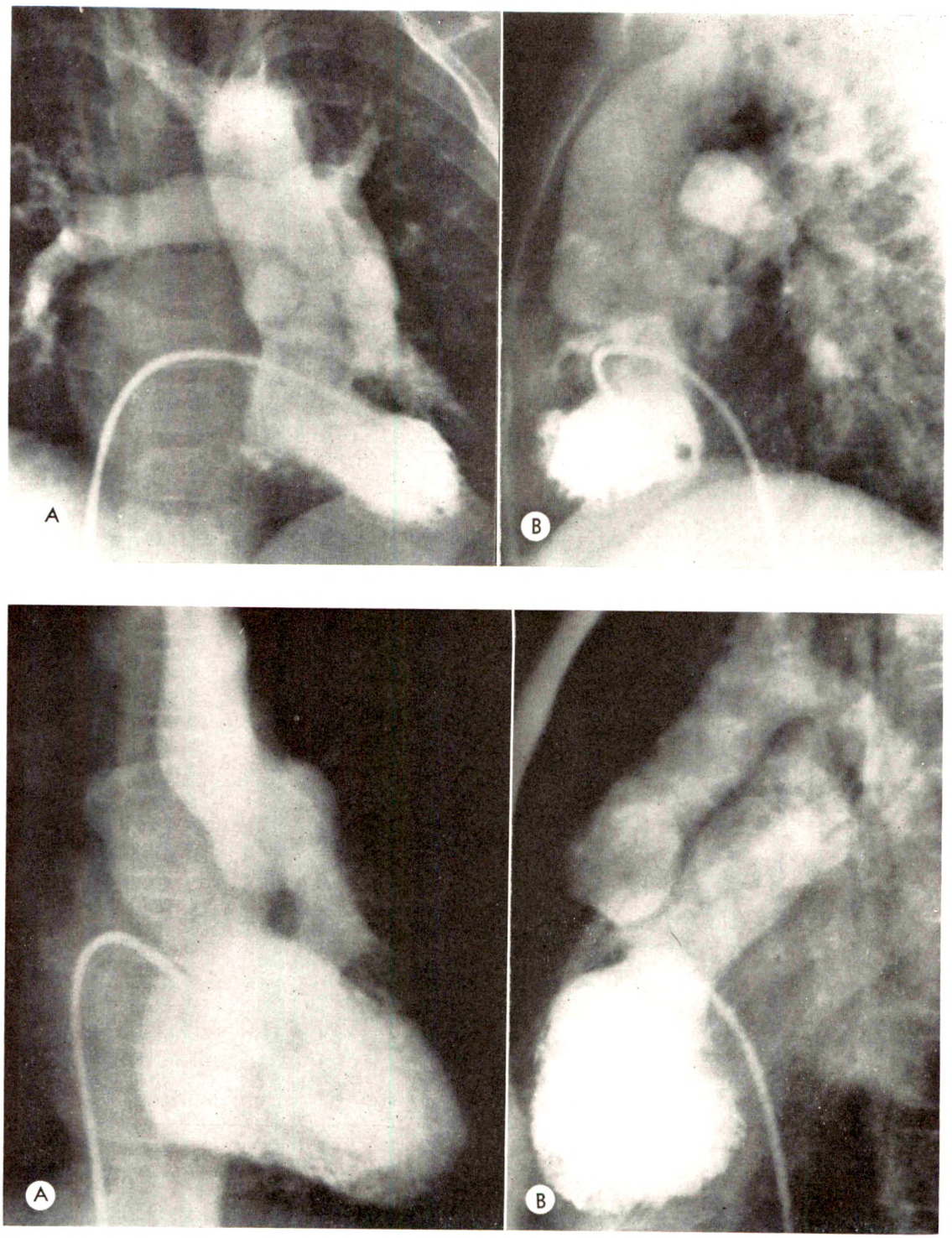


FIG. 4. (*A* and *B*) Single ventricle. Type I. Transposition of great arteries type. A 12 year old boy. Antero-posterior ventriculogram (*A*) shows a large single ventricle with left ventricular structure which gives rise to the pulmonary artery. The bulboventricular foramen connects the outflow chamber with the single left ventricle. The aorta is derived from the outflow chamber. Lateral view (*B*) demonstrates the aorta to be anterior to the pulmonary artery.

Fig. 3. (*A* and *B*) Single ventricle. Type I. Normal ventriculotruncal relationship. A 4 year old boy. Antero-posterior ventriculogram (*A*) shows single left ventricle communicating through bulboventricular foramen with the remnant right ventricle. The aorta derives from the left ventricle and the pulmonary artery from the outflow chamber. Lateral view (*B*) shows the aorta to be anterior to the pulmonary artery.

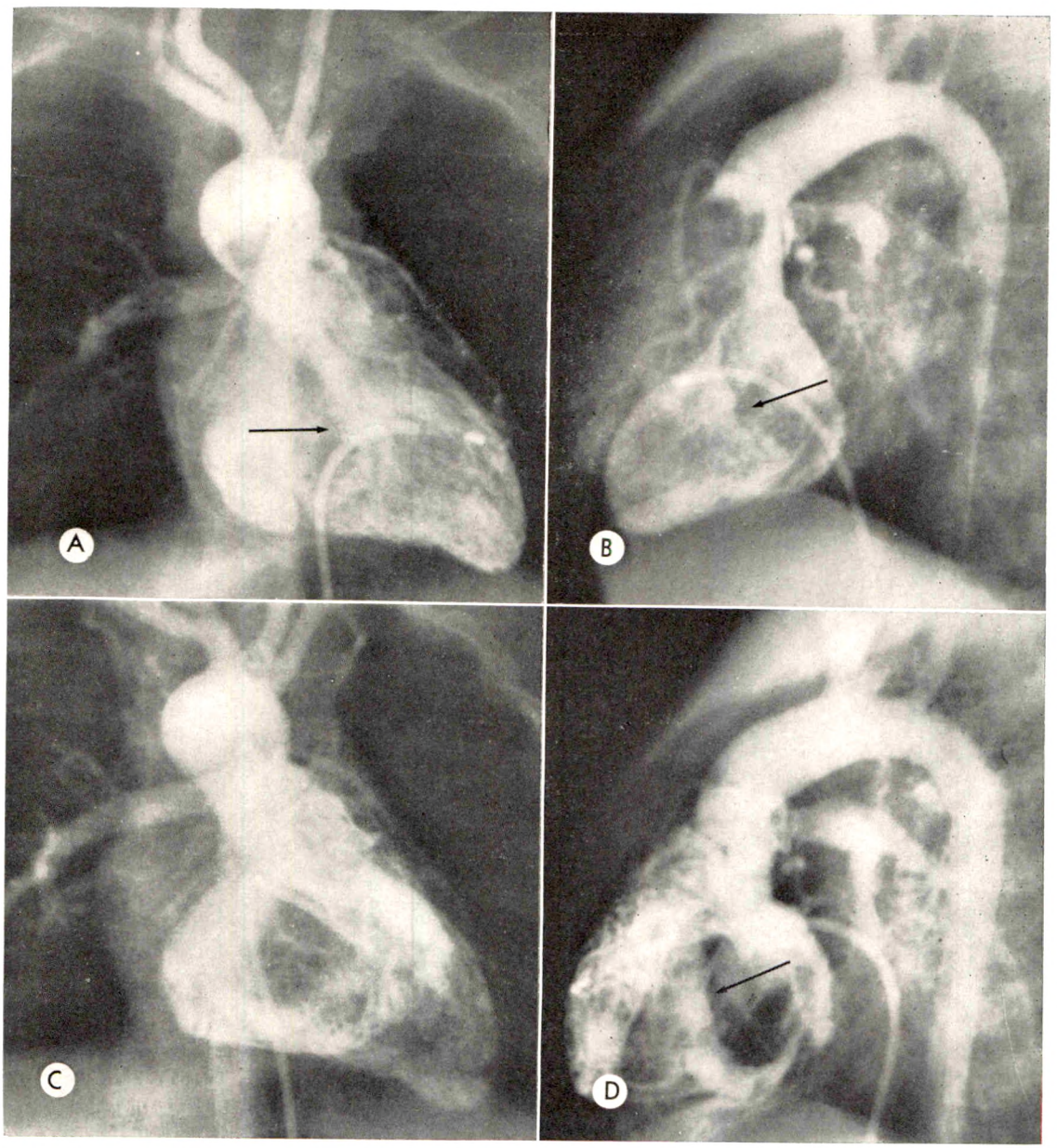


Fig. 5. (*A–D*) Single ventricle. Type I. Double-outlet right ventricle type. A 3 year old girl. The first ventriculograms (*A* and *B*) show a single left ventricle, where the common atrioventricular valve (arrow) opens: it is well demonstrated in the lateral view. The second ventriculograms (*C* and *D*) reveal a well developed outflow chamber, which gives rise to both great arteries. Catheter is inserted into this chamber through the large bulboventricular foramen. Subvalvular and valvular pulmonary stenosis is present.

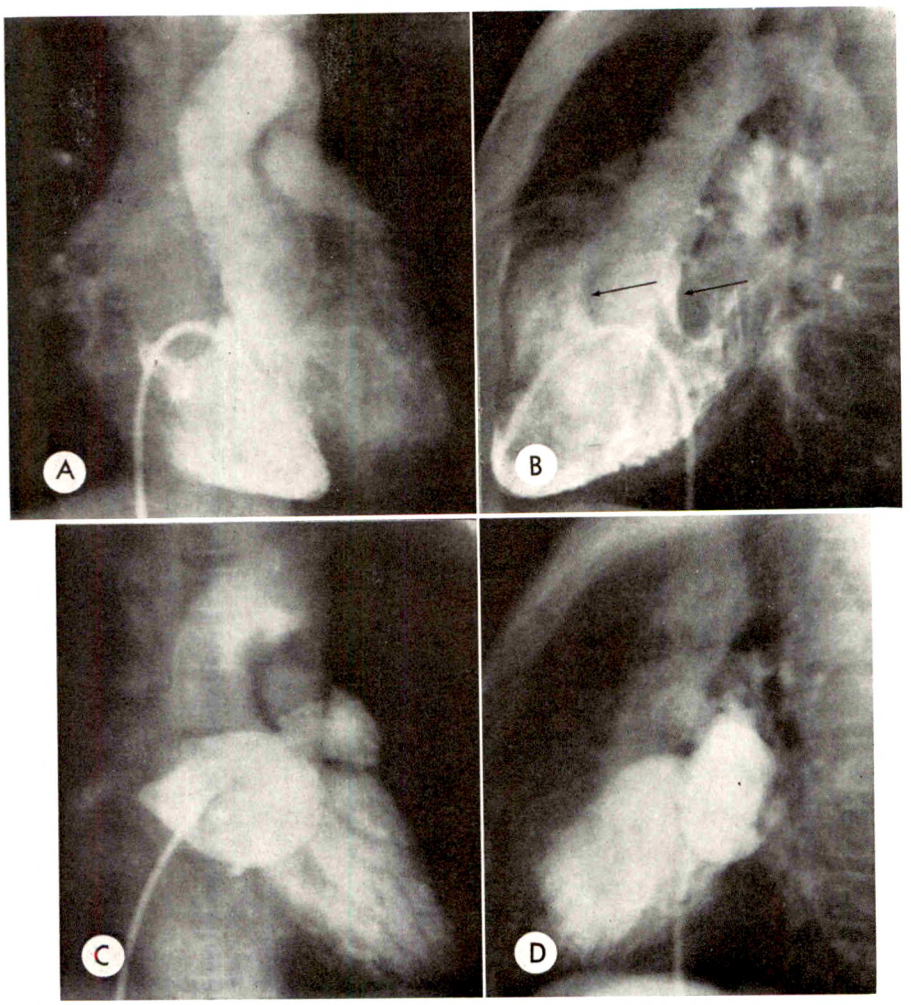


FIG. 6. (*A–D*) Single ventricle. Type II. Normal ventriculotruncal relationship. A 7 year old girl. Right ventricular structure is demonstrated in the first ventriculography (*A*). Catheter is inserted through the tricuspid valve. In lateral view (*B*), the tricuspid and mitral valves are visualized (arrows). At the same time, the second ventricular structure is demonstrated and both great arteries are visualized. Left atrial injection was performed with the catheter inserted through the interatrial communication. (*C* and *D*) The left atrium communicates with the left ventricular structure and the aorta is apparently derived from the left ventricle. Corrective surgery was successfully performed.

type was revealed in 3 of the 9 cases (33 per cent) of Type I, and 5 of the 17 cases (30 per cent) of Type II, and in all 16 cases of Type III. In those which had pulmonary atresia in Type I and II, it was impossible to determine ventriculotruncal relationship (Fig. 8, *A–D*).

The atrioventricular valves were demonstrated by selective ventriculograms as oval or semicircular translucencies (Fig. 6, *A–D*). Of the 14 (33 per cent) which had 2 atrioventricular valves, 2 were in Type I (22 per cent), 12 in Type II (71 per cent) and no case in Type III. Only 1 atrioventricular valve was revealed in the remaining 28 cases (67 per cent).

Concerning the anatomy of the atrium, only 1 atrium was noted in 24 cases (57 per cent) in this series. One atrium was most frequent in Type III (70 per cent), less frequent in Type I (56 per cent) and still less in Type II (47 per cent).

Pulmonary stenosis occurred in 6 cases of Type I, 10 cases of Type II, 9 cases of

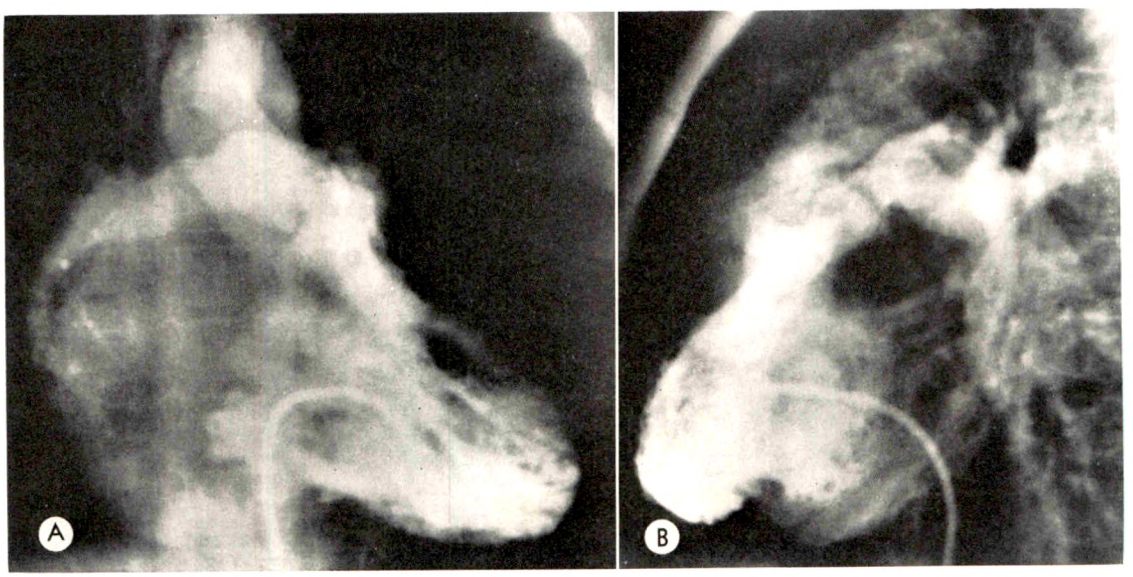


FIG. 7. (*A* and *B*) Single ventricle. Type II. Double-outlet right ventricle type. A 6 year old girl. Ventricular injection shows both great arteries to arise from anteriorly situated right ventricular structure. Valvular and subvalvular pulmonary stenoses are noted. The contrast medium spreads over to the left ventricular portion. Both ventricular portions develop well to similar size and a muscular ridge appears to separate them.

Type III, making 25 cases in all (60 per cent). Valvular and subvalvular stenoses are included (Table II). Pulmonary atresia was disclosed in 6 cases (14 per cent). Hypoplasia of the ipsilateral pulmonary artery was demonstrated in only 2 cases of Type III. Subaortic stenosis was detected in a case of Type I and in another case of Type II.

As regards other associated anomalies which were not uncommon in this malformation, a right aortic arch was noted in 10 cases (24 per cent), patent ductus arteriosus in 9 cases (21 per cent), absent inferior vena cava with azygos or hemiazygos continuation in 6 cases (14 per cent), bilateral superior vena cava in 8 cases (21 per cent), and anomalous pulmonary venous return in 4 cases (10 per cent).

DISCUSSION

In congenital heart disease, the incidence of single ventricle was reported as varying from 1.1[8] to 2–3[11] per cent. With regard to a comprehensive definition of single ventricle, the problem does not seem to have been settled in the literature. Van Praagh *et al.*[14] defined single ventricle as

TABLE II

INCIDENCE AND TYPE OF OBSTRUCTION TO THE PULMONARY CIRCULATION

Type of Single Ventricle	No. of Cases of Pulmonary Stenosis	Type of Pulmonary Stenosis		No. of Cases of Pulmonary Atresia
		Subvalvular	Valvular	
I	6 (67%)	5	4	1
II	10 (59%)	7	9	2
III	9 (56%)	8	8	3
Total	25 (60%)	20	21	6

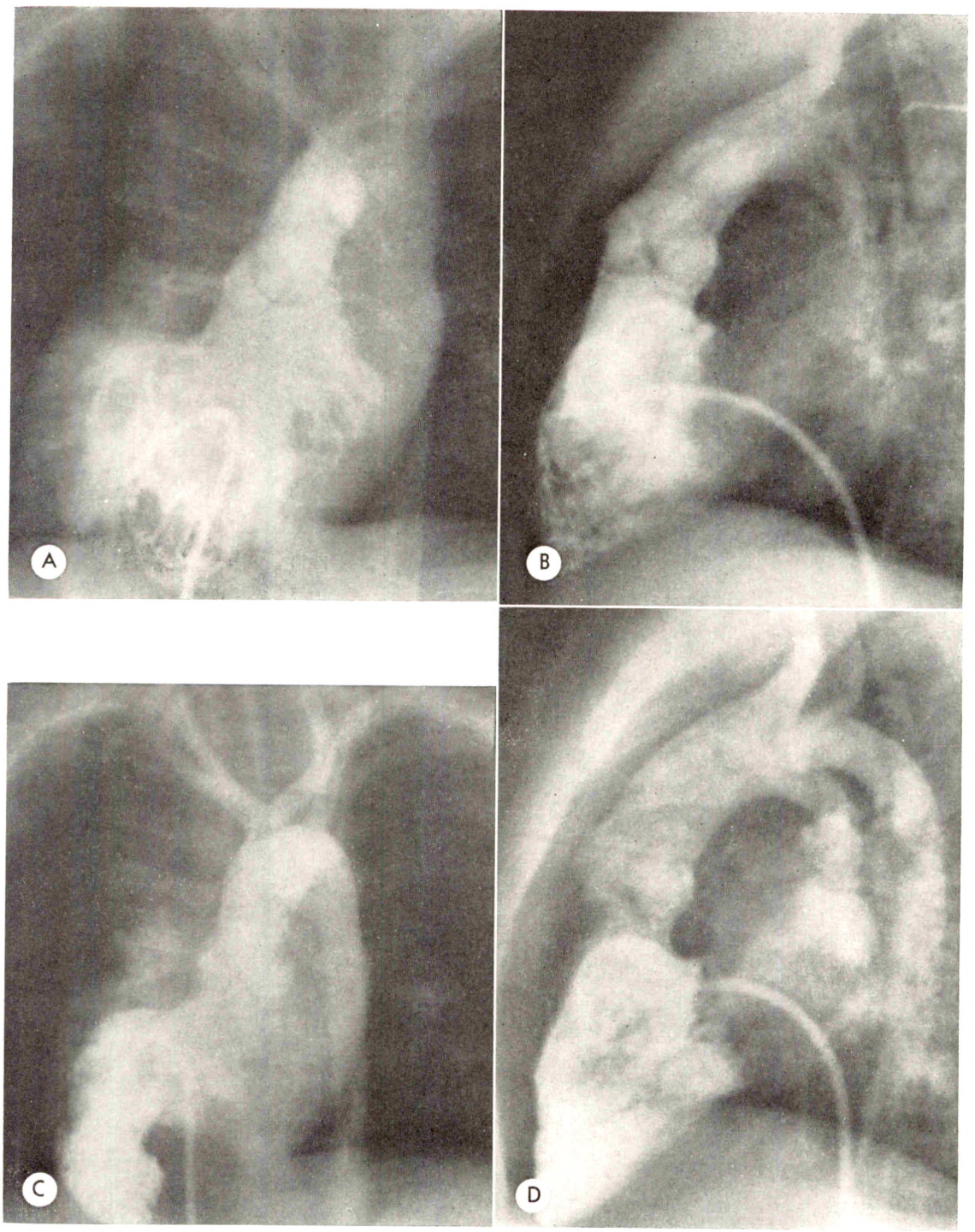


FIG. 8. (*A–D*) Single ventricle. Type II. Dextrocardia with uncertain ventriculotruncal relationship. A 1 year old boy. First ventriculograms (*A* and *B*) demonstrate right ventricular structure from which derives the aorta. The left ventricular structure is revealed by a second ventriculography (*C* and *D*). This structure appears to be separated from the right ventricular structure by a muscular ridge. Because of pulmonary atresia, the ventriculotruncal relationship is not determined, and the pulmonary blood flow is supplied by a large patent ductus arteriosus. The diagnosis was confirmed at autopsy.

the absence of the right or left sinus or of both ventricular sinuses, or the absence of the sinus portion of the ventricular septum. The term "single" and "common" were used interchangeably. Lev *et al.*[10] did not base their diagnosis on the absence of a sinus, since it could not be determined whether the wall belonged to the left or right of the common sinus. Single ventricle defined by Lev *et al.* corresponds to Van Praagh's Type A, and Lev's common ventricle corresponds to Van Praagh's Type C. From the embryo-anatomicopathologic point of view, de la Cruz and Miller[1] postulated that a double-inlet left ventricle exists when two separate atrioventricular ostia empty into a morphologic left ventricle. According to their concept, it is suggested that double-inlet left ventricle is to be differentiated from single ventricle, common ventricle or cor triloculare biatriatum. Since common ventricle, as defined by Elliott *et al.*,[3] consists of the infundibular chamber, and one ventricular chamber into which both atrioventricular valves empty, it corresponds to Van Praagh's Type A. In our presentation, the definition and classification by Van Praagh has been fundamentally used. This is the first to distinguish Type C, angiographically.

Careful contrast examination helps to distinguish single ventricle from other conditions. In single ventricle, remnant septum or muscular ridge may prevent diffusion of the contrast medium and thus may be misdiagnosed as another congenital malformation of the heart with 2 ventricles. Atrial injection does not give satisfactory information about the anatomy of the ventricle, because of marked dilution of the contrast medium, superimposition of the intra- and extracardiac structures, as well as the peculiar stream effect in single ventricle.[13] Ventricular injection is preferable to delineate the anatomy of the ventricle.

Except when the infundibular chamber has developed to a considerable size, Type I is easily recognized by the poorly developed anatomic features of the infundibular chamber. In Type II, one ventricular injection may lead to erroneous diagnosis, especially when a small amount of contrast medium is injected. Therefore, a rather large amount of contrast medium, commensurate with the cardiac size or body weight of the patient, is required, and a

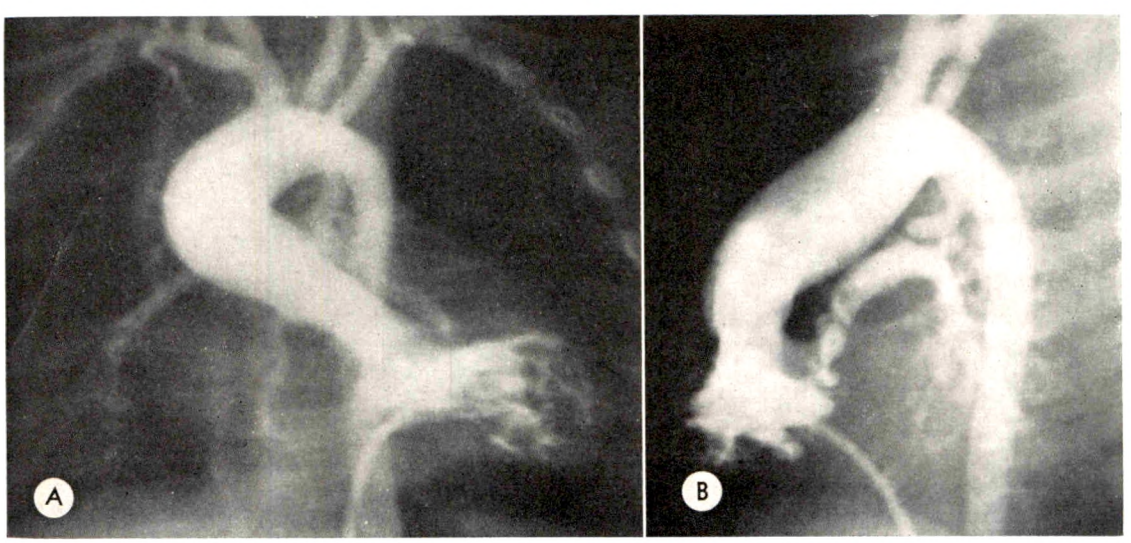


Fig. 9. (*A* and *B*) Single ventricle. Type III. Double-outlet right ventricle type. A 2 month old boy. Ventriculography discloses single right ventricle (*A*), which gives rise to both great arteries. Lateral view (*B*) shows subvalvular and valvular pulmonary stenosis. Ductus arteriosus is patent. The diagnosis was confirmed at autopsy.

second ventricular or atrial injection is sometimes necessary to define single ventricle with 2 ventricular structures separated by a muscular ridge. Furthermore, Type I with a well-developed outflow chamber simulates Type II. The confusion cannot be avoided in some cases. If an atrioventricular valve opens completely or participates in the right ventricular structure, as shown in Figure 7, *A* and *B*, it is not Type I, but Type II. In contrast, the outflow chamber in Type I has no atrioventricular valve attached to it (Fig. 5, *A–D*). Such precise analysis is not only of academic interest, but is also a surgical requirement. Depending on the anatomy of the single ventricle, corrective surgery may be possible. If 2 atrioventricular valves exist, Type II seems especially suitable for total correction. In the case shown in Figure 6, *A–D*, corrective surgery was performed and the patient still survives and is in good condition after 3 years.[7]

The incidence of each type of single ventricle mentioned in this study differs from the data reported in the literature. In the reports of Van Praagh *et al.*,[14] and Hallermann *et al.*[4] single left ventricle with an infundibular chamber is most frequent. In contrast, in our series, single ventricle without infundibular chamber is most frequent. The cause of the difference in the incidence remains in doubt, and may be explained on racial basis.

It may be unsuitable to apply the ordinary rule of ventriculotruncal relationship to single ventricle, in which only 1 ventricle exists and its complicated anatomy results in special hemodynamics. For surgical convenience, however, in this study this rule was employed for single ventricle. As a means of identifying the ventriculotruncal relationship in single ventricle, it is helpful to observe the presence of the fibrous continuity between the mitral and semilunar valves, as shown in the other congenital malformations of the heart.[2,5,6] Recent literature, however, has reported 4 cases of transposition of the great arteries with fibrous continuity between the aortic and mitral valves.[16] Therefore, the findings, based on the presence or absence of fibrous continuity, may be unreliable as a means to identify the ventriculotruncal relationship.

The number and anatomy of the atrioventricular valves are important, not only for the information about the ventriculotruncal relationship, but also as a key for surgical procedure. When precise findings concerning the anatomy of the ventricle and atrioventricular valves are not obtained by selective ventriculography, atrial injection may serve to give supplementary information, as described above.

Plain roentgenograms of the chest are not definitive means for making a diagnosis of single ventricle. Peculiar shape of the heart and cardiac enlargement with discordant visceral situs on the plain roentgenogram of cyanotic patients only suggest the presence of complex cardiac anomalies, which includes single ventricle. In this situation, angiocardiography is indispensable for making diagnosis and precise analysis of single ventricle. In order to detect associated anomalies of the great vessels as well, one should carefully perform the cardiac catheterization and always check the late phase of angiography showing the anomalies of pulmonary vein.

SUMMARY

The roentgenographic studies of 42 cases of single ventricle are presented.

The value of the plain roentgenograms was considerably limited as a diagnostic means. Single ventricle was angiocardiographically divided into 3 types. Type I, II and III corresponded to Van Praagh's Type A, C and B+D, respectively.

It was relatively easy to establish diagnosis of Type I, based on recognizing an infundibular chamber connected with the large single left ventricle. Those which had 2 well-developed ventricular structures were considered as Type II. Type III could not be further divided into Type B and D.

As a rule, determination of the ventriculotruncal relationship was based not on the

relative location of the great arteries, but on the connection of the ventricular structures and the great arteries. According to this rule, Type I and II had several ventriculotruncal relationships and Type III was classified exclusively as the double-outlet right ventricle type.

From the surgical standpoint of view, it was very important to establish the location and number of the atrioventricular valves. When these were not disclosed on the selective ventriculograms, atrial injection of the contrast medium was helpful to show the anatomy of the valves.

Precise angiographic observation is emphasized to determine the anatomy of single ventricle and its associated anomalies.

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AORTIC ANEURYSMS AS A LATE COMPLICATION IN THE SYNDROME OF SUPRAVALVULAR AORTIC STENOSIS, PECULIAR FACIES AND MENTAL RETARDATION*

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IN 1961, Williams and co-workers[16] described the combination of supravalvular aortic stenosis, peculiar facial expression and mental retardation as a new syndrome. The group of Beuren[1] published additional cases in 1962, and 1 year later some authors mentioned the striking similarity with the features of patients who survived infantile idiopathic hypercalcemia.[2] This relationship was then confirmed experimentally and by clinical observations.[8,9]

The usual cardiovascular lesions found in this syndrome are supravalvular aortic stenosis and multiple peripheral pulmonary stenoses. As additional malformations, coarctation of the aorta, ventricular septal defect and patent ductus arteriosus were found in a few cases; deformed aortic valves also were described.[5] Recently, anomalies of systemic peripheral arteries were reported, such as stenosed renal and mesenteric arteries.[4]

The purpose of this paper is to describe a further vascular complication of this syndrome: the formation of aortic aneurysms.

REPORT OF A CASE

This 7 year old boy was admitted to the hospital for evaluation of short stature and mental retardation. The family history was negative, pregnancy and birth were uneventful and the development in early infancy was apparently normal and without clinical signs of hypercalcemia. Serum calcium determinations were not done at the time.

A typical hypercalcemia facies (Fig. 1, *A* and *B*) was found, and the clinical cardiologic signs were suggestive of supravalvular aortic stenosis: there was no cyanosis; the right radial arterial pulse was normal; the pulses in the left arm and the femorals were slightly less palpable; the arterial cuff blood pressures were 135/70 mm. Hg on the right, and 110/95 on the left.

There was a slight left ventricular heave and a systolic thrill at the jugulum. A harsh loud Grade 4 systolic ejection murmur was heard over the 2nd right intercostal space radiating towards the carotid arteries. The first sound was normal and the second sound normally accentuated and split.

The electrocardiogram showed sinus rhythm, a frontal axis of +90°, and normal QRS without evidence of ventricular hypertrophy. The chest roentgenogram revealed a normal heart size and configuration, and a normal pulmonary vasculature.

Cardiac catheterization and angiocardiography. There were normal O_2 saturations in the right and left heart; the right atrial, right ventricular and pulmonary arterial pressures were normal, as were the pulmonary arterial wedge pressures. The systolic pressure gradient across the aortic root was 65 mm. Hg. On angiography (Fig. 2, *A* and *B*) a supravalvular stenosis was seen, the aorta was generally hypoplastic. No anomalies were found on the wall of the aorta or on the brachiocephalic vessels.

The child was asymptomatic, and it was felt at the time that no surgical treatment of the supravalvular stenosis was advisable. Seven years later, at the age of 14 years, the boy was admitted again to the hospital after a period of prolonged fever and malaise.

A pronounced hepato- and splenomegaly was found. The cardiologic signs were virtually unchanged; several blood cultures remained sterile. Biopsy of the liver revealed a granulomatous hepatitis, and biopsies of the spleen and of a small inguinal lymph node were normal. Under prednisone therapy the boy made

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FIG. 1. (*A* and *B*) Patient at age 7 years. Typical facies with hypergnathia, underdeveloped bridge of the nose, and epicanthal fold.

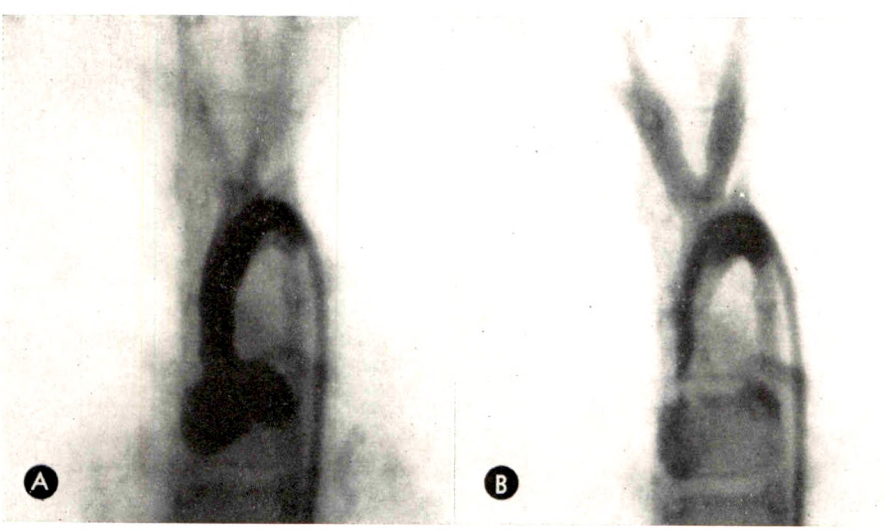


FIG. 2. (*A* and *B*) Posteroanterior aortograms at age 7 years. Small ascending aorta without distinct supravalvular narrowing. No wall changes are seen in the ascending aorta or aortic arch.

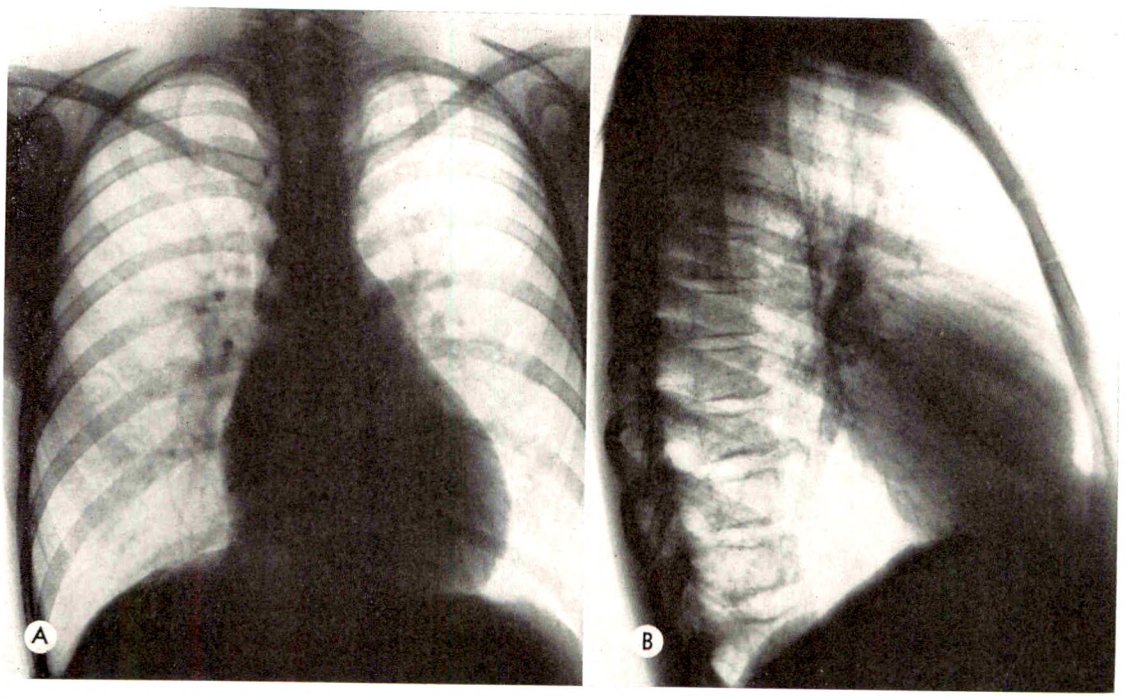


FIG. 3. (*A* and *B*) Posteroanterior and lateral roentgenograms of the chest of the patient, aged 15 years. The heart size and configuration are within normal limits. Note absence of calcifications.

a rapid recovery, the liver function returned to normal and the histologic changes were markedly improved. He had to be maintained under continuous immunosuppressive treatment with steroids and Imurel.

Repeat catheterization and angiocardiography were performed at the age of 15 years. At that time, most of the physical cardiologic signs were the same as 8 years earlier: the left ventricular heave was more pronounced and a strong pulsation of the left carotid artery was felt, whereas the right carotid, the radial and femoral pulses were similarly weak.

The electrocardiogram showed sinus rhythm, a frontal axis of +100°, atrial hypertrophy (probably left) and left ventricular hypertrophy with slight changes of repolarization. Frequent ventricular ectopic beats were recorded.

The chest roentgenogram revealed a normally sized heart and normal pulmonary vascular markings (Fig. 3, *A* and *B*).

Cardiac catheterization and angiocardiography. There were normal saturations on the right side and in the systemic circulation. Right atrial pressure was normal as was right ventricular end diastolic pressure. The systolic pressure in the right ventricle was at the upper limits of normal, and normal pressures in the distal right and pulmonary arteries were found. Pulmonary arterial wedge pressure showed a normal mean value, a normal V-wave and a elevated A-wave. End diastolic pressure in the left ventricle was at the upper normal limits, systolic pressure was markedly elevated to 190 mm. Hg, and a systolic gradient of 100 mm. Hg was found at the supravalvular level; a further systolic gradient of 30–40 mm. Hg was present at the site of the aortic isthmus. On angiocardiography (injection into the aortic root) the aortic valves were normal, dilated tortuous coronaries were seen and immediately distally a narrow supravalvular stenosis was visualized. The aorta showed an aneurysmal dilatation just beyond the stenosis, and a second aneurysm was found at the origin of the left carotid artery. The aorta was small, especially the descending part, and a coarctation at the usual site was seen in the left anterior oblique projection (Fig. 4; and 5).

Injection into the main pulmonary artery revealed a normal pulmonary vascular tree.

Operation. Surgery was performed while the patient was still under immunosuppressive therapy. The aortic arch and the supravalvular stenosis were removed and replaced by a

teflon prosthesis. The brachiocephalic trunk and the left carotid artery were reimplanted.

Histologic findings. The intima was fibrosed and there was a fibrosing inflammation of the adventitia. The main changes occurred in the media; severe inflammatory and degenerative changes with scar formation and destruction of the elastic fibers and musculature were seen.

Postoperative course. The postoperative course was uneventful, and the boy is doing very well now, 1 year after the operation.

DISCUSSION

Comparison of the 2 hemodynamic studies which were 8 years apart shows that the systolic pressure gradient across the stenosis rose from 65 to 100 mm. Hg. Both measurements were done under similar conditions; *i.e.*, general anesthesia and comparable cardiac output, the increased body surface being taken into account. In addition, an isthmic coarctation was found at the second catheterization. Although the increased pressure gradient could be caused by the growth related rise of the cardiac output in the presence of an unchanged stenotic area, the angiocardiogram suggests an actual narrowing of the stenosis.

Progressive obstruction of a subvalvular stenosis can be easily understood by reactive muscular hypertrophy of the outflow tract, and thickening and calcification of malformed valves is the known anatomic cause for increasing valvular stenosis.



Fig. 4. Posteroanterior aortogram at age 15 years. Note tortuous dilated coronary arteries, the narrow supravalvular region of the ascending aorta and the 2 aneurysms, one at the end of the stenotic area, the other at the origin of the left carotid artery.

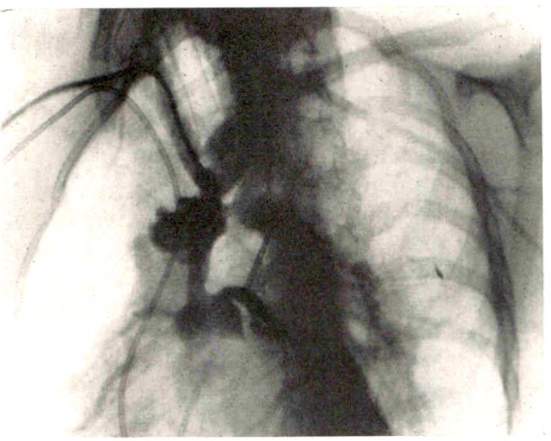


Fig. 5. Aortic root injection, left anterior oblique position. In addition to the features already seen in Figure 4, the isthmic stenosis is outlined.

Little information is available on the natural history of supravalvular stenosis, and in the 3 cases described, the stenotic area decreased over the observation period of 4 years.[4,5,11] In a few cases of coarctation of the aorta where increasing stenosis and the underlying pathologic changes were reported, intimal changes such as fibrosis and endothelial proliferation were found.[6]

Vessel wall defects as seen in our case are probably the cause of all progressive narrowing above the valve, supravalvular or coarctation.

The most striking changes, however, which were observed at the repeat catheterization were the 2 aortic aneurysms. Extreme post-stenotic dilatation might present itself as an aneurysmal wall deformity, but a second aneurysm distally precludes such a pathogenesis. A jet-lesion from a post-stenotic blood stream and consecutive vessel wall dilatation is also improbable, especially since histologic examination of the excised vessel showed no intima destruction which would be invariably present in such cases.[14]

The most probable etiology for both narrowing of the aorta and aneurysm forma-

tion is possibly the hypercalcemia syndrome with its pathologic arteries. Although no histologic reports of arteries from children with the William syndrome are available, the necropsy data of infants who died of idiopathic hypercalcemia could be used for comparison. The arterial changes revealed intimal thickening, degeneration of the internal elastic lamina and medial degenerative changes. In the aorta swelling and degeneration of the elastic fibers were observed, and there were also atheromatous thickenings with thinning of the underlying media and calcifications.[10]

Another source for comparison with our histologic findings is from animals, where hypervitaminosis D was produced experimentally. The typical changes in the arteries were degeneration of the muscle cells and calcification of the elastic fibers. Furthermore, the ascending aorta showed aneurysmal dilatation.[6]

It is thus very tempting to relate the stenotic changes and particularly the formation of the aneurysms to the underlying disease, namely the hypercalcemia syndrome.

Aneurysms of the ascending aorta combined with supravalvular aortic stenosis, however, were described in an 18 year old boy[13] and in a girl aged 18 years.[13] These patients did not belong to the group of the hypercalcemia syndrome, and in both instances a bacterial endocarditis preceded the detection of the aneurysms. Neither operative findings nor subsequent histologic examination revealed their etiology.

The microscopic appearance of the specimen, however, had some common features with the inflammation as described in chronic granulomatous disease. Incidentally, Sutcliffe[15] described among several cases with chronic granulomatous disease 1 child with stenosis of the carotid and subclavian arteries and post-stenotic dilatations. At thoracotomy the aorta was found to be imbedded in a mass of granulomatous tissue. This was, however, not the case in our patient; furthermore, the clinical picture has no resemblance to chronic granulomatous disease, the NPD test was negative, and chronic granulomatous hepatitis is not a feature of this disease.

A relationship of the liver disease with the vascular changes seems therefore unlikely, and if we consider the Williams' syndrome as the defective healing of infantile hypercalcemia, one can assume that this disease is associated with changes in the aortic wall. The damaged vessel predisposes to further alterations, such as the formation of aneurysms.

Perhaps aortic aneurysms in the supravalvular aortic stenosis syndrome would not be found to be such a rare occurrence, if serial angiocardiographic studies could be performed in all these children.

SUMMARY

In a 15 year old boy with the hypercalcemia syndrome and severe supravalvular aortic stenosis, aneurysms of the ascending aorta and the aortic arch—not present at the first catheterization 8 years earlier—were found.

This unusual complication is probably caused by the general vascular deficiency seen in this syndrome.

Stenosis and aneurysms were successfully repaired by a teflon prosthesis replacing the ascending aorta and the aortic arch.

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STERNAL ABNORMALITIES IN PATIENTS WITH CONGENITAL HEART DISEASE*

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MANY authors have reported an increased incidence of premature fusion of sternal sutures in patients with congenital heart disease (CHD); however, no studies to establish the incidence of this finding have been previously undertaken.[1,4,7,9,11] In a prospective study of 123 adolescent and young adult patients, we examined the association of CHD with scoliosis and with retarded skeletal growth and maturation; these studies have been reported previously.[14,21,22]

The purpose of this report is to discuss the sternal abnormalities encountered in our patients, including premature fusion of sternal sutures, pectus carinatum, and duplication of the manubrial ossification centers.

MATERIAL AND METHOD

One hundred and twenty-three patients, 12–42 years old, were selected at random from patients attending the Adolescent Cardiac Clinic of The Johns Hopkins Hospital. Only 4 patients, however, were over 30. The diagnosis of heart disease had been confirmed by cardiac catheterization, by surgery, or by both, in all but 3 acyanotic patients. The distribution of diagnoses of the 65 acyanotic and 58 cyanotic patients is listed as follows:

ACYANOTIC ADOLESCENT CHD PATIENTS

Ventricular septal defect	23
Ostium secundum ASD*	9
Ostium primum ASD	2
Sinus venosus ASD	1
Patent ductus arteriosus	1
Pulmonary valvular stenosis	5
Valvular aortic stenosis	12
Supravalvular aortic stenosis†	2
Subvalvular aortic stenosis	2
Coarctation of the aorta	4
Aortic insufficiency	1
Undiagnosed acyanotic CHD	3

* ASD = atrial septal defect.
† No clinical or historical evidence of the infantile hypercalcemia syndrome.

CYANOTIC ADOLESCENT CHD PATIENTS

Tetralogy of Fallot	23
Anomalous muscle bundle, right ventricle	1
Ebstein's malformation	4
Tricuspid atresia	3
Truncus arteriosus	5
Levo transposition with PS*	3
Levo transposition without PS	7
Dextro transposition with PS	4
Dextro transposition without PS	1
Single ventricle with PS	1
Double outlet right ventricle with PS	3
Other cyanotic heart disease	3

* PS = Pulmonary valvular or infundibular stenosis.

Patients with a total operative correction of tetralogy of Fallot were not included in the sample, but patients with only a palliative shunt procedure were.

A lateral chest roentgenogram of each patient was obtained. For most patients, a chronologic series of chest roentgenograms existed, so that it was possible to trace the status of their sterna from childhood. When patients had had a previous median sternotomy, only roentgenograms taken prior to surgery were used for evaluation. In each case, the lateral chest roentgenograms were evaluated for sternal segment fusion, the presence of pectus carinatum, and the number of manubrial ossification centers.

In order to document more accurately the time of appearance of sternal abnormalities on the chest roentgenograms, and

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to obtain a more accurate incidence of double-manubrial centers in CHD, the chest roentgenograms of an additional group of 128 randomly selected infants with CHD were evaluated. These patients from the Infant Cardiac Clinic of the Johns Hopkins Hospital ranged in age from 3 to 24 months and included 56 cyanotic and 72 acyanotic patients. The diagnoses had been confirmed at surgery and/or cardiac catheterization in all but 1 of the cyanotic infants. In 41 of the acyanotic infants, however, only a clinical diagnosis had been made. Their diagnoses are tabulated as follows:

ACYANOTIC INFANT CHD PATIENTS

Ventricular septal defect	30
Ostium secundum ASD*	8
Ostium primum ASD	4
Patent ductus arteriosus	8
Valvular aortic stenosis	5
Pulmonary valvular stenosis	10
Coarctation of the aorta	2
ASD with total anomalous pulmonary venous connection	1
Vascular ring	1
Undiagnosed acyanotic CHD	3

* ASD = Atrial septal defect.

CYANOTIC INFANT CHD PATIENTS

Tetralogy of Fallot	27
Tricuspid atresia	2
Truncus arteriosus	3
Double outlet right ventricle with pulmonary stenosis	2
Levo transposition without PS*	3
Dextro transposition with PS	5
Dextro transposition without PS	10
Single ventricle with PS	1
Other cyanotic heart disease	3

* PS = Pulmonary valvular or infundibular stenosis.

ANATOMY

The human sternum is divided into the manubrium, the body or mesosternum, and the xiphoid process. The sternum is preformed in cartilage and ossifies from several centers.[2,12,17,18] The ossification center of the manubrium appears first, in the 6th fetal month, and is generally single. It is followed in the 6th-7th fetal month by the superior of the 4 to 7 centers that form the body.[12,17] Ossification of the body proceeds caudally and is usually complete by the end of the first year of life.[17] The xiphoid may remain cartilaginous throughout life, but generally ossifies between the 5th and 18th years (Fig. 1).[18]

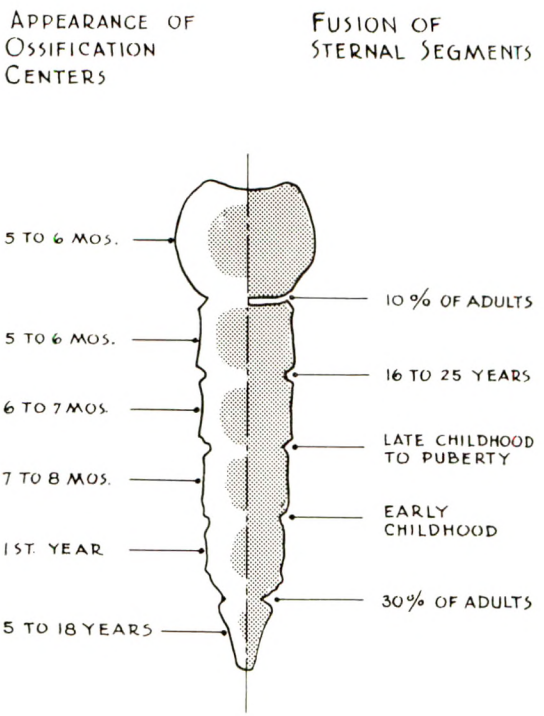


FIG. 1. The temporal sequence of the appearance of sternal ossification centers and their fusion. On the left the age in months refers to fetal age. (Courtesy of Dr. Frederic Silverman.)

In contrast to the caudal progression in the appearance of ossification centers, fusion of the sternal segments proceeds from caudal to cephalad. In early childhood, the 3rd and 4th sternal segments form a synostosis, and synostosis progresses upwards to the 1st and 2nd segments by the age of 25 years[18] (Fig. 1).

RESULTS

PREMATURE STERNAL FUSION

Of the adolescent and young adult patients, 119 had lateral roentgenograms which could be evaluated for fusion of the sternal sutures. Fifteen patients (13 per cent) had complete fusion of the sternum including the manubriosternal (MS) su-

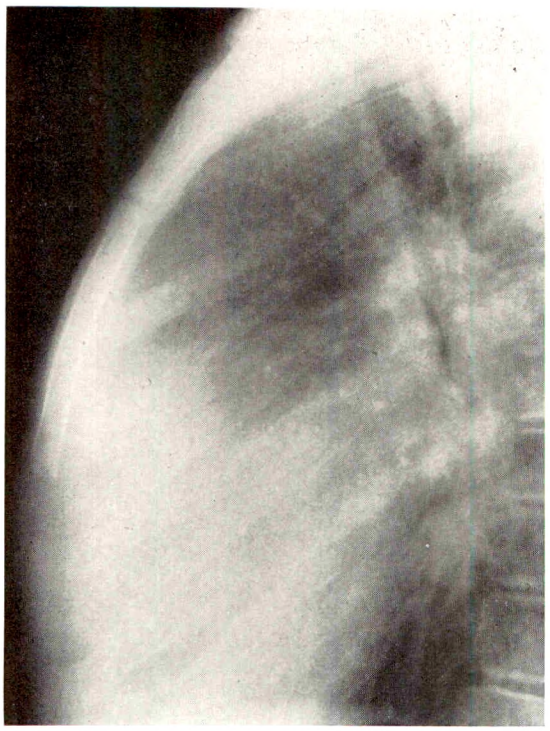


Fig. 2. Fourteen year old girl with levo transposition and fusion of the mesosternum. The manubriosternal joint remains unfused.

ture. The only data available on sternal fusion in normals deal separately with manubriosternal and mesosternal fusion, so we have divided our results for comparison. Of our 119 patients, a total of 74 (62 per cent) had fusion of all mesosternal sutures and 26 had fusion of some but not all of their sternal sutures (Fig. 2).

An attempt was made to determine whether sex, kind of CHD (*i.e.*, acyanotic or cyanotic), or specific diagnosis correlated with the extent of fusion. Table I, which summarizes these data for mesosternal fusion, does not include the 4 patients over 30 years of age, all of whose sutures were fused as expected. The females demonstrated significantly ($p < .01$) more fusion (80 per cent) than did the males (51 per cent). The extent of mesosternal fusion increased with age, but because the median and average ages of the male and female groups were almost identical, they are comparable. When the age factor was taken into account, the cyanotics had only a slightly greater incidence of mesosternal fusion than the acyanotic patients; and this was not statistically significant. The cyanotic females, although the oldest group, had by far the greatest percentage of fusion. The numbers are too small to be mathematically significant, but 19/20 (95 per cent) of the females with cyanotic CHD had completely fused their mesosternums, the only exception being a 12 year old with partial fusion. The acyanotic female patients, although the youngest as a group, had the second highest percentage of mesosternal fusion. No correlation between the degree of sternal fusion and a specific congenital defect was noted.

Early mesosternal fusion is reflected in

Table I

Incidence of fused manubrio-sternal and mesosternal sutures—adolescent CHD

	No. of Patients	Median Age	Average Age	Mesosternal Joint Fusion		Manubriosternal Joint Fusion	
				No.	Per Cent	No.	Per Cent
Females	40	17	17.4	32	80	9	22
Males	75	16	17.2	38	51	9	12
Acyanotic	59	16	16.4	30	51	5	8.5
Cyanotic	56	18	18.3	40	71	13	23
Acyanotic Females	20	16	15.4	13	65	1	5
Cyanotic Females	20	21	19.6	19	95	8	40
Acyanotic Males	39	16	16.9	17	44	4	10
Cyanotic Males	36	18	17.7	21	58	5	14

the infant age group also. Of the 128 infants, all of whose roentgenograms could be evaluated, 2 patients (1.6 per cent) showed complete fusion of their mesosternum, and 16 patients had some but not all sternal sutures synostosed. Of these 18 patients, 12 (67 per cent) were females again reflecting the increased fusion in females. Ten of the 18 patients had an acyanotic form of CHD (Fig. 3, *A* and *B*).

One hundred and eighteen adolescent and young adult patients had adequate lateral roentgenograms to evaluate manubriosternal (MS) fusion. Eighteen (16 per cent) had synostosis of the MS joint, and thirteen (72 per cent) of these were cyanotic patients. The sex incidence was even, but in this study men outnumbered women almost 2 to 1 so, in fact, women were a majority among patients with sternal manubrial fusion. Small numbers and the age factor make mathematically significant statements impossible, but again the highest number and percentage of fusion was found in the females with cyanotic heart disease where 8/20 (40 per cent) had MS fusion. Only 2/138 (1.6 per cent) of the infants with CHD had MS fusion. One was a female with a truncus arteriosus and the other a male with an ASD and VSD (Fig. 4).

PECTUS CARINATUM

An abnormal sternal bowing easily appreciated on the lateral chest roentgenogram was seen in 19/123 (15 per cent) of the adolescent and young adult cases as judged by 4 observers (K.F., R. W., C. J., and J. P. D.) (Fig. 5, *A* and *B*). Cyanotic patients outnumbered acyanotic patients with this deformity 15 to 4. The sex ratio was again even despite the 2:1 preponderance of males in the study. None of the 128 infant patients with CHD was thought to have a clearly demonstrated pectus carinatum deformity.

MANUBRIAL CENTERS

Of 130 infants with CHD, 26 (20 per cent) had 2 ossification centers. No patient

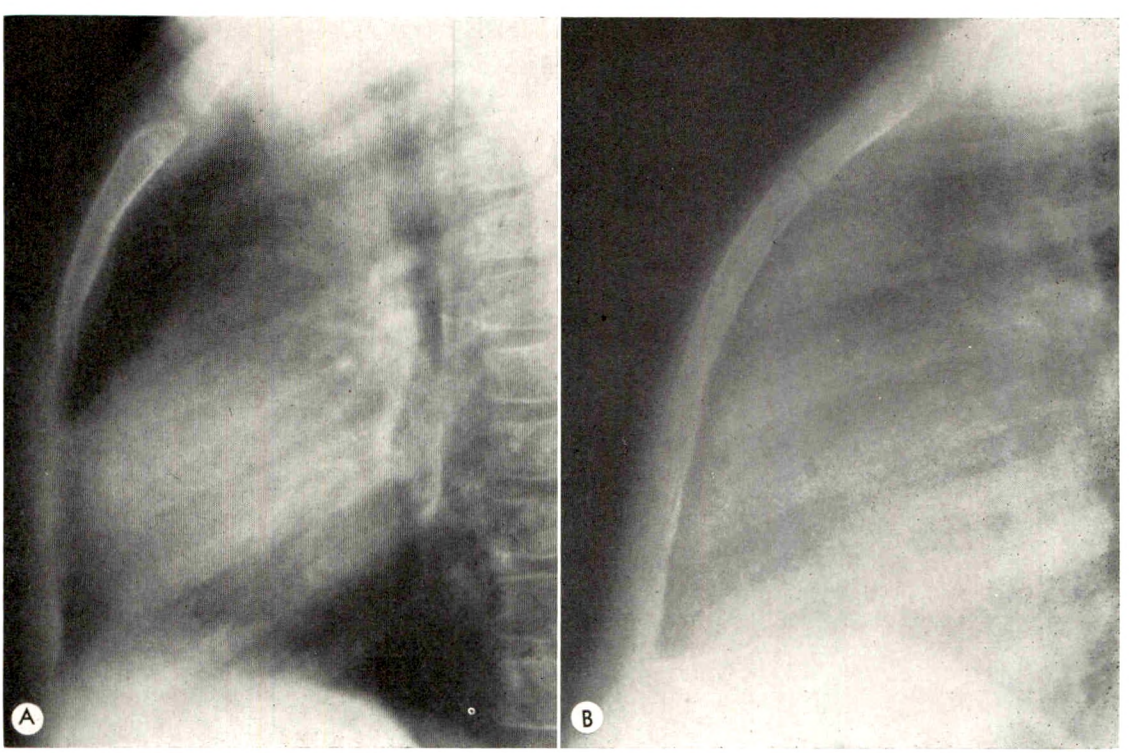


FIG. 3. Two 24 month old infants with (*A*) double outlet right ventricle, and (*B*) ventricular septal defect. A short fused mesosternum is present in *A* and a fused mesosternum with mild pectus carinatum in *B*.

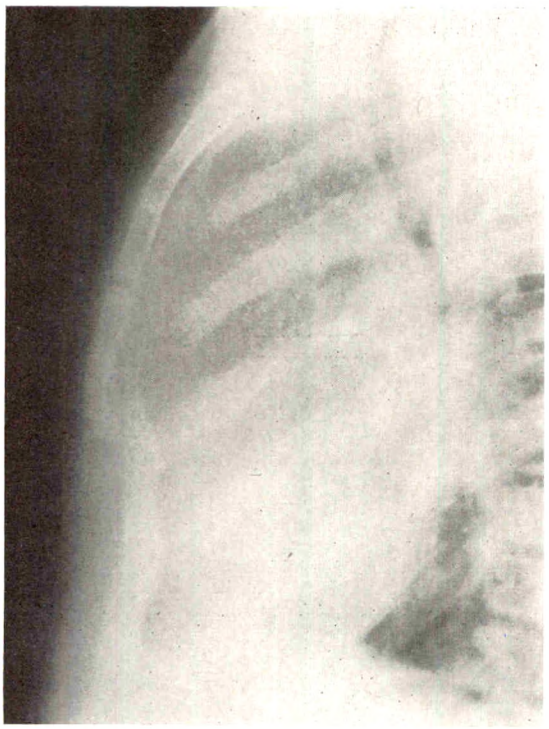


FIG. 4. Infant male patient with atrial and ventricular septal defect and fused manubriosternal joint.

was seen with 3 ossification centers, and no one diagnostic category was disproportionately represented. Since patients with Trisomy 21 (Down's syndrome) have an increased incidence of double manubrial ossification centers, these patients were not included in our study.[8,13]

DISCUSSION

Our data support the conclusion that there is a tendency toward premature sternal fusion among patients with congenital heart disease. This has not been documented previously by a prospective study, but several authors have reported cases of premature fusion.[1,4,7,9,11] Gabrielsen and Ladyman,[11] who reviewed these earlier case reports, were able to collect 11 cases and added 13 more of their own. A search of the English language literature did not reveal any further reports of cases since 1963.

A. M. Paterson in 1903 published the most complete data available on mesosternal suture fusion in normals.[18] Our data indicate a higher percentage of mesosternal fusion in patients with CHD at all ages except 15 years, where Paterson found 3 per cent more fusion. Although the number of cases is relatively small, this is not a chance occurrence and represents a real difference.

The difference in incidence of fusion is also readily apparent in the infant group. Among our 128 infants, 0–2 years of age, we found 2 patients with complete fusion of the body of the sternum and 15 patients with one or more of their sternal sutures fused. Paterson who looked at 18 anatomic specimens of sterna of normal infants in this age group was unable to find any with sternal fusion. In addition to these 18 normal cases, we were able to add 45 examples of our own of normal sterna which also demonstrated no fusion. These latter cases were obtained from studying the chest roentgenograms of 45 consecutive infants who had been studied for pneumonia and had no clinical evidence of CHD.

Comparing our data with the normal data of Ashley[3] on manubriosternal fusion again confirms the tendency to early synostosis found in patients with CHD. Ashley observed personally or determined from the literature the status of the MS suture in 2,787 anatomic specimens and divided his sample into 10 year age spans. The comparison with our data is shown below, but because there were only 4 patients above 30 in our study, their data are not included

Age Span (yr.)	MS Fusion, Normal (Ashley)	Total Cases and Per Cent CHD (current study)
1–9	0/70 = 0%	2/128 = 1.6%
10–19	3/169 = 1.8%	6/83 = 7.2%
20–29	18/334 = 5.4%	9/32 = 28%

In all age groups (even in the 1–9 year age group in which our patients are all younger than 2 years) the patients with CHD show an increased incidence of MS fusion compared to normals.

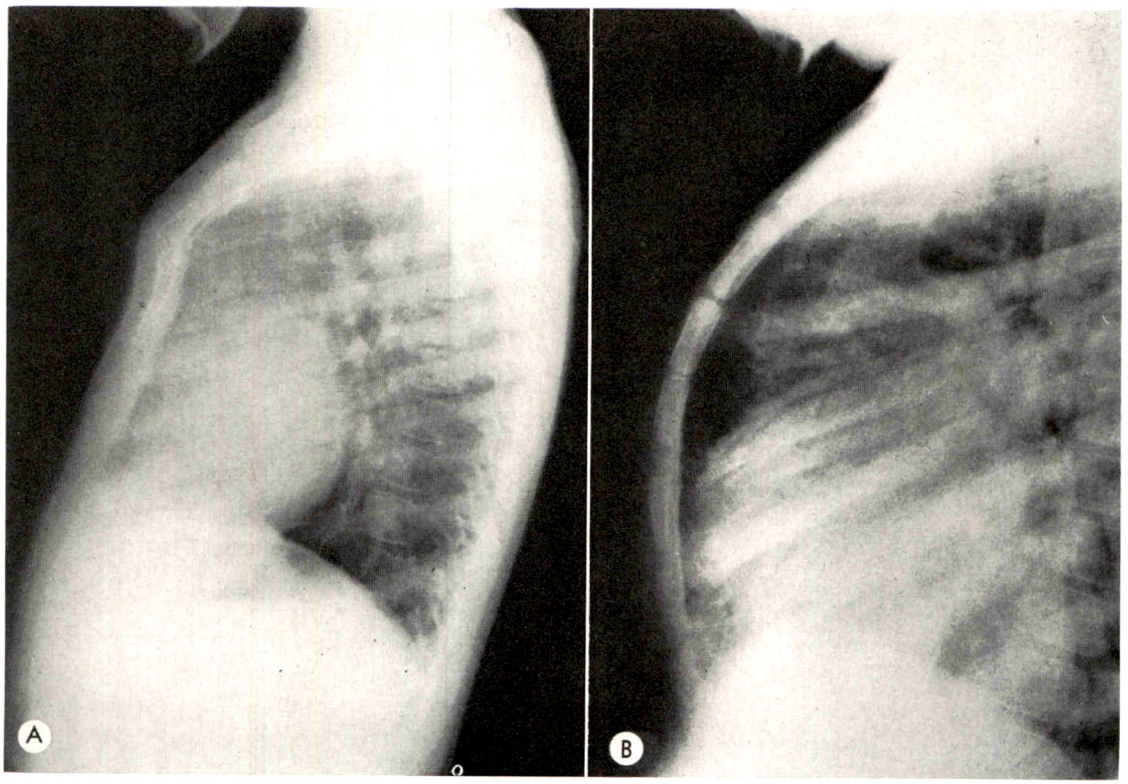


Fig. 5. (*A*) Fifteen year old patient with tetralogy of Fallot and short fused sternum with "pigeon breast" deformity consisting of acute angulation at the junction of the manubrium and mesosternum. (*B*) Four year old patient with ventricular septal defect, unfused sternum and arcuate deformity of sternum.

It was apparent from our study that premature fusion of the sternum resulted from an advancing synostotic process rather than from a primary nonsegmentation of the sternum. In all infant and adult cases of complete or partial fusion, suture lines were seen on earlier lateral chest roentgenograms. Primary nonsegmentation of the sternum may be the etiology in rare cases, however.[16]

The etiology of premature fusion of the sternum is certainly not clear. Both Chen[6] and Bryson[5] have demonstrated in mice the significance of the contact of ribs with the developing sternum in promoting segmentation. A chemical and/or mechanical factor appears to be involved. However, identification of factors influencing obliteration of the sutures has not been achieved experimentally. By analogy though, Gabrielsen and Ladyman[11] propose that acceleration of the synostotic process may be due to mechanical factors mediated through the effect of the ribs on the sternum by the size or position of the heart in congenital heart disease. Alternatively, they raise the possibility of a common developmental "injury" which may produce early closure of sternal sutures and a congenital heart defect.

Paterson[18] makes no statement as to the effect of sex on the incidence of fusion in his sample. Presumably, he found none, thus, our finding of increased sternal fusion in females with CHD is a real difference. We cannot attribute any etiologic significance to this finding.

The question of manubriosternal joint synostosis is clearer. For the manubrium to synostose with the body of the sternum, a cartilaginous union must pre-exist at this joint.[3,18] The more common situation is that a fibrous junction exists there (76.4 per cent), and a cartilaginous union exists only when the fibrous lamina, which first ap-

pears in the third fetal month, does not develop—an event Paterson believes occurs in 23.6 per cent of the normal population.[18] Ashley believes that the other factor affecting the rate of fusion of this joint is the size of the gap between the manubrium and body which exists at birth.[3] Thus, in congenital heart disease, it could be either that the gap is smaller than in normals or that a cartilaginous lamina develops in a greater number of cases to account for the increased incidence of fusion.

PECTUS CARINATUM

The 19 patients with abnormal sternal bowing (pectus carinatum) are roentgenographically similar to those first described by Davies[10] in his study of sternal deformity in patients with ventricular septal defects. He described this deformity as an increase in the anteroposterior diameter of the chest with the upper sternum protruded and the lower sternum relatively indrawn. As did Davies, we found the deformity to vary from a sharply angled protrusion (Fig. 5*A*) to a smooth arcuate type of protrusion (Fig. 3*B*; and 5*B*). He found this sternal configuration in 50 per cent (22/45) of VSD patients studied roentgenographically, but in none of his 80 patients with an ASD.[10]

In a group of small children with CHD, Corone *et al.*[7] found this type of sternal bowing in 15 per cent (92/600) of roentgenograms. These investigators demonstrated that the finding was not limited to patients with VSDs, but was present in patients with: atrial septal defects (9 per cent); tetralogy of Fallot (10 per cent); truncus arteriosus (18 per cent); transposition of the great vessels (TGV) (66 per cent); and AV communis deformities (23 per cent). Like Davies, they found bowing in 50 per cent of patients with a VSD. Similarly, Polis and Sluyts[19] found pectus carinatum in forms of CHD other than VSD.

Although our total incidence of pectus deformity (15 per cent) is commensurate with that obtained in other studies, its distribution among the various diagnoses is different. The higher incidence of pectus deformity in cyanotic patients, 15/58 (26 per cent), as compared to acyanotic patients, 4/65 (6.2 per cent) is significant ($p < .01$) and has not been previously reported. This may reflect the fact that the more "severely" affected acyanotic patients have their lesions corrected or die before adolescence and are not included in our study. In their large series, Corone and his colleagues[7] found that 68/263 (26 per cent) of acyanotic patients had a pectus deformity, while only 24/141 (17 per cent) of the cyanotic patients were involved. In our series, with the exception of Ebstein's malformation, all cyanotic diagnostic categories with more than 1 patient had at least one example of a pectus carinatum deformity. Like Corone *et al.*,[7] we found no pectus deformity in our patients with coarctation of the aorta (4 patients), aortic valve abnormalities (16 patients), or with pulmonic stenosis (5 patients). Nine per cent of our VSD patients had a pectus carinatum deformity.

The etiology of this thoracic abnormality has been debated. The effect of an enlarged right ventricle pressing on the sternum, a high incidence of respiratory infections, diminished compliance of the lung, and premature fusion of sternal segments are some of the proposed causes of pectus carinatum deformity.[9,10,15,20] That as yet this finding has not been found in patients with acyanotic heart disease without a shunt is intriguing but difficult to interpret. Maxwell,[15] who argues that the pectus carinatum deformity is often the end result of frequent respiratory infections, notes that he also has not seen this deformity in patients with pure pulmonary stenosis or coarctation. In these conditions, there is no abnormal increase in respiratory infections. We cannot attribute any specific etiologic significance to the preponderance of cyanotic patients among the group with pectus carinatum, but since these patients do not have an increased incidence of respiratory infections, it is unlikely that this factor is etiologically important.

Our data support the acquired nature of this deformity as we have not seen an un-

equivocal pectus carinatum abnormality on the roentgenograms of infants below the age of 18 months. In addition, we have noted the development of the pectus carinatum in young children who did not show the deformity as an infant. Currarino and Silverman[9] have proposed that one cause of pectus carinatum deformity is obliteration of sternal sutures which are inadequately formed in the fetal period and, thus, ossify prematurely. Their illustrations show a sharply angled as well as shortened sternum similar to Figure 5*A*. This, indeed, may be one cause, but probably does not account for the smooth "arcuate" type of pectus carinatum which can occur without premature fusion of sutures (Fig. 5*B*). In our study, of the 19 patients with pectus carinatum deformity, only 11 had a fused sternum.

DOUBLE MANUBRIAL CENTERS

The optimum age in which to demonstrate a double manubrial ossification center on lateral chest roentgenograms is from 1 month to 4 years.[13] Thus, our incidence of 7/123 (5.7 per cent) in adolescent and young adult population is suspect of being falsely low, because in many cases roentgenograms from this early age were not available. Our incidence of 26/130 (20 per cent) of infants with CHD having a double center is a more reliable incidence figure. Twelve acyanotic and 14 cyanotic patients had double manubrial centers with males predominating (17:9). This compares with 15 per cent obtained by Horns and O'Loughlin[13] in a study of 106 patients with CHD.[13] In normals, 14–20 per cent have been found to have a double manubrial center.[8,13] No predominance of double manubrial centers in a particular diagnostic category was seen. Thus, we have demonstrated no increased incidence of double manubrial centers among patients with CHD.

SUMMARY

In a prospective study of 123 adolescent and young adult patients and 128 infant patients with congenital heart disease, we found an incidence of 62 per cent of complete mesosternal fusion in the older age group and 1.6 per cent among the infants. This represented an increased incidence of mesosternal fusion over normals. Females demonstrated more mesosternal fusion (80 per cent) than did males (51 per cent), and 95 per cent of cyanotic females had fused mesosterna. Manubriosternal fusion in our population (16 per cent) was increased above normals at all age groups. The incidence of pectus carinatum deformity (15 per cent) in our population was similar to the previous reports of this deformity in patients with CHD, but the finding of a higher incidence (26 per cent) of pectus carinatum deformity among cyanotic patients as compared with acyanotic patients (6.2 per cent) represented a new observation. No increased incidence of double manubrial centers was found among patients with CHD.

Our study supports the theory that early fusion of the sterna is acquired and not the result of primary nonsegmentation, but the factors leading to early sternal fusion in patients with CHD are still uncertain. Many proposals for the etiology of pectus carinatum deformity are presented, but our study does not differentiate among these.

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Vol. 119, No. 3

ABERRANT RIGHT SUBCLAVIAN ARTERY*

FINDINGS SEEN ON PLAIN CHEST ROENTGENOGRAMS

By JAMES J. BRANSCOM, M.D., *and* JOHN H. M. AUSTIN, M.D.
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ABERRANT right subclavian artery occurs in approximately 0.5 per cent of the population.[1,3,6] Roentgenographically, the characteristic oblique indentation by the artery upon the posterior wall of the barium-filled esophagus is well-known.[1,2,4,5,8] It is surprising then that the appearance on plain chest roentgenograms of the abnormalities caused by this condition has received little attention. Stewart and co-workers[8] described 2 cases, in which diagnosis was based on esophagograms only, and in which posteroanterior chest roentgenograms demonstrated an oblique linear density ascending to the right from the superior margin of the shadow of the aortic arch. These authors attributed this linear density to an aberrant right subclavian artery. Similar findings have been mentioned by Felson *et al.*[2] and by Stauffer and Pote.[7] These authors also depended only upon esophagographic documentation.

On reviewing plain chest roentgenograms of several patients with arteriographically-proved aberrant right subclavian arteries, we noticed abnormal shadows superior to the aortic arch, in both posteroanterior and lateral projections. Because this finding has not previously been reported to have been found in lateral projections and because it has not been reported to occur in posteroanterior projections, when arch aortograms were used to determine the diagnosis, the following is presented.

METHOD AND RESULTS

All arch aortograms obtained during the period 1967 through 1971 at the University of California, San Francisco, San Francisco General Hospital, and the Veterans Administration Hospital, San Francisco, California, were reviewed for cases of aberrant right subclavian artery. Twelve patients were found to have such a condition. Posteroanterior and lateral plain chest roentgenograms were evaluated for each patient. Five of the 12 patients had roentgenograms showing no abnormal shadows in the region of the known aberrant right subclavian artery. These 5 patients ranged in age from 59 years to 78 years (mean, 69 years). Ages of the remaining 7 patients, who showed the abnormal densities, ranged from 47 to 81 years (mean, 67 years). The mean ages of the 2 groups were not significantly different. None of the patients had complained of dysphagia.

On posteroanterior roentgenograms, each of the group of 7 patients demonstrated a shadow denoting water density, which arose from the superior margin of the aortic arch and extended superiorly to the right, blending into the mediastinal silhouette (Fig. 1*A*). Arteriograms confirmed that this shadow represented the origin of the aberrant artery (Fig. 1, *C* and *D*). The length of the shadow ranged from 1.3 to 3.5 cm. In 1 patient (Fig. 2, *A* and *B*), atherosclerotic calcification was visible within the shadow. In another patient, the aberrant artery had an area of aneurysmal dilatation (Fig. 3, *A* and *B*).

A substudy was made of 80 of the patients in our review who had arteriographically-proved normal origin of the right subclavian artery. A density similar to that seen in the 7 patients mentioned above was noted on the posteroanterior roentgenograms of 16 (20 per cent) of these 80 patients (Fig. 4). In this control group, however, the length of the shadow was 0.5 to 1.9 cm.

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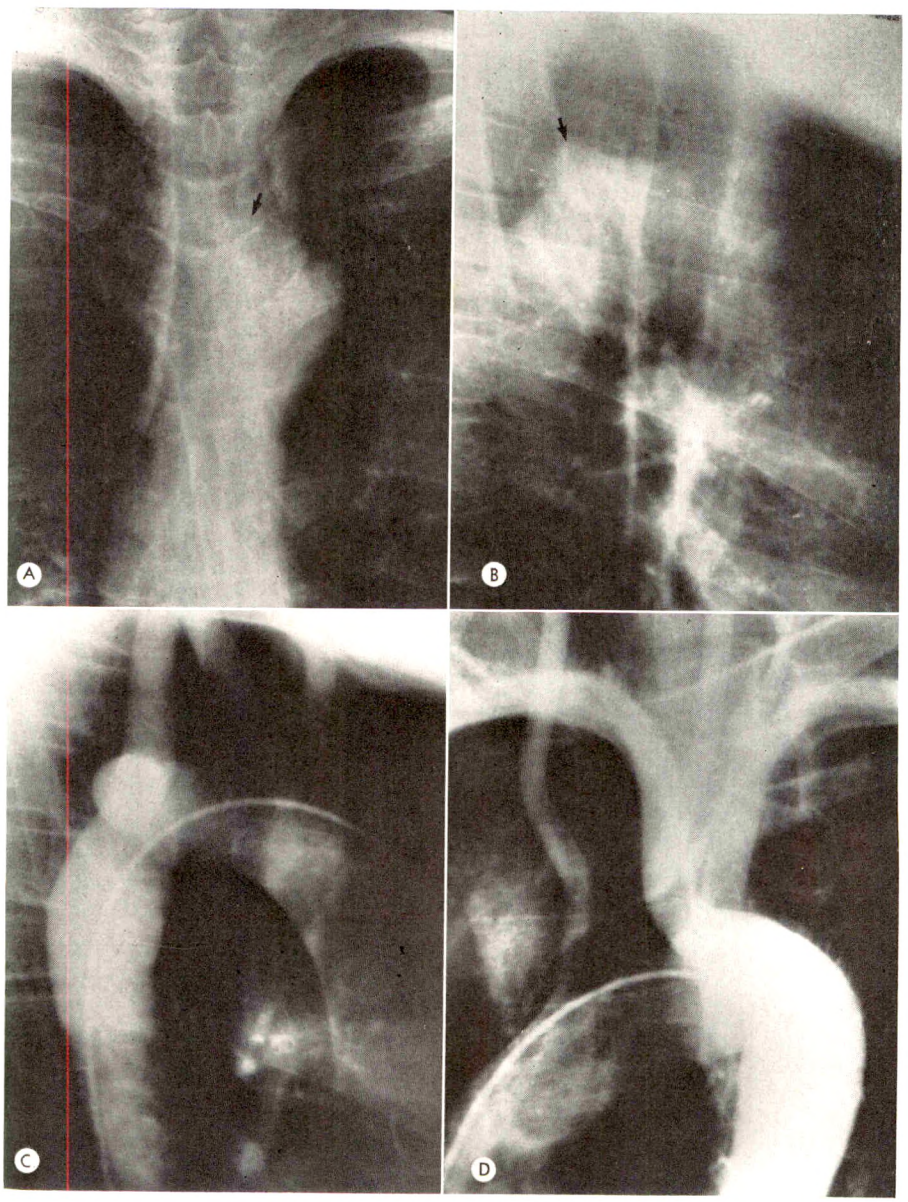


FIG. 1. Aberrant right subclavian artery. (*A*) Posteroanterior roentgenogram shows a soft tissue density which extends obliquely from the superior margin of the aortic arch into the mediastinal silhouette (arrow). (*B*) Lateral roentgenogram of the same patient demonstrates a rounded density continuous with the superior margin of the aortic arch (arrow). (*C*) Lateral and (*D*) right posterior oblique arch arteriograms of the same patient show that this density represents the origin of an aberrant right subclavian artery.

On lateral roentgenograms, 2 patients from the group of 7 demonstrated a rounded, localized density arising from the superior margin of the aortic arch (Fig. 1*B*; and 3*A*). Aortograms of each patient demonstrated the density to be an aberrant right subclavian artery (Fig. 1, *C* and *D*; and 3*B*).

CONCLUSION

An aberrant right subclavian artery may be visible on plain chest roentgenograms.

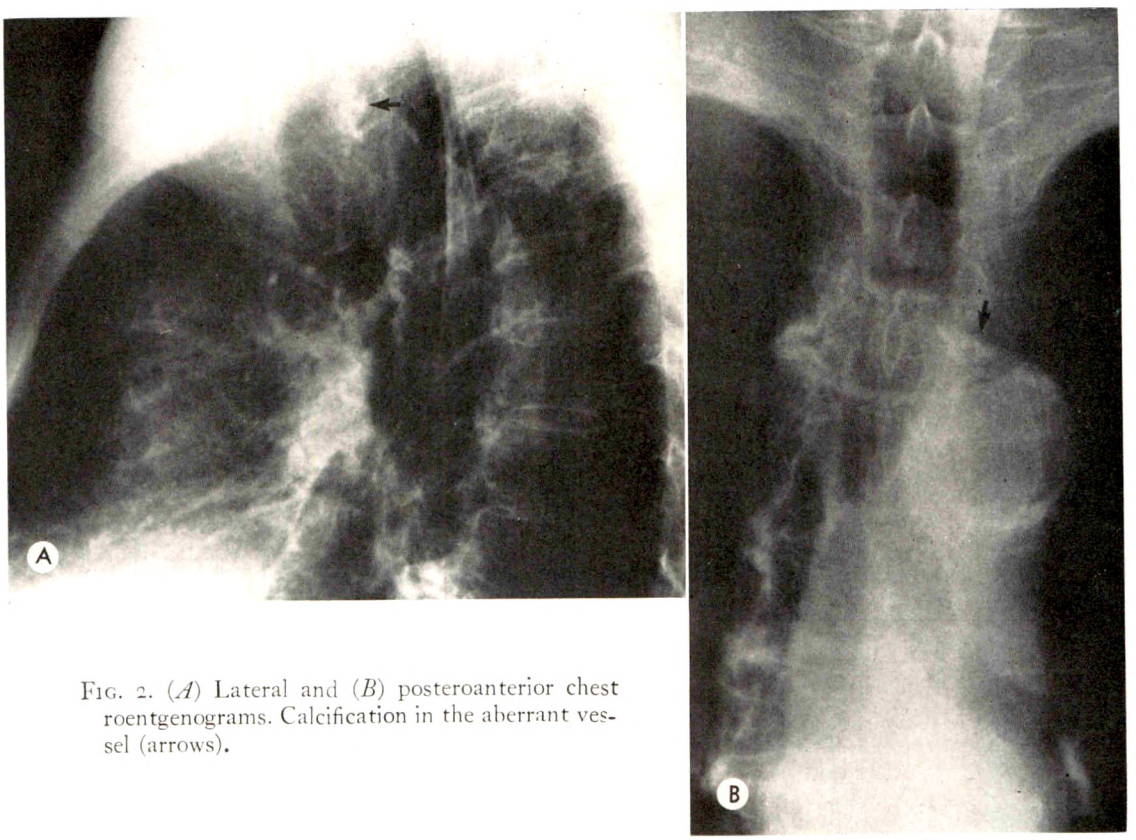


Fig. 2. (*A*) Lateral and (*B*) posteroanterior chest roentgenograms. Calcification in the aberrant vessel (arrows).

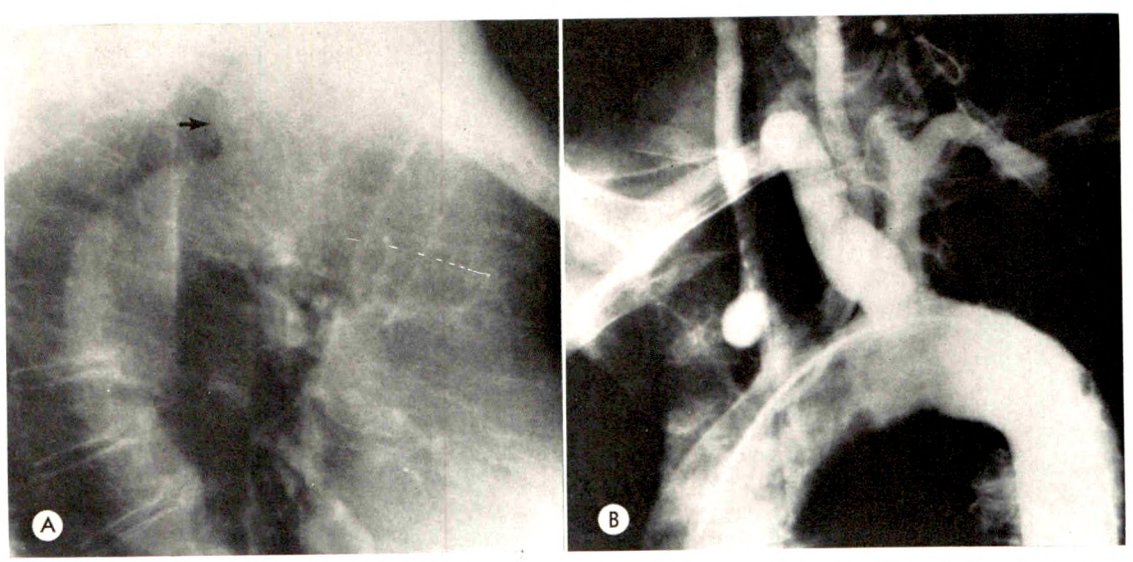


Fig. 3. (*A*) The aberrant right subclavian artery (arrow) is visible on plain lateral chest roentgenogram. (*B*) Arch aortogram, right posterior oblique projection, shows the artery with area of aneurysmal dilatation at its origin.

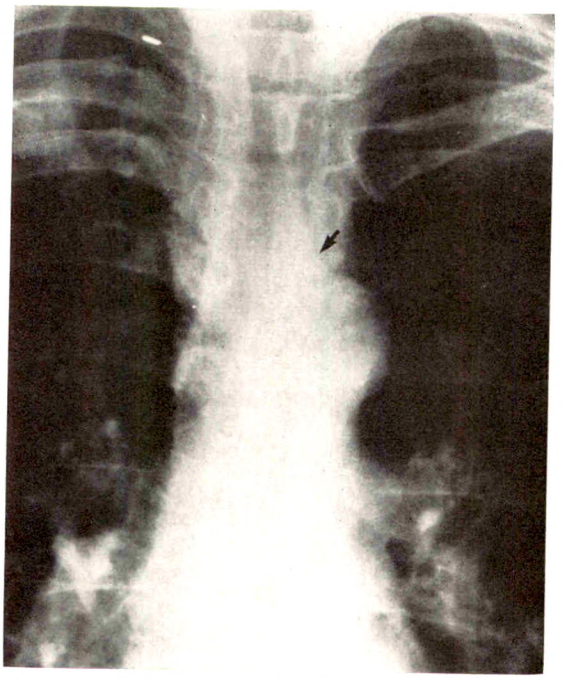


FIG. 4. Density (arrow) which mimics that of an aberrant right subclavian artery, in a patient with aortic arch branching, proved to be normal. This density is shorter and ascends on a steeper slope, approximately 45°, than that of an aberrant right subclavian artery.

For 2 of 12 patients, the lateral projection showed the aberrant right subclavian artery as a rounded, localized density continuous with the superior margin of the aortic arch, a finding previously undescribed. In 7 of 12 patients, the posteroanterior projection demonstrated a density ascending obliquely into the mediastinum from the superior margin of the aortic arch. In 80 control patients with normally-originating right subclavian arteries, a similar, but usually shorter oblique density was occasionally seen in the frontal projection.

Recognition of the appearance of aberrant right subclavian artery on plain chest roentgenograms may prevent its confusion with other causes of mass densities in the superior, middle mediastinum.

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ANGIOGRAPHIC DEMONSTRATION OF COLLATERAL CIRCULATION IN A PATIENT WITH THE SUPERIOR VENA CAVAL SYNDROME*

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AN infusion excretory urography was performed on a 29 year old female patient with known superior vena caval obstruction secondary to anaplastic carcinoma involving the anterior mediastinum. Part of the collateral venous circulation was demonstrated on the "10 minute" film, which was exposed with the infusion still running (Fig. 1). The "20 minute" film revealed no trace of the collateral venous circulation. With the patient's permission, we obtained bilateral upper extremity venograms to better show the anatomy of her collateral circulation. Excellent visualization of almost all the possible collateral pathways in superior vena caval obstruction was demonstrated (Fig. 2).

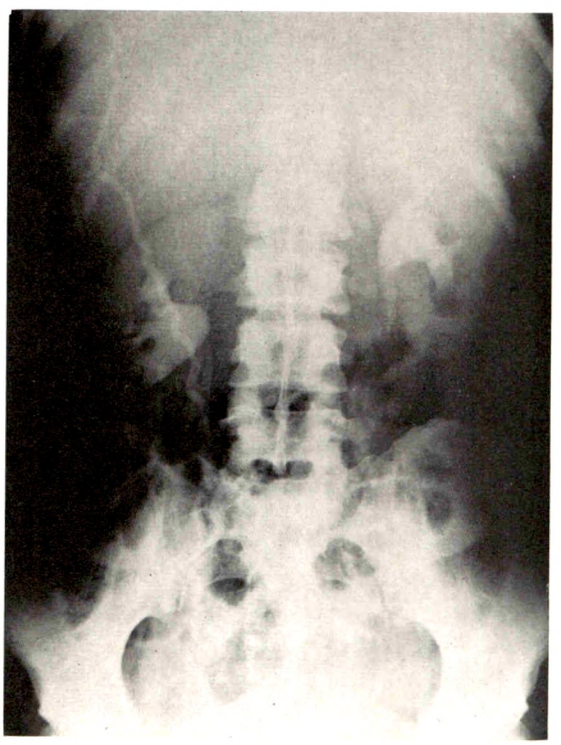


FIG. 1. Ten minute infusion excretory urogram, which demonstrates the thoracoepigastric and superficial inferior epigastric veins.

REPORT OF A CASE

A 29 year old white female presented November 28, 1972 at the University Hospitals with a 3 to 4 week history of swelling of her face, neck, chest and arms. This had been preceded by a 6 to 7 week history of chronic nonproductive cough and a feeling of tightness in her chest. At the same time she had had several episodes of low-grade fever without chills or sweats. One week prior to her admission the swelling of her face had become so severe that she had difficulty in opening her eyes. At this time she began to have shortness of breath, associated with chest pain which radiated to her back and lasted several hours. Her appetite had been good and she denied weight loss.

Past medical history. Gravida II, Para II, Aborta 0. She had no previous surgery or serious illness. There was no family history of heart disease, diabetes, cancer or tuberculosis.

Physical examination. The blood pressure was 120/78 mm. Hg in both arms. The pulse was 100/minute and the respirations 22/minute. She was thin with an obviously swollen face and neck, and erythema of the supraclavicular regions. A slight plethora of the thorax, head and upper extremities was also noted. A $1\frac{1}{2}\times 2$ cm. rubbery lymph node was palpable in the right supraclavicular area and a 3×3 cm. soft, tender mass overlapped the right clavicle. No axillary or inguinal lymph nodes were present. The breasts, lungs and heart were within normal limits. A prominent venous pattern was noted over the upper abdomen. The remainder of the abdominal examination and the genitalia were within normal limits.

Laboratory data. Analysis of the blood and urine was within normal limits. Fungal and

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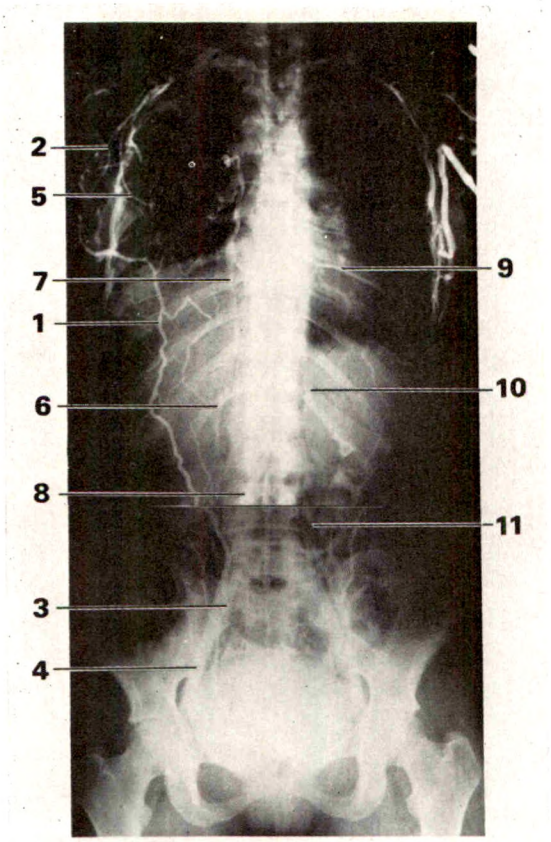


FIG. 2. Bilateral and antecubital fossa venogram demonstrating obstruction of both subclavian veins. The collateral circulation is demonstrated with eventual faint visualization of the inferior vena cava. The veins are: 1. thoracoepigastric vein; 2. lateral thoracic vein; 3. superficial inferior epigastric veins; 4. femoral vein; 5. costoaxillary veins; 6. superior epigastric vein; 7. internal thoracic vein (internal mammary vein); 8. inferior vena cava; 9. posterior intercostal vein; 10. hemiazygos vein; 11. left ascending lumbar vein.

tuberculosis skin tests, bone marrow examination and bone, liver, and thyroid scans were all normal.

Chest roentgenograms. This revealed a right mid-anterior mediastinal mass at the level of the carina. The lung fields were normal and no pleural effusion was demonstrated.

Course. On November 30, 1972, the patient underwent supraclavicular and anterior tracheal lymph node biopsies and a thrombosed segment of the external jugular vein was removed. The pathologic findings consisted of chronic inflammation of the lymph nodes and a thrombus in the external jugular vein without evidence of tumor. On December 11, 1972, she had a right thoracotomy. A solid firm mass of tumor was found, which entirely encased the structures of the mediastinum. The superior vena cava was thrombosed to the level of the right atrium. The trachea and right main stem bronchus were surrounded with tumor. Frozen section at that time revealed an undifferentiated carcinoma, probably metastatic. The chest was closed and she had an uneventful postoperative course. Permanent pathologic sections revealed only undifferentiated carcinoma, probably metastatic. She was referred to the Department of Urology on January 9, 1973 for an evaluation of her urinary tract as part of the search for her primary lesion.

DISCUSSION

Collateral circulation from the upper part of the body develops because of congenital variation, obstruction, displacement and dilatation of the superior vena cava.[6] Congenital causes are variations of the venous drainage into the right atrium such as situs inversus, dextrocardia, persistent left superior vena cava, anomalous pulmonary veins and various congenital cardiac anomalies. Obstruction occurs because of mediastinal and pulmonary tumors. Displacement may occur with mediastinal tumors, syphilitic aortitis and aortic aneurysms. Enlargement of the thyroid gland, surgical procedures in the chest, atelectasis and emphysema may also cause displacement of the superior vena cava or innominate veins. Dilatation of the superior vena cava occurs secondary to right-sided heart failure of any cause.

There are 4 main pathways of collateral circulation: internal mammary; vertebral; azygos; and lateral thoracic routes.[1,3,5]

The internal mammary pathway consists of the internal mammary, superior and inferior epigastric, musculophrenic, intercostal and superficial veins of the thorax to the iliac veins and the inferior vena cava.

The vertebral pathway is made up of the innominate, vertebral, intervertebral and vertebral plexus, intercostal, lumbar and

sacral veins to the azygos and internal mammary veins. Blood from the dural sinuses may also take this pathway.

The azygos pathway consists of the azygos, hemiazygos, ascending lumbar and lumbar veins connecting the superior and inferior vena cava.

The lateral thoracic pathway consists of the lateral thoracic, thoracoepigastric, superficial epigastric, superficial circumflex, long saphenous, femoral veins and finally the inferior vena cava.

There is no standard route for the collateral circulation to take. The internal mammary pathway connects with the vertebral and azygos systems. Both the azygos and lateral thoracic systems connect with the inferior vena caval system. Therefore, several routes or combinations are available.[5]

While combinations of these routes are no doubt used, Klassen *et al.*[4] have pointed out that the predominant collateral pathway depends on the level of obstruction. With obstruction above the azygos-superior vena caval anastomosis, the main pathway to the right atrium from the upper extremities and head is *via* tributaries of the subclavian vein to the intercostal veins to the azygos vein to the superior vena cava. When the orifice of the azygos vein is involved, the blood flow is reversed and the blood is directed to the inferior vena cava. Blood flow with obstruction proximal to the azygos orifice has not been well delineated. Carlson[2] ligated the superior vena cava below the azygos orifice in 2 dogs; however, both dogs died in the immediate postoperative period. No one has attempted this procedure experimentally since 1934.

In our case the internal mammary, lateral thoracic and lower portion of the hemiazygos pathways were visualized. The vertebral route is most certainly used by this patient, but was not visualized because contrast medium was not injected into the cerebral circulation. In this type of obstruction the return flow from the head would have to go *via* the dural sinus to the vertebral circulation. The flow would then go into the intercostal and lumbar veins to the azygos, hemiazygos system and finally to the inferior vena caval system. The superficial blood flow from the scalp and face would be routed to collaterals about the shoulders and then into the lateral thoracic pathway. Another route would be by way of the superior posterior intercostal veins *via* superficial circulation of the superior posterior thorax.

Following the injection of the contrast medium into the right median cubital vein, the basilic and axillary veins are not seen; however, there is good visualization of the collateral veins about the right shoulder. The lateral thoracic vein and costoaxillary veins are well visualized. Contrast medium flows through the costoaxillary veins to the internal mammary vein and then into the internal mammary pathway.

Following injection of contrast medium into the left median cubital vein, the basilic and axillary veins are well seen. There is an abrupt occlusion of the axillary vein and contrast medium is then routed through the lateral thoracic pathway. The lateral thoracic vein also communicates with the seventh, eighth and ninth posterior intercostal veins on the left and then enters into the hemiazygos vein. The azygos, accessory hemiazygos and upper portion of the hemiazygos veins are not visualized because nonopacified blood is directed caudally in this system. This does not allow opacified blood to enter these veins in sufficient quantity for them to be visualized.

SUMMARY

A well documented case of complete superior vena caval obstruction by malignant mediastinal tumor is described.

Three of the possible venous collateral pathways are demonstrated by bilateral arm venography. Of added interest is the incidental partial demonstration of the collateral flow during infusion pyelography.

We were unable to find as detailed an

anatomic demonstration of the collateral circulation in the literature in a single patient.

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GUNSHOT WOUNDS OF THE AORTA WITH PERIPHERAL ARTERIAL BULLET EMBOLISM*

REPORT OF 2 CASES

By CYRUS P. KLEIN, M.D.†
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IN a civilian community supplied with small caliber hand guns that discharge low velocity bullets, it is possible to have occasion to see a patient in whom such a bullet has entered the aorta and spent its projectile energy within the lumen of this vessel. Bullet penetration of the aorta is not always fatal. If the projectile is small enough, and the arterial system elastic enough, the wound of entry into the arterial channel may close without exsanguination. A small projectile thus captured will tumble along intraluminally with the current of blood flow until it is swept far enough away to be arrested by the diminishing diameter of the peripheral vasculature.

The occurrence of this phenomenon is rare. In 1968, Trimble[17] summarized the cases published until that time. There were 33 going back to 1885. He added 2 additional cases. Generally, publication is in a surgical journal and consists of a single case report. We are not aware of this phenomenon being presented previously in a radiology journal. A report of 2 cases of this interesting condition occurring within a 2 month period is submitted.

REPORT OF CASES

CASE 1. P.J.G., a 22 year old Caucasian female, was admitted to Wadley Hospital on August 1, 1972, 6½ hours after having been shot in the left shoulder with a .22 caliber short missile. There was an obvious wound of entrance but no wound of exit. The patient had no blood pressure or pulse in the right arm. There was hemopneumothorax on the left. The right lung was well expanded and showed no abnormality. The bullet was seen in the right axilla. The cardiac silhouette was not enlarged (Fig. 1).

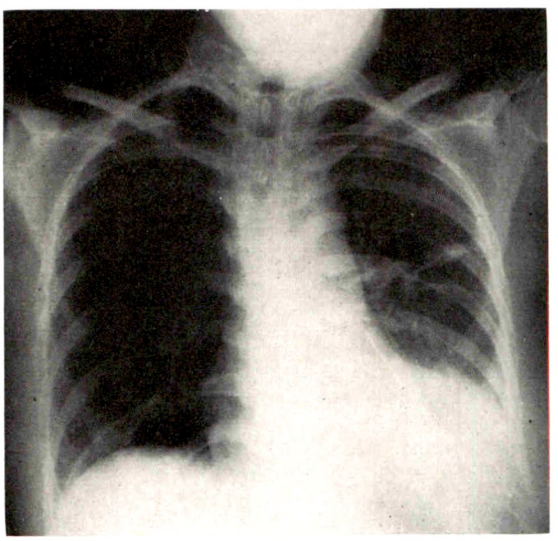


FIG. 1. Case 1. A bullet has entered the left side of the chest leaving a trail of contusion in the left lung and hemorrhage in the left pleural space. The right lung was not injured.

A suction tube was placed in the left pleural space for decompression. Esophagographic examination showed no abnormality. Arch aortography was done using the Seldinger method of entry into the arterial system through the left femoral artery. The bullet, which was observed in the right axillary area, was confirmed to be within the right axillary artery and was occluding it (Fig. 2).

Without further delay, the patient was taken to surgery. A small incision was made over the right axillary artery. There was no hemorrhage or ecchymosis in this area. The right axillary artery had not been damaged. A palpable foreign body was found within the lumen of the vessel. Arteriotomy was performed and the small bullet was removed. Excellent peripheral pulse was established.

The patient received transfusions because of hemorrhage from her left lung. The hemothorax and pulmonary contusion cleared rapidly

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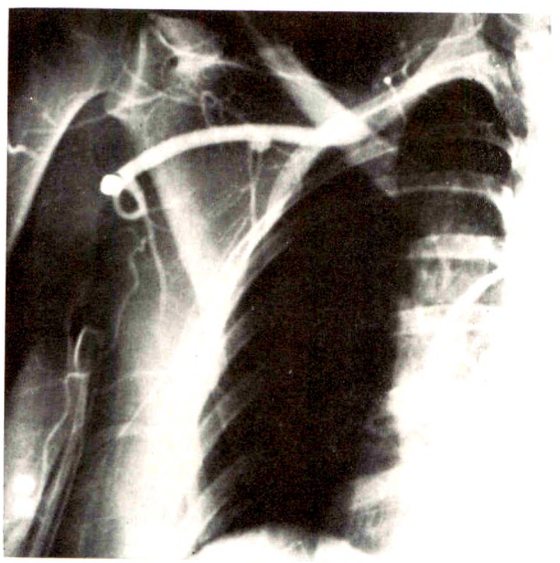


Fig. 2. Case I. Aortic arch angiogram shows the bullet embolized to the right brachial artery.

and the chest tube was removed 5 days after it had been inserted. The patient was discharged on the 7th day after admission. The wound of entry into the arterial system was not explored, but the tract of contusion in the left lung seen on the roentgenogram led from the left axilla to the mediastinum at the level of the base of the ascending aorta.

Case II. L.T., a 26 year old Negro male, was admitted to Wadley Hospital on September 20, 1972, after having sustained a .22 caliber gunshot wound in the right side of the chest earlier in the evening. No bullet was demonstrable in the roentgenograms of the chest or abdomen. There was no wound of exit. The patient stated that he was running after he had been shot and he thought that he had hurt his left leg.

Examination showed a patient who was alert, well oriented, and in no acute distress, but he complained of abdominal pain. His chest was clear at physical examination. There was abdominal rigidity and slight rebound tenderness. Good pulses were felt in the groin, both popliteal arteries and in the right foot. There was no pulse in the left foot.

A roentgenogram of the left leg showed a bullet lodged below the left knee (Fig. 4). A chest roentgenogram was made with a coin strapped to the right lower thorax to mark the point of entry of the bullet (Fig. 3). Increased density in the right lower lung was attributed to intrapulmonary hemorrhage. The left lung was clear.

Abdominal exploration was made using a right thoracoabdominal incision. A perforation was found in the lateral portion of the diaphragm and there was perforation of the right lobe of the liver. Blood was in the retroperitoneal area surrounding the aorta in the region of the celiac axis, and there was blood in the retroperitoneal portion of the right flank surrounding the kidney. The point of entry of the bullet into the aorta could not be seen by the surgeon. Drains were placed in both sides of the chest and in the right infrahepatic area.

Following this procedure, a medial incision was made below the left knee. When the gastrocnemius muscle was retracted posteriorly, the bullet could be palpated within the popliteal artery. The artery was opened and the bullet extracted, but Fogarty catheters had to be passed proximally and distally to remove a thrombus before good flow could be established in the popliteal artery.

The patient made a satisfactory recovery without any postoperative complications. He was discharged on the 16th day with all drains and sutures removed.

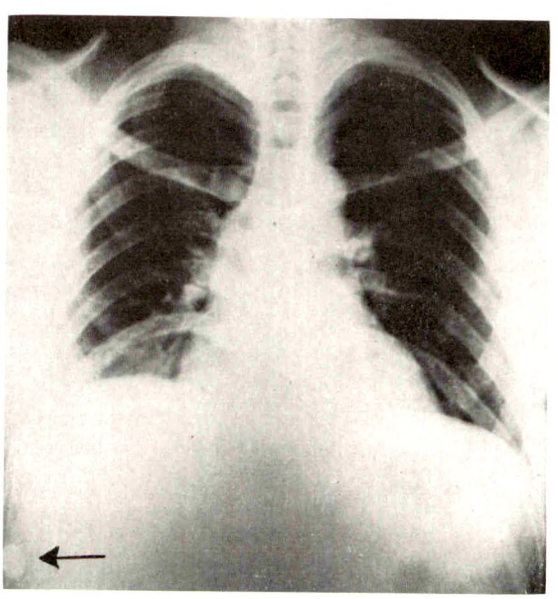


Fig. 3. Case II. A coin strapped to the skin, indicated by an arrow, marks the wound of entry of a bullet which perforated the right leaf of the diaphragm and the right lobe of the liver. Retroperitoneal hemorrhage and right lower lobe pulmonary contusion were present.

DISCUSSION

Our 2 cases were young individuals, and, in the other reported cases, survivors of penetrating aortic bullet wounds also were youthful. An elastic aorta would seem to be essential to avoid fatal hemorrhage.

Trimble's[17] collected series gathered from the literature shows bullet embolization 3 times more frequent in the lower extremities than in the upper extremities. The forces acting on a migrating embolus which determine where it goes are the force of blood flow, gravity, and position of the body. Embolic missiles that enter the left side of the aortic arch or the abdominal aorta may be expected to be found in a lower extremity. Those that enter the arterial system through the left side of the heart or the right side of the aortic arch have the chance of going to either an upper or lower extremity.

In our second case the bullet embolized to the left popliteal artery. The right and left common iliac arteries arise from the bifurcation at different angles. Attention to this has been drawn by Garzon and Gleidman,[2] and by Keeley.[6] The left artery is 30° from the midline, more nearly a straight continuation of the aorta than the right iliac which is 45° from the midline. Emboli to the lower extremities are 3 times more frequent on the left side than on the right side.

The importance of prompt removal of the peripherally located missile after embolism is generally stressed in the literature. Gangrene may supervene if embolectomy is delayed. In the summary of cases presented by Keeley[6] in 1951, there were 10 survivors in 29 cases. All of the survivors had embolectomy, but, not withstanding this, 4 of the 10 required amputation. Movin *et al.*,[10] and Painter and Britt[13] concluded that time itself is not the critical factor in survival of an extremity after embolus, but whether clot propagation occludes both the deep and superficial femoral arteries. Larn and McIntyre's[9] unusual case was an exception and did not have the bullet embolus discovered in the leg until

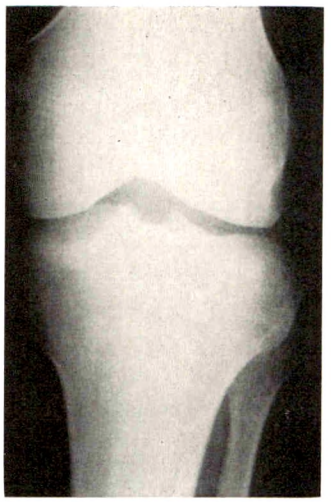


Fig. 4. Case II. The bullet was discovered embolized to the left popliteal artery.

10 months after their patient, a 16 year old boy, was shot. Our second case more characteristically showed intraluminal clot developing within a few hours.

SUMMARY

1. Two cases of gunshot wound of the aorta with peripheral arterial bullet embolism are reported.

2. Survival after bullet perforation of the aorta appears to need the elasticity of youth to permit the wound of the vessel to close without immediate hemorrhagic death.

3. Whenever a patient with an abdominal or thoracic gunshot wound of entry, and no wound of exit, does not have the projectile in the area of the wound, suspicion of embolization should be entertained.

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THE SPLEEN AND CONGENITAL HEART DISEASE*

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THE association of cardiac malposition and congenital heart disease with splenic anomalies (specifically either asplenia or polysplenia) has been known since Ivemark's classical paper in 1955.[6] During the ensuing years, other visceral anomalies such as malrotation of the gut, horizontal liver, interruption of the inferior vena cava with azygos continuation, and symmetry of the lungs have been described as occurring frequently with these developmental complexes.[1,2,8–10,12–15]

We have studied 41 patients with proven splenic anomalies coexisting with congenital heart disease, to determine the incidence of various visceral and cardiac anomalies, and to characterize and distinguish the roentgenographic features of the asplenic and polysplenic syndromes.

MATERIAL AND METHOD

Forty-one patients with congenital heart disease associated with either asplenia or polysplenia were reviewed in a retrospective study at the University of Minnesota Hospitals. The records of the patients were studied to obtain data of the clinical history and physical examination. Thoracic roentgenograms were reviewed to determine the state of the pulmonary vasculature and the location of the cardiac apex, stomach, liver, and great vessels. Angiograms were studied particularly for the position of the inferior vena cava and thoracic aorta. Finally, operative records and autopsy protocols were studied to define the anatomic details.

The asplenia syndrome was considered present if no spleen was found at autopsy or if Howell-Jolly bodies were present in a peripheral blood smear. Polysplenia was diagnosed if multiple splenic masses were found either at autopsy or on angiography of the celiac axis.

To assist in understanding cardiac malpositions, Elliott *et al.*[4] suggested the principle of concordance. In normal individuals, and most patients with cardiac malposition, the inferior vena cava, liver, and anatomic right atrium are present on one side of the body; the aorta, stomach, and anatomic left atrium on the opposite side. If the inferior vena cava is on the right side, situs solitus is present; if on the left, situs inversus is present.

In patients in whom the rules of concordance do *not* follow, situs cannot be determined; and often a splenic anomaly is present. Such patients frequently have abdominal heterotaxia, meaning the anomalous placement of organs or major blood vessels within the abdomen. Normally the inferior vena cava, major portion of the liver, cecum, appendix, and gallbladder are on one side of the abdomen, while the stomach, spleen, descending and sigmoid colon are on the contralateral side. Innumerable combinations of malpositioned abdominal organs may exist such as midline liver, aorta, and inferior vena cava lying side by side or crossing each other, stomach and liver on the same side (called "transposition of the stomach"), the cecum and sigmoid on the same side, etc.

The information about the patients with asplenia will be considered separately from that of polysplenia. Furthermore, anatomic information will be presented first, then roentgenographic correlations.

RESULTS

Among the 41 patients studied, 19 patients with asplenia were found and 22 patients with polysplenia.

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TABLE I

CHARACTERISTICS OF ASPLENIA

(19 Cases)

Abnormality	Present	Absent	Unknown
Cardiac			
Common atrium or ASD	18 (85%)	1	0
Common ventricle or VSD	19 (100%)	0	0
Bilateral SVCs	11	6	2
Endocardial cushion defect, AV canal (complete or partial), common AV valve	14	3	2
"l" transposition of GVs with PS or PA	10 (89%)	9	0
"d" transposition of GVs with PS or PA	7	12	0
TAPVC	10 (53%)	9	0
Partial APVC	1	18	0
Absent coronary sinus	5	10	4
Dextrocardia	8	11	0
Pulmonary			
Bilateral trilobed lungs	13	3	3
Decreased pulmonary vasculature (roentgenographically)	14 (77%)	4	1
Abdominal			
Abdominal heterotaxia	18 (95%)	1	0
IVC and descending aorta on same side of spine	9 (47%)	10	0
Malrotation of gut	9	9	1
Horizontal liver	16 (84%)	3	0
Discordance apex—stomach bubble	12	7	0
Miscellaneous			
Howell-Jolly bodies seen in peripheral blood smear	13	—	—

ASD = atrial septal defect; VSD = ventricular septal defect; SVC = superior vena cava; AV = atrial ventricular; "l" = levo; GV = great vessels; PS = pulmonary stenosis; PA = pulmonary atresia; "d" = dextro; TAPVC = total anomalous pulmonary venous connection; IVC = inferior vena cava.

ASPLENIA

In the 19 patients with asplenia the diagnosis was established at necropsy in 13, and in the other 6 asplenia was diagnosed by the presence of Howell-Jolly bodies. The pertinent findings are summarized in Table 1.

Invariably a severe cardiac malformation was present, and each of the patients was cyanotic. Among the 19 patients, there were 15 males and 4 females. Seventeen of the patients were initially studied in the first year of life, usually within the first month of life.

In each patient either a ventricular septal defect or single ventricle was present, and in all but 1 either a common atrium or atrial septal defect. Often these defects were portions of an endocardial cushion defect (14 cases). Seventeen of the 19 showed transposition of the great vessels, which was either of the "l" or "d" type. In each of these 17, pulmonary stenosis or pulmonary atresia was coexistent (Fig. 1, *A* and *B*). Total anomalous pulmonary venous connection either to the superior vena cava or to an infradiaphragmatic site was found in 10 patients (53 per cent) (Fig. 2, *A–C*).

Examination of the lungs showed 3 lobes in each lung in each of the necropsied patients. In these patients bilateral epiarterial bronchi were found.

Various types of abdominal heterotaxia were found in 18 patients, while in the remaining patient, the abdominal contents

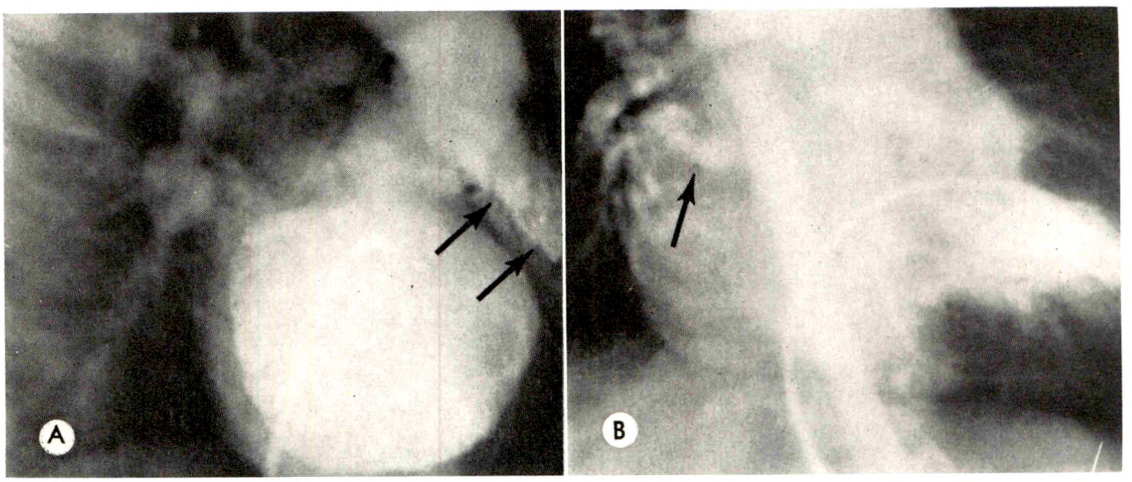


Fig. 1. (*A*) Lateral and (*B*) anteroposterior ventriculograms showing single ventricle, "l" transposition of great vessels, and pulmonary atresia. Pulmonary arteries opacify *via* bronchial arteries (single arrow). Note also rudimentary chamber (double arrow).

were situs inversus. Most of these patients showed a horizontal liver and half of them malrotation of the intestine. The latter usually represented malrotation of the mid-gut loop. In the abdomen, the inferior vena cava and descending aorta were ipsilateral in 9 patients.

Roentgenographic correlation. Thoracic roentgenograms usually showed either a horizontal liver or a discordant relationship between the gastric air bubble and the cardiac apex (Fig. 3; and 4). Dextrocardia was seen in 8 patients and obvious discordance of apex and stomach in 12 patients. Furthermore, these roentgenograms showed diminished pulmonary vasculature in 77 per cent of the patients. The presence of 3 lobes in each lung cannot be diagnosed unless middle lobe fissures can be seen bilaterally (Fig. 5).

Abdominal venous angiography may show an abnormal interrelationship between the inferior vena cava and abdominal aorta. These should be on opposite sides of the abdomen, and we have found them ipsilaterally only in asplenia (Fig. 6).

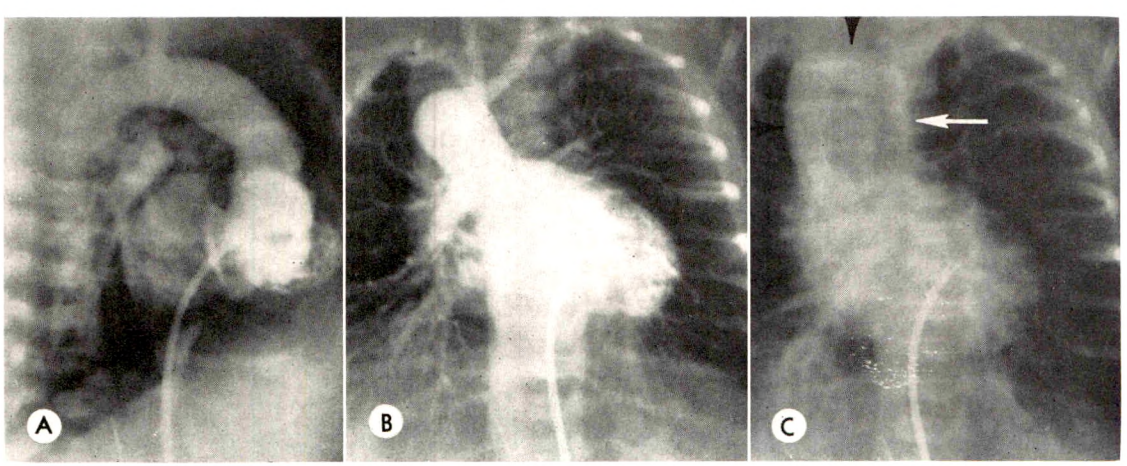


Fig. 2. (*A* and *B*) Asplenia with "d" transposition of great vessels. Single ventricle and pulmonary atresia. (*C*) Late roentgenogram shows total anomalous pulmonary venous connection to superior vena cava (arrows).

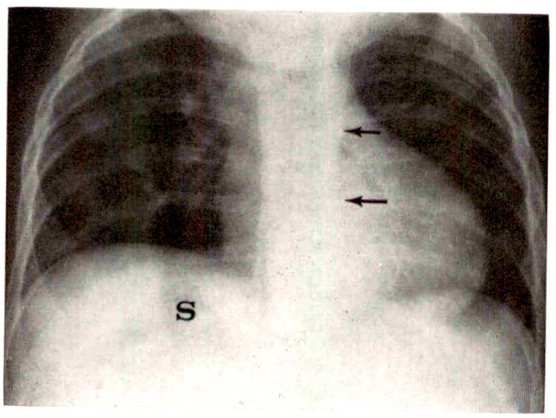


FIG. 3. Typical thoracic roentgenogram in asplenia. Horizontal liver and discordance of aorta (arrows) and gastric bubble (S).

Barium studies have demonstrated the malrotation of the gastrointestinal tract in several of these cases (Fig. 7, *A* and *B*).

POLYSPLENIA

Of the 22 patients with polysplenia, 20

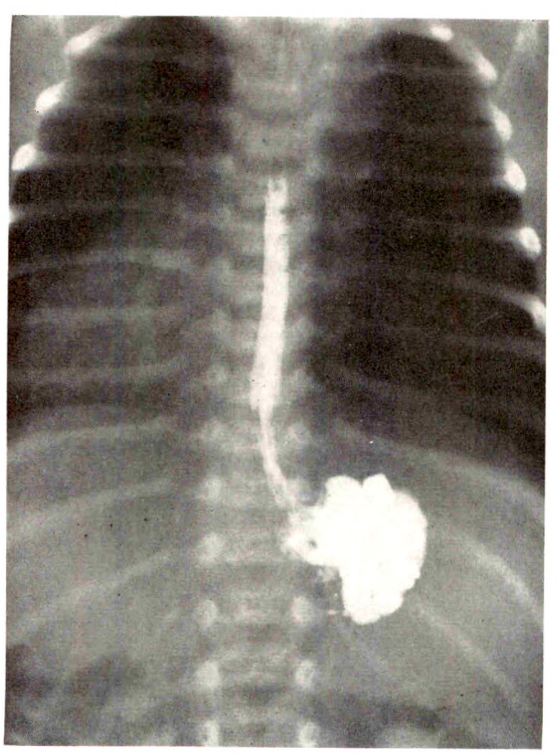


FIG. 4. Thoracic roentgenogram in patient with asplenia. Aorta and gastric air bubble on the left (concordant) but horizontal liver indicates abdominal heterotaxia.



FIG. 5. Portion of thoracic roentgenogram in patient with asplenia. Minor fissures (arrows) are evident in both lungs.

were proven by autopsy and 2 by angiographic demonstration of multiple splenic masses. The pertinent data are shown in Table II.

Among the 22 patients, there were 12 males and 10 females. Eighteen (82 per cent) of the patients were cyanotic, but usually not to the degree found in patients with asplenia. Many of these patients were older than the patients with asplenia suggesting that the cardiac disease was not as severe.

Communications at the atrial and ventricular level were common: 16 showed an atrial septal defect or common atrium, and

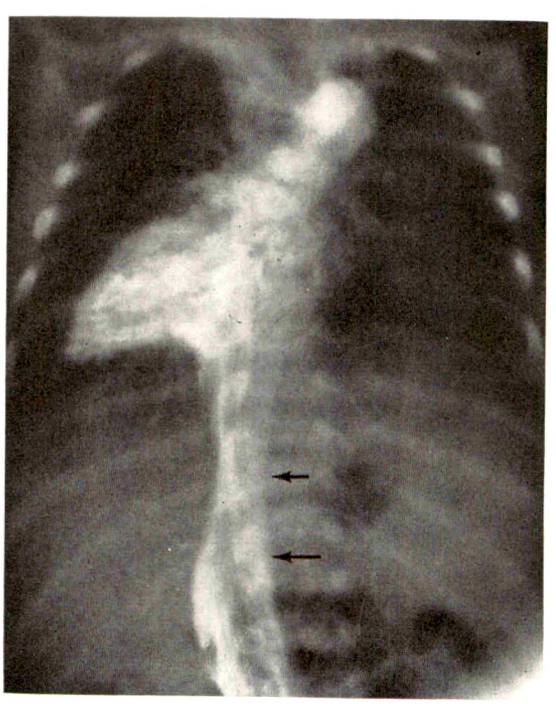


FIG. 6. Venous angiogram from lower extremity in patient with asplenia. Both the inferior vena cava and descending aorta are on the right side (arrows).

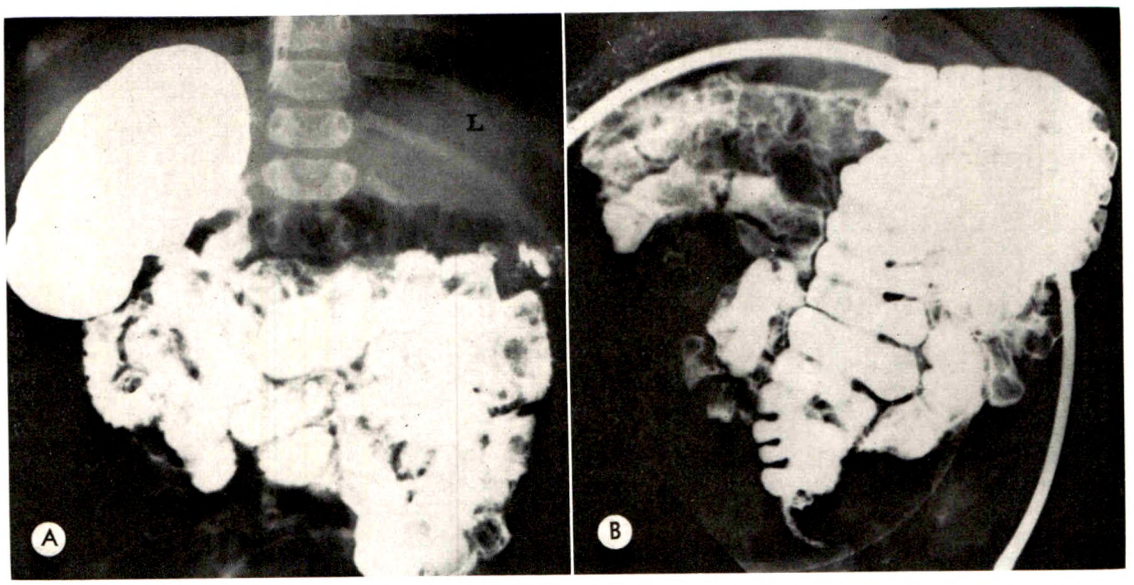


FIG. 7. Upper gastrointestinal series in asplenia. (*A*) Early and (*B*) late views. Malrotation of the gut. The entire colon is on the left side of the abdomen.

TABLE II

CHARACTERISTICS OF POLYSPLENIA

(22 Cases)

Abnormality	Present	Absent	Unknown
Cardiac			
Common atrium or ASD	16 (73%)	6	0
Common ventricle or VSD	17 (80%)	5	0
Bilateral SVCs	11 (50%)	11	0
Interruption of hepatic segment of IVC with azygos continuation	10	11	1
Absent coronary sinus	8	14	0
TAPVC	6	16	0
Partial APVC	7	15	0
Endocardial cushion defect	6	16	0
Dextrocardia	8	14	0
Pulmonary			
Bilateral bilobed lungs	7	14	1
Increased pulmonary vasculature	12 (55%)		
Decreased pulmonary vasculature	1		
Normal pulmonary vasculature	7		
Venous obstruction	2		
Abdominal			
Abdominal heterotaxia	14 (64%)	7	0
Malrotation of gut	11 (50%)	11	0
Horizontal liver	6 (27%)	16	0
Absent gallbladder	2	18	2
Discordance apex—stomach bubble	12	10	0

For abbreviations see Table I.

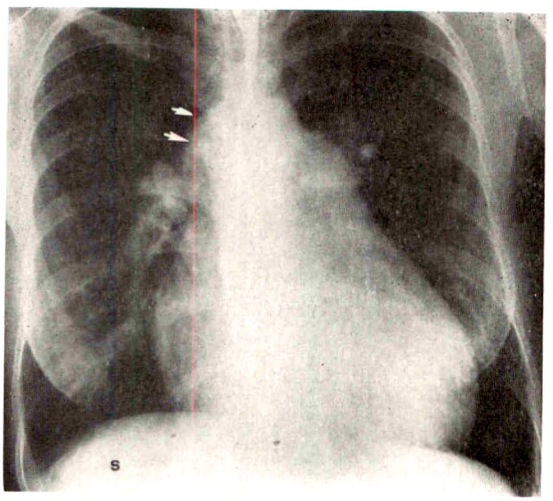


FIG. 8. Thoracic roentgenogram of patient with polysplenia, showing cardiomegaly and increased pulmonary vasculature. Cardiac apex, aorta, and gastric air bubble (S) are discordant and there is enlarged azygos vein on the right due to interruption of the inferior vena cava (arrows).

17 showed a ventricular septal defect or single ventricle. Six of these patients revealed an endocardial cushion defect. In contrast to asplenia, neither transposition of the great vessels nor obstruction to pulmonary outflow were found. The cyanosis seen in 18 (82 per cent) of the patients was probably due to the admixture lesions and possibly pulmonary vascular disease.

Bilateral superior venae cavae were found in 11 patients, and interruption of the inferior vena cava with azygos continuation was present in 10. Anomalies of pulmonary venous connection were found in 13 patients, being total in 6 and partial in 7. The anomalous connection was directly to the atrium in each instance.

A third of the patients had 2 lobes in each lung and bilateral hypoarterial bronchi. Two-thirds of the patients revealed abdominal heterotaxia, and half showed malrotation of the gut. A horizontal liver was found in only 6 patients.

Roentgenographic correlation. Thoracic roentgenograms revealed increased pulmonary vasculature in 12, 7 with normal pulmonary vasculature, 2 with pulmonary venous obstruction, and decreased pulmonary vasculature in the remaining patients.

On the thoracic roentgenogram, the finding of a mass density opposite the aortic arch representing an enlarged azygos vein is very significant (Fig. 8). Angiographic demonstration of this anomaly results in a "candy cane" appearance in the lateral view (Fig. 9, *A* and *B*). Also, the finding of a discordant gastric bubble on the thoracic roentgenogram may lead one to consider the diagnosis of polysplenia. Dextrocardia was seen in 8 patients, and discordance of stomach and cardiac apex was seen in 10 patients.

Isotope scans have not proved to be helpful to us in delineating the splenic mass because of the poor resolution of this technique and the similar appearance of the closely located multiple spleens to a normal spleen (Fig. 10). In our experience the technetium 99m pertechnetate scans were further complicated by the superimposition of a midline liver in several of the cases. A very definite method of demonstrating the multiple spleens is by selective celiac angiography[16] (Fig. 11, *A* and *B*).

DISCUSSION

The polysplenia and asplenia syndromes represent infrequently occurring but interesting developmental complexes which can be suggested by the radiologist from his interpretation of thoracic roentgenograms. The practical importance of recognizing the abnormal splenic state is the ability to predict the form of coexistent cardiac defect with considerable accuracy.

These syndromes represent developmental deviations occurring at an early embryonic stage. Apparently there is a failure to develop the normal pattern of body asymmetry so that individuals with a splenic anomaly show symmetry of organ development. Bilaterally placed organs are identical, and unilateral organs may be duplicated or absent. Asplenia has been termed bilateral right-sidedness or dextroisomerism, since both lungs have 3 lobes and the spleen is absent.[7,14] Polysplenia, in contrast, has been called bilateral left-sidedness because each lung has 2 lobes, the spleens are multiple, and the hepatic seg-

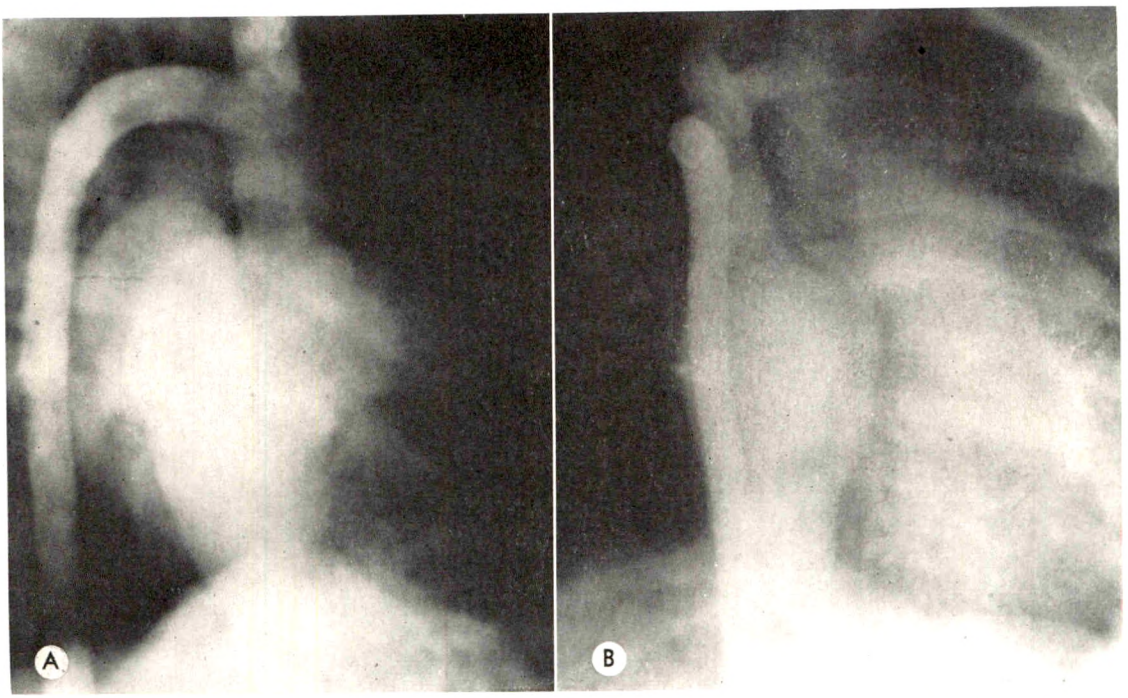


Fig. 9. (*A*) Lateral and (*B*) anteroposterior venous angiograms from saphenous vein in patient with polysplenia. Typical "candy cane" appearance of azygos continuation of interrupted inferior vena cava in lateral view.

ment of the inferior vena cava is absent and occasionally the gallbladder is absent.[10]

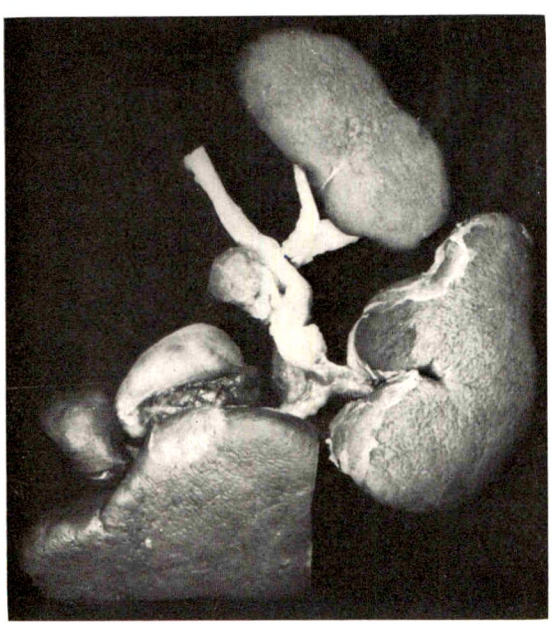


Fig. 10. Anatomic appearance of multiple spleens. (Reproduced by permission of The American Heart Association, Inc., Moller *et al.*, and *Circulation*.[10])

As a result of the symmetric development, the normal concordance of organ relationship is lost, and it is difficult to determine sides (*i.e.*, right or left). Abdominal heterotaxia is present, and in fact this has been called "heterotaxy syndrome" by some authors.[5]

The electrocardiogram may also be helpful in predicting asplenia or polysplenia. Several papers have discussed the evaluation of the P-wave axis and pacemaker sites.[3,14]

When analyzing a thoracic roentgenogram, particularly if cardiac malposition is evident, the presence of concordance should be established. This may be difficult if the gastric bubble cannot be identified. If it can be seen, and found discordant, abdominal heterotaxia may be recognized, and an abnormal splenic state suspected.

Further diagnostic clues may allow distinction of asplenia from polysplenia. Among patients with asplenia the liver almost always assumes a horizontal position across the upper abdomen, and the stomach may or may not appear discordant.

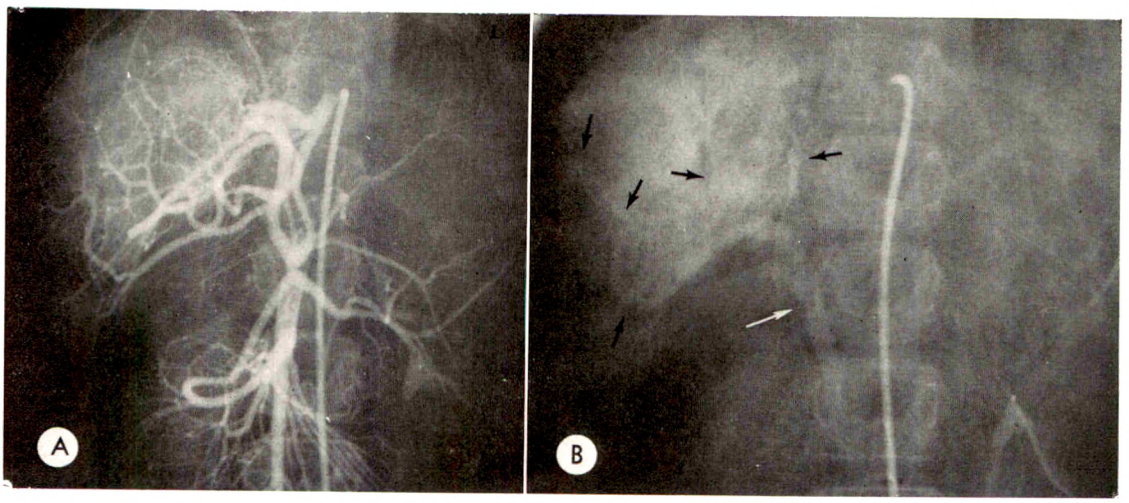


FIG. 11. (*A*) Selective arteriogram in patient with polysplenia showing common trunk for superior mesenteric artery and celiac axis. (*B*) Cluster of multiple spleens well demonstrated in the right upper quadrant (arrows). The splenic vein is well opacified (white arrow).

This hepatic finding in a cyanotic infant with diminished pulmonary blood flow on the thoracic roentgenogram raises the index of suspicion of asplenia considerably.

The congenital cardiac malformations in these patients are invariably complex, but they clinically and hemodynamically mimic tetralogy of Fallot with a right-to-left shunt at the ventricular level. The basic physiologic abnormality of the ventricular communication, transposition, and pulmonary stenosis or atresia is identical to that of tetralogy. Despite the frequent occurrence of atrial anomalies, these are not clinically significant.

The angiographic findings of a tetralogy variant with total anomalous pulmonary venous connection strongly indicate a splenic anomaly. Another feature suggesting this diagnosis is the side-by-side position of the inferior vena cava and abdominal aorta.

Usually these patients are seen in infancy. The radiologist suspecting the diagnosis of asplenia on the basis of his roentgenographic findings should suggest that Howell-Jolly bodies be sought. Many physicians have a fatalistic view of these patients because of the complexity of the cardiac defects, but they can be palliated by systemic-pulmonary shunt procedures.

Polysplenia represents a more variable syndrome than asplenia. The important clues on the thoracic roentgenogram are the findings of abdominal heterotaxia, cardiac malpositions, and a prominent azygos vein. The angiographic demonstration of interruption of the inferior vena cava assists in making this diagnosis.

The location of the inferior vena cava has great significance in establishing situs, either normal situs solitus, situs inversus, or abdominal heterotaxia. Conversely, the superior vena cava is much less significant since there is an increased incidence of bilateral superior vena cava in both asplenia and polysplenia. The importance of the inferior vena cava suggests that when there is a question of splenic anomaly related to congenital heart disease, a forward angiography from the lower inferior vena cava would be very helpful in establishing the location of the inferior vena cava and how it enters the heart. It may enter *via* the right atrium, left atrium, common atrium, or *via* an azygos continuation of the inferior vena cava.

The triad of cardiac malformations, abdominal heterotaxia, and splenic anomalies is not invariable. We have studied 7 additional patients with abdominal heterotaxia and cardiac malformations similar to

asplenia, but in whom the spleen was normal. Freedom[5] has also reported such patients. Conversely, several reports have described splenic anomalies with abdominal heterotaxia but without congenital heart disease.[6,11]

CONCLUSIONS AND SUMMARY

Asplenia and polysplenia are frequently associated with complex cardiac malformations and other visceral abnormalities. These latter may include malrotation of the gut, horizontal liver, interrupted inferior vena cava with azygos continuation, and symmetry of the lungs.

The findings on thoracic and abdominal roentgenograms may frequently help one predict the type of splenic abnormality, but this is not invariable. The knowledge of the spleen is helpful in suggesting the most likely cardiac malformations.

Forty-one patients with proven asplenia or polysplenia coexisting with congenital heart disease were analyzed in detail. In these cases the abdominal or thoracic roentgenograms as well as the angiographic findings were reviewed. The findings were correlated with cardiac catheterizations, surgical operations, and postmortem examinations. Certain characteristics were found to be prevalent in each of these.

The presumptive diagnosis from the plain film roentgenograms can be made in classical situations, but it is not as clear-cut as one would hope.

The fact that this constellation of findings can be seen in cases with a normal spleen is also discussed.

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ANGIOGRAPHIC FINDINGS IN THE ADRENALS DURING STRESS

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THE vascular supply to the adrenals originates from 3 main sources: the renal artery; directly from the aorta; and from the right and left inferior phrenic arteries. According to Michels[4] the inferior phrenic arteries, in most cases, have an independent origin from the celiac artery. The left inferior phrenic artery arises more frequently (42 per cent) from the celiac artery than the right (32 per cent). In 18 per cent of cases the inferior phrenic arteries arise as a common trunk from the celiac artery.

When celiac angiography is performed, there is sometimes a rather dense accumulation of the contrast medium in the adrenals, and at times no staining at all is seen in spite of good contrast medium filling of the inferior phrenic arteries from the celiac artery. The reason for this is not known.

In emergency celiac angiographies performed because of gastrointestinal bleeding or trauma, we have observed a more frequent staining of the adrenals than in normally scheduled patients and in 3 patients extravasation of the contrast medium in the adrenals was seen, suggesting hemorrhage.

MATERIAL AND METHOD

Fourteen patients, ages 13–79 years, were examined with angiography of the celiac artery, 7 of them because of hematemesis and melena, 6 because of trauma to the abdomen and 1 for evaluation of a postoperative subphrenic hemorrhage. The angiography was performed with a red Ödman-Ledin catheter (Kifa, Sweden, ID/OD 1.4/2.2 mm.) with 2 side-holes 1 cm. from the tip. Forty ml. of contrast medium, meglumine metrizoate (Isopaque coronar, Nyco, Norway) was injected at a rate of 10–12 ml./second and exposures were made one/second for 2 seconds, two/second for 4 seconds and one every other second for 10 seconds.

RESULTS

In 8 cases the angiography was performed to localize the source of intestinal bleeding. Seven of these patients had some period of shock during the hours preceding the angiographic procedure (Table 1), but only 2 had a systolic blood pressure below 100 mm. Hg during the angiography. Among the patients with traumatic injury to the liver or spleen only 1 had been in shock prior to angiography and that patient was the only one who had a systolic blood pressure below 100 at the time of the procedure.

Accumulation of contrast medium in the adrenals was noted in 7 cases on the right side and in 8 on the left. In 2 cases the degree of accumulation of the contrast medium in the adrenal parenchyma could not be evaluated because of extravasation. In 7 patients a rather intense staining of at least one of the adrenals was noted and 5 of these patients had experienced some period of shock before the angiography (Fig. 1; and 2).

Adrenal hemorrhage was seen in 3 patients and documented through extravasation of the contrast medium (Fig. 3; and 4). One of these patients was operated upon because of a cholecystoduodenal fistula. Postoperatively, he was severely ill with subphrenic hemorrhage and shock. The patient died 1 day after the angiography. The 2 other patients had severe hemor-

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TABLE I

SUMMARY OF CASES

Case No.	Reason for Angiographic Examination	Presence of Shock before Examination	Systolic Blood Pressure at Examination (mm. Hg)	Degree of Contrast Medium Accumulation in the Adrenals		Extravasation of Contrast Medium in the Adrenals	
				Right	Left	Right	Left
1.	Intestinal bleeding	Yes	105	Intense	None	None	None
2.	Intestinal bleeding	No	150	None	Intense	None	None
3.	Splenic rupture	No	125	None	Slight	None	None
4.	Splenic rupture	No	120	Slight	Intense	None	None
5.	Intestinal bleeding	Yes	90–60	Intense	Intense		
6.	Liver rupture	No	120	None	Slight	None	None
7.	Liver rupture	No	145	Slight	None	None	None
8.	Intestinal bleeding	Yes	100	Slight	Slight	None	None
9.	Liver rupture	No	120	Slight	None	None	None
10.	Splenic rupture	Yes	90	None	Intense	None	None
11.	Intestinal bleeding	Yes	90	Intense	None	None	None
12.	Retroperitoneal bleeding	Yes	120	None	Not evaluable	None	Severe
13.	Intestinal bleeding	Yes	110	None	Not evaluable	None	Severe
14.	Intestinal bleeding	Yes	100	None	Intense	None	Slight

rhages from a benign gastric ulcer and were in shock before the angiography. One of them died shortly after an operation for excision of the ulcer.

DISCUSSION

Experimental investigations by Sjöstrand[8] have shown that during different conditions of stress, the blood flow through the adrenal cortex may significantly increase. Here the stimulation of the adrenals is presumably mediated by the hypophysis, and this concept is supported by the works of O'Donnell *et al.*,[6] who reported adrenal cortical hyperplasia in patients that were treated with ACTH before death, and by Tonutti,[10] who demonstrated that the morphologic changes produced by difteria toxin in the adrenal glands of guinea pigs could be prevented by previous hypophysectomy.

Moreover, cases are reported where the excessive ACTH stimulation of the adrenal gland during conditions of stress may have been an important factor in the production of adrenal hemorrhagic necrosis.[5,11] Gann and Egdahl[1] showed in dogs that the secretion of corticosteroids was stimulated by

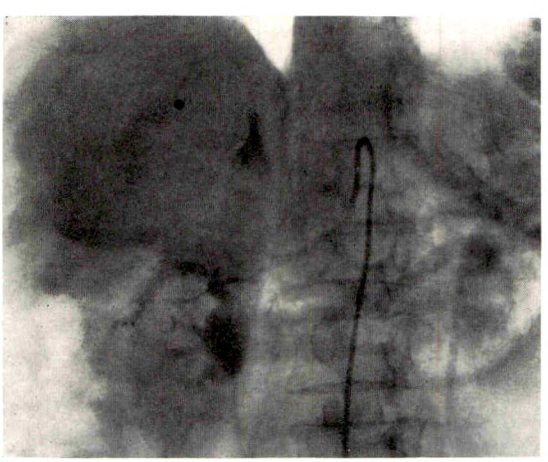


FIG. 1. Case 1. Intestinal bleeding. Celiac angiography, venous phase. Dense accumulation of the contrast medium in the right adrenal. Bleeding duodenal ulcer with extravasation of the contrast medium in the duodenum.



FIG. 2. Case 5. Intestinal bleeding. Celiac angiography, venous phase. Dense accumulation of the contrast medium in both adrenals, the left projected over the spine.

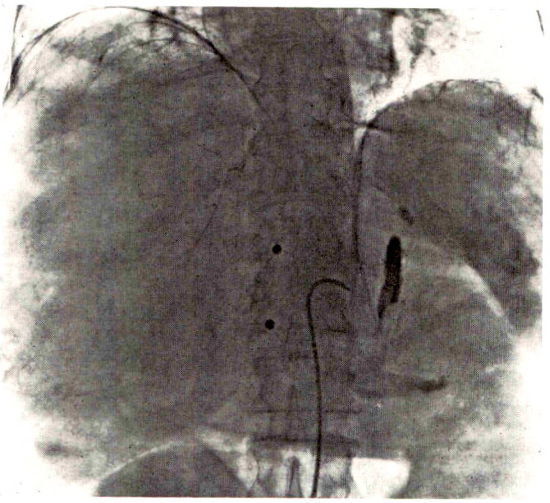


FIG. 3. Case 12. Postoperative subphrenic hemorrhage. Celiac angiography, venous phase. Extravasation of the contrast medium in the left adrenal.

hypotension and hypovolemia secondary to hemorrhage. Even in the absence of hypotension, hypovolemia stimulated the secretion of corticosteroids to about the same values as when the adrenals were stimulated by ACTH.

In the majority of cases reported by Greendyke,[2] severe or prolonged stress seemed to be a common factor in the production of adrenal hemorrhage. Shock was present in 52 per cent of his 33 patients in the last days of their lives.

Thrash and Iri[9] reported 6 autopsy cases with extensive adrenal infarction. All patients underwent at least one episode of shock during the last days of their illness. In 1 case the adrenals showed multiple confluent areas of infarction, which were surrounded by zones of hyperemia. In 3 of the 6 cases the adrenal infarction was only diagnosed at microscopic examination and the authors suggested that adrenal infarction is more common than previously recognized.

Russell[7] made a study of the adrenals of 162 patients in whom one or more severe episodes of shock prior to death were documented. A control series of 200 patients was selected in whom hypotensive episodes prior to death could be excluded. In the first group, 25 cases showed significant changes in the adrenals, consisting of hemorrhages (17 cases), and tissue necrosis which ranged from focal cytolysis and cell degeneration to complete cortical infarction (16 cases). In the control series none of these adrenal changes were observed.

From the cited references the angiographic findings in our patients can be well explained. In all cases some conditions of stress preceded or were present during the angiographic procedure. This may have caused an increased release of corticotropin with hyperemia and hyperplasia of the adrenal cortex which was reflected by the accumulation of the contrast medium in that area. During conditions of prolonged stress, factors such as excessive ACTH stimulation of the adrenal glands in shock are important steps in the production of adrenal necrosis and hemorrhage.[2] In 3 of our patients, where adrenal hemorrhage was diagnosed, the angiographic examination was preceded by just such a prolonged stress. During acute severe hypotension, reduced adrenal blood flow may prevent the adrenal cortical reaction to the corticotropin released by the hypophysis,[3] and adrenal hyperemia or hemorrhage will not appear.

Central vein thrombosis is a common finding at microscopic examination of hemorrhagic adrenals. The etiologic sig-

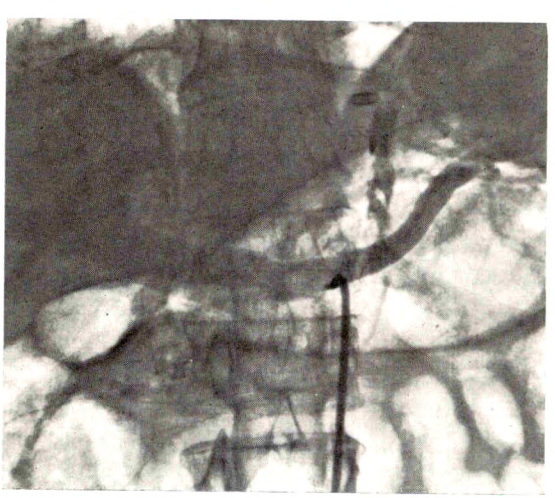


FIG. 4. Case 13. Intestinal bleeding. Celiac angiography, venous phase. Extravasation in the left adrenal. Slight staining of the right adrenal.

nificance of this thrombosis in the production of adrenal hemorrhage is uncertain. Adrenal cortical necrosis may appear without central vein thrombosis[2] and cortical necrosis cannot be excluded as the cause of the thrombosis.

At angiography of the abdominal aorta or the celiac artery, accumulation of the contrast medium is sometimes seen in the adrenals of patients without a history of trauma or bleeding. The possibility of the angiographic procedure itself causing increased stress in these patients must be taken into account.

A more frequent use of cortisone to avoid adrenal hemorrhage in severely ill patients is suggested and perhaps the injection of ACTH prior to adrenal angiography can increase the probability of diagnosing small adrenal cortical adenomas.

SUMMARY

Fourteen patients are reported in whom angiography of the celiac artery was performed during a condition of stress secondary to hemorrhage or trauma. In 12 of these patients there was a marked accumulation of the contrast medium in the adrenals and in 3 of them adrenal hemorrhage was present.

The probable mechanism in this reaction is discussed.

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ADRENAL VENOGRAPHY: CLINICAL-ROENTGENOGRAPHIC CORRELATION IN 80 PATIENTS*

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SINCE Masoni's description in 1957 of the feasibility of adrenal vein catheterization,[8] many reports have appeared confirming the usefulness of this technique for the study of suspected adrenal pathology.[1,3,7,10,13] We have had the opportunity of studying 80 patients in this fashion during the past 5 years. Roentgenographic analysis and appropriate hormone assays of adrenal venous blood were performed.

It is the purpose of this paper to describe our experience with this group of patients.

METHOD

The right adrenal veins of the first 10 patients were catheterized, using preformed green KIFA catheters according to the method described by Reuter *et al.*[13] Right adrenal catheterization was accomplished in subsequent patients using a Cordis Ducor femoral visceral III catheter. The right adrenal vein enters the vena cava on its posterior aspect at the T11–12 level. It has been our experience that initial difficulty in finding the right adrenal vein orifice was related to failure to explore the posterior aspect of the vena cava with the catheter. Difficulty occurs when the catheter tip is oriented laterally resulting in entrance into one of the accessory hepatic veins (Fig. 1). The left side was catheterized using preformed green KIFA catheters without mechanical guide wire or catheter manipulating devices. This method has been described elsewhere and has been quite reliable.[11] The position of the catheters in the adrenal veins was confirmed with 0.5 to 1 cc. test injections of meglumine diatrizoate (renografin 60). Adrenal vein samples were withdrawn prior to filming, ensuring useful information by hormone assay in the event of subsequent extravasation. This routine has proved particularly valuable in patients with aldosteronism. Blood samples could be obtained by gentle traction with a syringe on the left. On the right, samples were most often obtained by allowing the blood to drip freely from the catheter into a test tube. A sample was also obtained from the vena cava below the level of the renal veins. These samples were analyzed for two or more of the following corticoids:

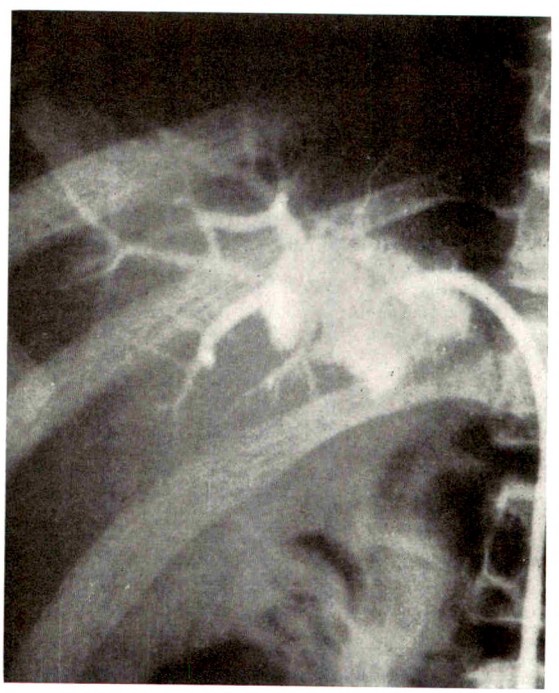


FIG. 1. Accessory hepatic vein. Incorrect catheter placement. Test injection opacifies several hepatic veins and causes a parenchymal blush. The catheter entered an accessory hepatic vein which is not rare since these branches enter the inferior vena cava adjacent to the adrenal vein. Some transient pain accompanies adrenal vein injections, while the patient is rarely aware of hepatic venous injections.

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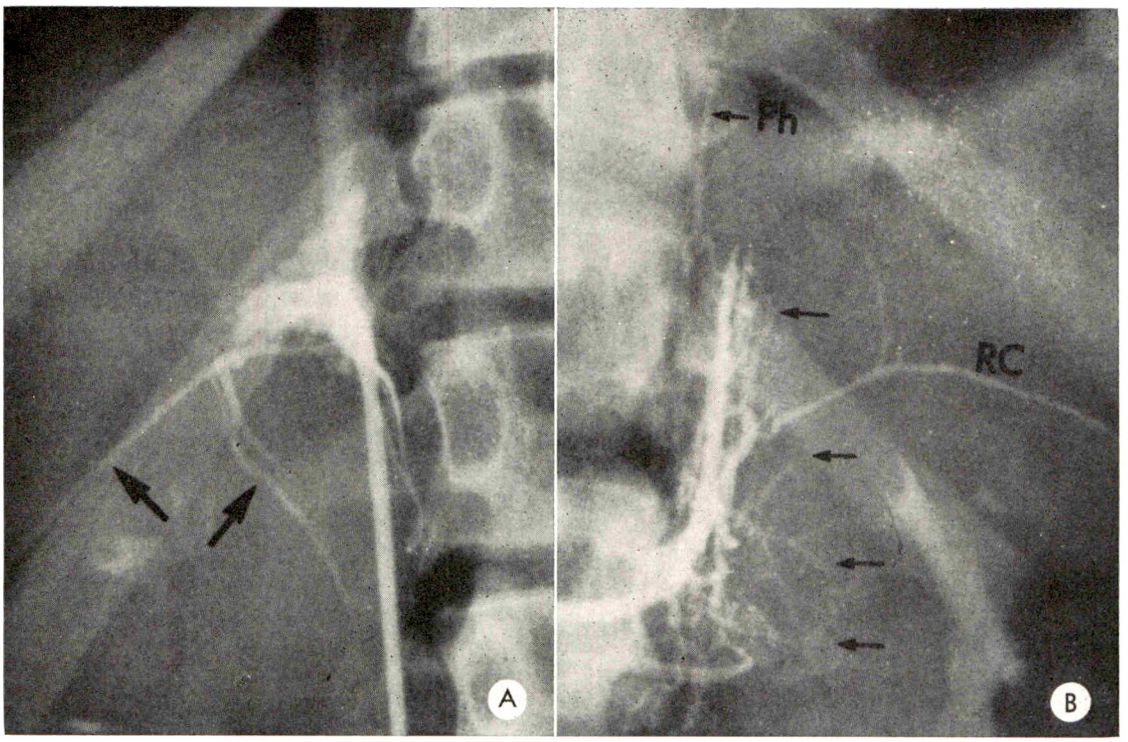


Fig. 2. Normal adrenal venograms. (*A*) Right. There is a parenchymal blush as well as filling of venous tributaries. Opacification of several communicating renal capsular branches (arrows) is seen. Normal location, size and shape of the gland are demonstrated. (*B*) Left in another patient. The left adrenal is normally larger than the right. The extent and shape of the gland are demonstrated by the branching tributaries (arrows). The left adrenal is also more anterior and medial in its relation to the kidney and extends towards the renal hilum. There is opacification of communicating renal capsular (RC) and phrenic (Ph) veins.

cortisol, aldosterone, androstenedione, testosterone, dehydro-epiandrosterone sulphate and androsterone sulphate. Of these hormones, assay of cortisol and aldosterone has proved to be of the greatest clinical value. Aldosterone assay has been of critical importance in the evaluation of patients with primary hyperaldosteronism. Cortisol is a useful "marker" confirming the fact that one has obtained adrenal venous blood.

Catheterization of the right adrenal was attempted in 77 patients and was successful in 72. Catheterization of the left adrenal was attempted in 78 patients and was successful in 72. Seven of the failures occurred in the first 14 patients studied.

Patients discussed in this paper fall in the following groups: (1) Cushing's syndrome—22 patients; (2) virilization—15 patients; (3) primary aldosteronism—10 patients; (4) pheochromocytoma—5 patients; (5) gynecomastia—17 patients; (6) miscellaneous—11 patients.

ROENTGENOGRAPHIC FINDINGS

The right adrenal vein is quite short and drains directly into the vena cava at the level of T11–12 in the majority of cases. Rarely, this vein may join an accessory hepatic vein before entering the vena cava. This central vein drains the gland after it receives fine branching tributaries (Fig. 2*A*). Collateral branches are often opacified which connect with the inferior phrenic, renal capsular and other retroperitoneal veins. In most cases, the right gland has the general shape of an equilateral triangle. Postmortem injection studies have shown some convexity to the medial border of this gland in as high as 81 per cent of cases.[9]

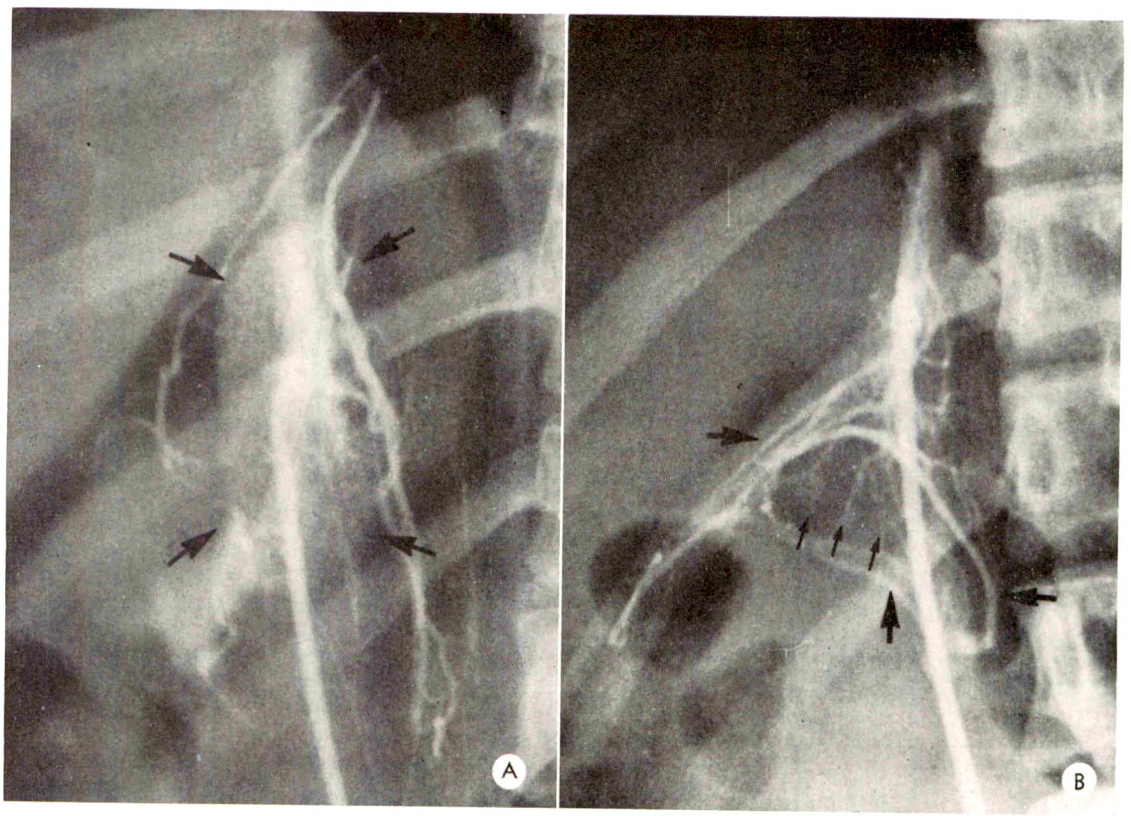


FIG. 3. Hyperplasia. (*A*) A parenchymal blush (arrows) in this hyperplastic gland demonstrates generalized enlargement and increased convexity of the glandular contours. Several communicating retroperitoneal veins are also opacified. (*B*) The increased size of the gland is outlined by parenchymal (small arrows) and periadrenal veins (large arrows). There is also increased separation of intraglandular tributaries (small arrows) confirming hyperplasia. (*A* reproduced with permission of *Ann. Int. Med.*)

Thirty-four per cent of these patients had more than 1 convex right adrenal border.

On the left, the central vein is larger and drains into the superior aspect of the renal vein near the left pedicle of the superimposed vertebral body (Fig. 2*B*). The large central vein usually receives the inferior phrenic vein before it enters the renal vein. Other collateral connections to renal, renal capsular, ascending lumbar, splenic and other retroperitoneal veins may be seen. The normally larger left gland has a leaf-like shape with a tendency to generalized convexity. The triangular shape seen on the right is less common on the left.

The shape of the gland is of importance in the diagnosis of hyperplasia. It has been shown that hyperfunctioning glands are not necessarily enlarged either roentgenographically or at surgery.[14] Nevertheless, it has been our experience that hyperplastic glands often have an increased convexity (Fig. 3*A*). This change in appearance is obviously more easily assessed on the right, where a triangular appearance is more consistently seen in normals.

The normal ranges for size have been estimated by measuring areas of glands from roentgenograms.[9] On the right, the mean area is 7.5 cm.^{2}, while on the left, it is 9.0 cm.^{2}. The apparent size of a gland from a venogram obtained *in vivo* will be a reflection of the degree of reflux. Obviously, inadequate filling of the venous system will make the gland appear smaller.

In addition to size and shape, the appearance of the intraglandular branching pattern may help in the diagnosis of hyper-

plasia. Increased separation of these branches occurs as the parenchyma hypertrophies (Fig. 3*B*). Using the criteria of size, shape and intraglandular branching pattern, a diagnosis of hyperplasia was made in 8 of 17 of our patients with Cushing's syndrome due to adrenal hyperfunction.

The venographic appearance of adrenal tumors is a reflection both of their vascularity and the degree of reflux into the lesion. For example, one aldosteronoma studied appeared quite avascular on an initial injection. A second injection with the catheter tip in better position demonstrated a vascular adenoma (Fig. 4, *A* and *B*).

It was not possible to differentiate benign and malignant tumors in the patients with Cushing's syndrome on the basis of their venographic appearance. As with the patient with the aldosteronoma shown in Figure 4, *A* and *B*, the apparent vascularity of the malignant and benign tumors associated with Cushing's syndrome was also related to the degree of reflux obtained during venography. One patient with a carcinoma had several satellite nodules found at surgery that were not demonstrated by venography. This was a reflection of the fact that the nodules did not drain into the main adrenal vein and hence were not opacified by this method. In 1 patient with a carcinoma, a well developed plexus of veins was seen at the inferior margin of the mass providing additional drainage of the tumor (Fig. 5). This was not seen in any other tumor of this series and its significance is not clear.

The size of the lesion is of interest since aldosteronomas are usually small when detected. We found a range of 0.7–2.5 cm. in

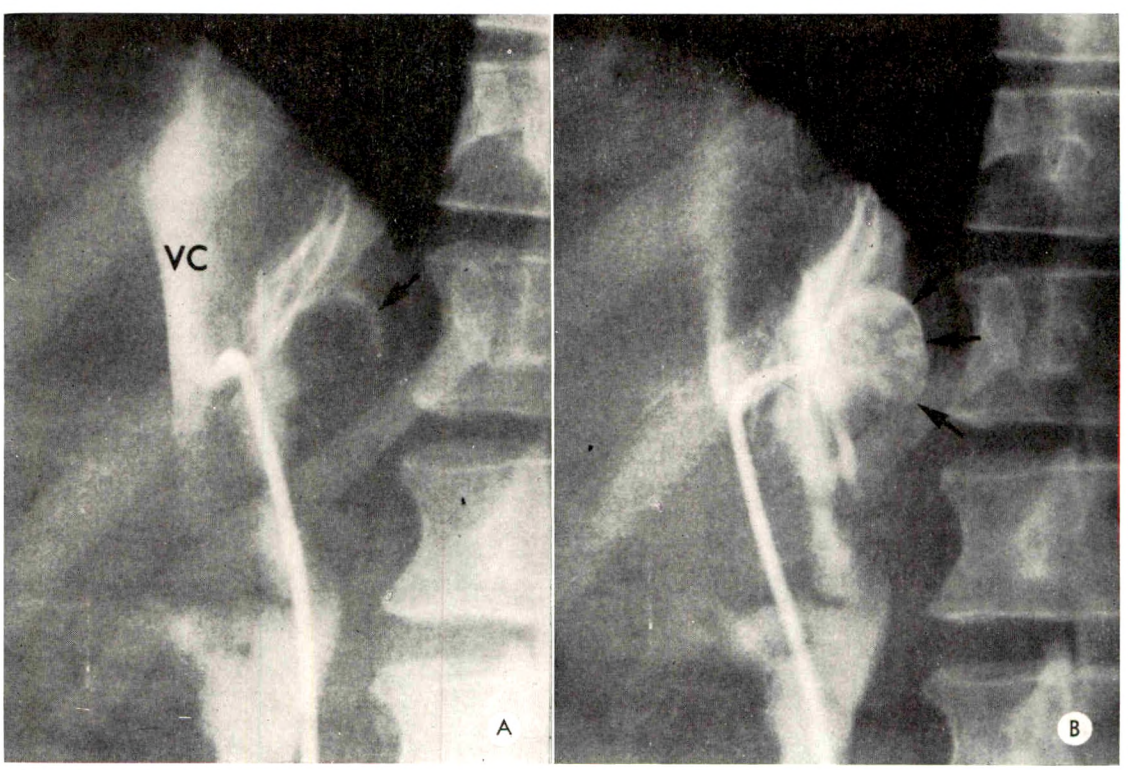


Fig. 4. Aldosteronoma, right. Relation of degree of reflux to apparent vascularity of a lesion. (*A*) Oblique view with catheter tip positioned laterally. There is poor filling of the adrenal venous system with considerable flow into the vena cava (VC). A circumferential vein outlines the tumor giving the appearance of a relatively avascular mass (arrow). (*B*) Same oblique view with catheter tip oriented along the long axis of the adrenal vein. The true vascular nature of the tumor is now well demonstrated by visualization of tumor vessels (arrows).

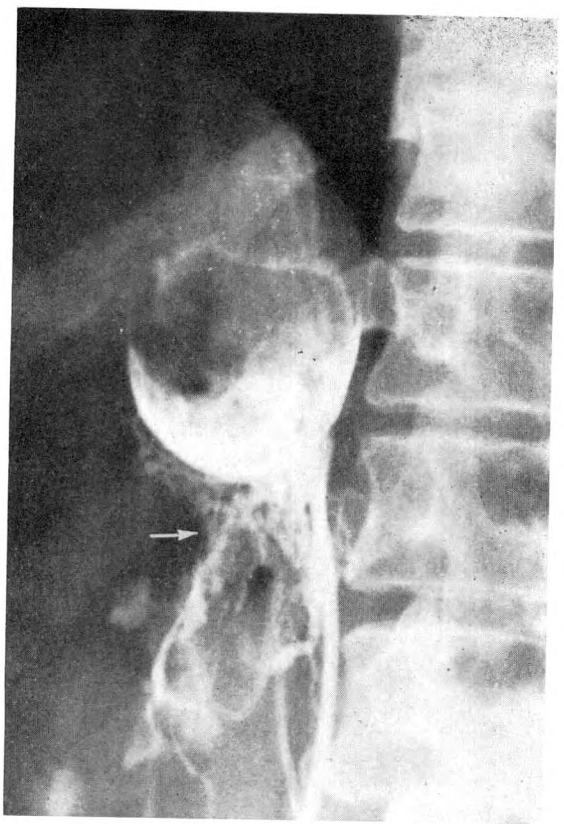


FIG. 5. Cushing's syndrome due to adenocarcinoma. A single round mass is demonstrated. An unusually well developed plexus of veins drains the inferior margin of the tumor (arrow).

10 cases, while 5 tumors causing Cushing's syndrome ranged from 2.5 to 5.2 cm.

The adrenal cortical tumors tend to displace adjacent veins and often appear well circumscribed as a result (Fig. 6). The pheochromocytomas studied by venography appeared less well circumscribed even though these lesions were histologically benign and noninvasive. It was possible to identify a residual normal triangular cap of cortical tissue at the superior aspect of these medullary tumors (Fig. 7). Roentgenographic differentiation of these neoplasms is usually only of academic interest since the clinical syndromes associated with cortical and medullary tumors are quite different.

1. CUSHING'S SYNDROME

The clinical diagnosis of Cushing's syndrome is usually further supported by urine analysis of 17-hydroxicorticoids, cortisol secretion rates and 11-deoxycortisol secretion rates. In addition, the dexamethasone suppression test was used to differentiate patients with hyperfunction from those with tumors. The endocrine work-up gives a good indication of whether hyperfunction or neoplasm is present. The venographic procedure confirms the diagnosis and lateralizes the lesion. Five of 22 patients with Cushing's syndrome had tumors demonstrated by adrenal venography (Table 1). Three of these neoplasms were malignant. In each case a single round mass was demonstrated. Although the vascular pattern did not make differentiation of benign and malignant lesions possible, malignancy is often suspected on the basis of the rapid onset of clinical findings and the laboratory work-up.

The remaining 17 patients with Cush-

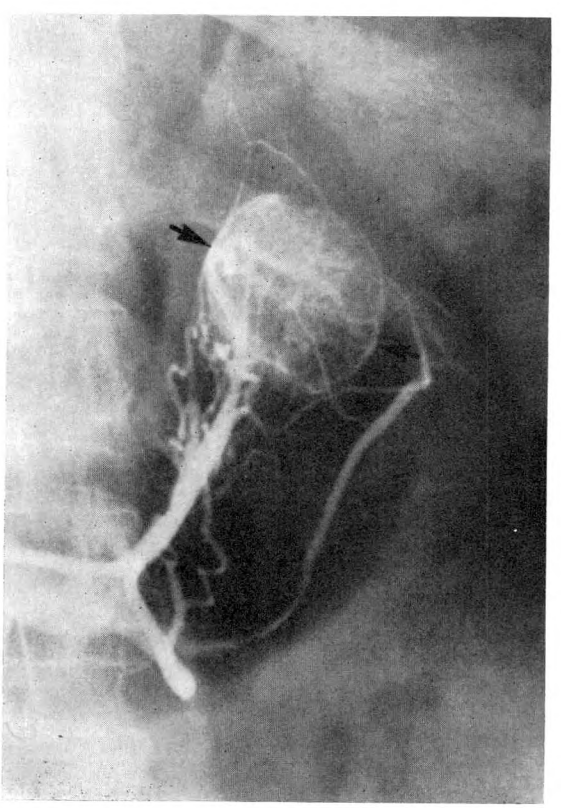


FIG. 6. Cushing's syndrome due to adenoma. The mass is well circumscribed by peripheral veins (arrows). The vascular nature of the lesion is well demonstrated by numerous small tumor vessels.

TABLE I

CUSHING'S SYNDROME

Case No.	Sex	Age	Venographic Findings		Treatment, Comments
			Right	Left	
1. B.M.	F	60	Tumor	O	Right adrenal adenoma (4.5 cm.)
2. I.G.	F	50	O	Tumor	Left adrenal adenoma (2.5 cm.)
3. H.A.	F	44	Single tumor	NL	Round encapsulated mass with three adjacent nodules (0.5–3.5 cm.); adrenal carcinoma; recurrence 1½ years later
4. B.B.	F	38	Tumor	NL	Encapsulated right adrenal carcinoma (5.2 cm.); recurred 2 years later
5. R.F.	F	57	Tumor	NL	Encapsulated carcinoma
6. D.G.	M	60	O	Hyperplastic	Bilateral adrenalectomy; right adrenal 12 gm.; left adrenal 14 gm.; pituitary irradiation
7. S.L.	F	50	NL	NL	Pituitary irradiation
8. D.H.	M	36	NL	NL	Pituitary irradiation
9. R.T.	M	30	Hyperplastic	O	Bilateral adrenalectomy; right adrenal, 15 gm.; left adrenal, 13.5 gm.; pituitary irradiation
10. M.B.	F	46	Hyperplastic	Hyperplastic	Pituitary irradiation
11. M.F.	F	30	NL	NA	Previous left adrenalectomy; pituitary irradiation
12. G.H.	F	60	Hyperplastic	O	Adrenal hyperplasia, weights not available
13. J.F.	M	14	NL	NL	Obesity, hypertension, carbohydrate intolerance
14. S.S.	F	37	NL	NL	Enlarged sella turcica; suprasellar extension of neuroectodermal tumor
15. L.E.	M	29	NL	NL	Pituitary irradiation
16. I.D.	F	29	NL	NL	Pituitary irradiation
17. D.Z.	F	32	NL	NL	Pituitary irradiation
18. N.S.	F	20	Hyperplastic	Hyperplastic	Pituitary irradiation
19. S.B.	F	40	NL	NL	Pituitary irradiation
20. H.P.	M	24	NL	NL	Pituitary irradiation
21. B.H.	F	58	Hyperplastic	Hyperplastic	Pituitary irradiation
22. C.R.	F	36	Hyperplastic	Hyperplastic	Pituitary irradiation

O = Venography unsuccessful; NA = Venography not attempted; NL = Venography normal.

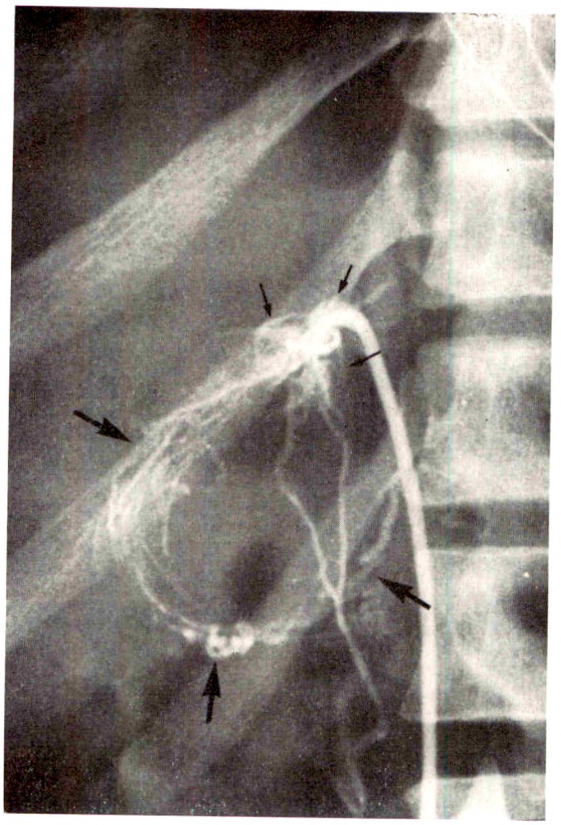


FIG. 7. Pheochromocytoma. The tumor is poorly demarcated but demonstrated by peripheral draining and tumor veins (large arrows). A group of normal veins indicate the outline of a cap of normal intact cortex at the superior aspect of the tumor (small arrows).

ing's syndrome had bilateral adrenal hyperfunction. This hyperfunction was associated with Cushing's disease in 15 patients. One patient had an associated suprasellar tumor and one had a pancreatic carcinoid responsible for the adrenal hyperfunction. As noted earlier, an adrenal gland may exhibit increased function without being enlarged roentgenographically or at surgery. Soffer *et al.*[14] reported enlarged adrenals by weight at surgery in only 11 of 24 patients with adrenal hyperfunction and Cushing's syndrome. Thus, it is not surprising that the glands were considered to be hyperplastic roentgenologically in only 8 patients and of normal size in 9 patients in this current series. One of the important benefits of demonstrating the hyperfunctioning gland adequately is that a tumor is excluded, making nonsurgical treatment a more confident course for the clinician.

2. VIRILIZATION

Hirsutism and menstrual abnormalities are not uncommon symptoms. However, underlying adrenal pathology as a cause is rare. For this reason, we have restricted venographic evaluation of females with virilization to those who have developed these findings after puberty and in patients with elevated urinary 17-ketosteroids not suppressed by dexamethasone. The 15 patients with virilization selected for study presented with oligomenorrhea and amenorrhea as well as hirsutism. Some patients also demonstrated acne, hypermuscularity and clitoral hypertrophy. Three patients in this group had adenomas. These tumors were indistinguishable roentgenographically from those causing Cushing's syndrome (Fig. 8). Resection of the tumor in 1 patient, a 17 year old girl, was followed by the menarche 1 month after resection. Three

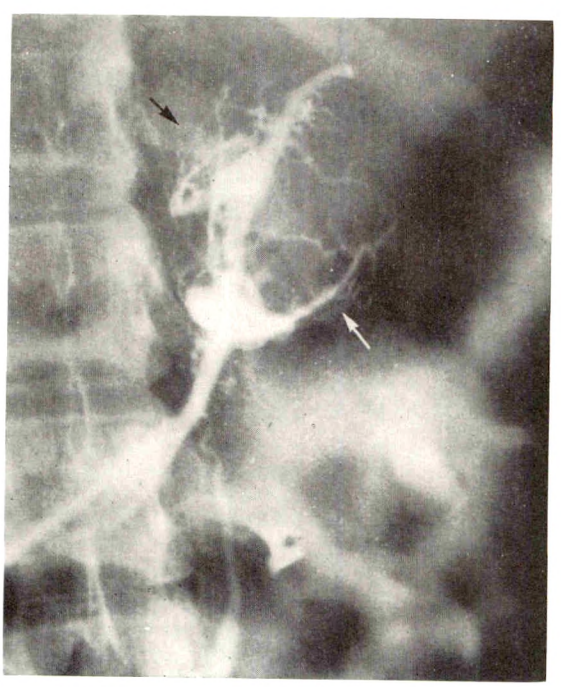


FIG. 8. Virilizing adenoma. A single round mass is demonstrated (arrows). This lesion is roentgenographically indistinguishable from the adenomas associated with Cushing's syndrome.

patients had congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Their glands appeared generally enlarged at venography. One of the patients in this group with idiopathic hirsutism and amenorrhea also had adrenals that appeared enlarged by venography. The remaining 9 patients had normal appearing adrenals.

Blair and Reuter[1] studied a group of 50 women with virilization. They reported roentgenographic changes with hypertrophy in 11. There is ongoing research to determine the source of the increased androgen production in females with virilization by combined collection of blood from adrenal and ovarian veins.

3. PRIMARY HYPERALDOSTERONISM

The 10 patients in this group presented with the classical findings of hypertension and hypokalemia. Excessive aldosterone production and suppressed plasma renin activity were also demonstrated. All patients were studied roentgenographically and the last 8 had adrenal vein aldosterone concentrations measured by the method previously reported.[12] Each of the 10 patients in this group had a solitary aldosteronoma at surgery (Table II). Following resection, there was a rapid return of electrolyte abnormalities to normal and a gradual decrease in blood pressure. Venographic demonstration of the tumor was unequivocal in 6 of the 10 patients (Fig. 9). In 1 patient the left gland was thought to be abnormal roentgenographically, but the contour of the tumor was not demonstrated with confidence. In 1 patient the right side was obscured by extravasation and the left was thought to be abnormal. However, adrenal blood assay correctly localized the tumor to the right side as confirmed by

TABLE II

PRIMARY ALDOSTERONISM: VENOGRAPHY AND ALDOSTERONE ASSAY

Case No.	Age	Sex	Adrenal Venography		Adrenal Aldosterone Assay	Surgical Findings
			Right	Left		
1. A.F.	49	F	NL	Tumor	Left tumor	Left adenoma (1.0 cm.)
2. B.L.	40	F	NL	Tumor	Left tumor	Left adenoma (1.2 cm.)
3. M.P.	40	F	NL	Tumor	Left tumor	Left adenoma (1.3 cm.)
4. J.G.	50	M	Tumor and minimal extravasation	o	X	Right adenoma (0.7 cm.)
5. E.R.	36	F	Extravasation	Possible tumor	Right tumor	Right adenoma (2.3 cm.)
6. E.S.	61	M	NL	NL	Right tumor	Right adenoma (1.1 cm.)
7. S.G.	70	M	NL	Tumor	Left tumor	Left adenoma (0.7 cm.)
8. R.S.	53	M	NL	Tumor	Left tumor	Left adenoma (2 cm.)
9. M.C.	49	F	Tumor	NL	Right tumor	Right adenoma (1.8 cm.)
10. M.L.	55	M	o	Removal at time of nephrectomy	X	Right adenoma

NL = Normal; o = Not successful; X = Assay not performed.

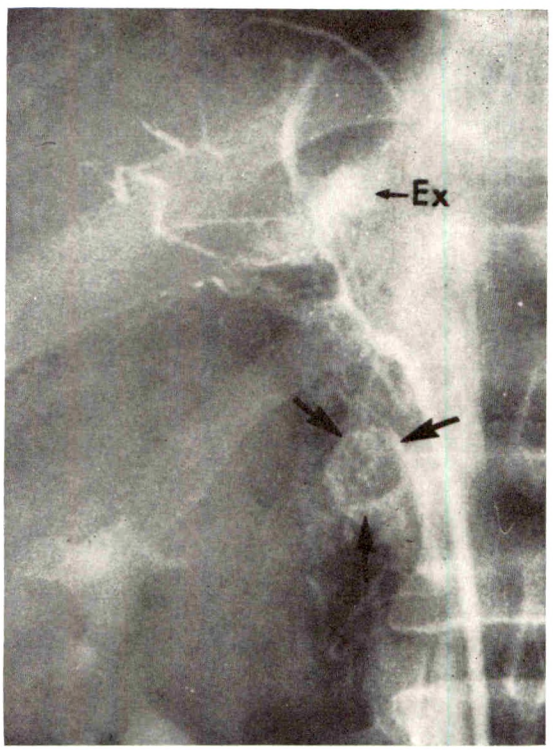


FIG. 9. Aldosteronoma. A single 7 mm. tumor is demonstrated (arrows). There is extravasation at the superior aspect of the gland (Ex).

bilateral adrenal exploration. In 1 patient catheterization of the right adrenal vein was inadequate, but the presence of the tumor was known, since the opposite adrenal had been previously resected at the time of nephrectomy for a renal neoplasm. One patient had apparently normal glands at venography, but a tumor was nonetheless localized to the right side by aldosterone assay of the adrenal venous blood and confirmed at surgery.

In all our patients undergoing successful catheterization, the tumor was correctly localized by aldosterone assay of the adrenal venous blood. The concentration of aldosterone was 20 to 107 times greater on the side of the tumor as compared to the normal side. Thus, analysis of the blood sample is a more reliable means of localizing the lesion than demonstration by venography. This has been the experience of other workers who report between 57 and 80 per cent success in demonstrating the side of the lesion by venography. Horton and Finck[6] were able to assign the correct side by adrenal venous blood assay in all of 14 patients with aldosteronomas, while Kahn *et al.*[7] were successful in 10 of 12 patients. We recommend routine collection of adrenal blood samples just prior to filming. In this way, the important blood sample is available for analysis in the event of extravasation or a technically poor study.

4. PHEOCHROMOCYTOMA

Five patients with this tumor have been studied by adrenal venography. In 3 cases the lesion was demonstrated on the right side. In 2 patients the adrenals were normal roentgenographically and proved not to be the site of the lesion. Since these tumors may arise in chromaffin tissue in extraadrenal locations, angiographic evaluation of these patients cannot be considered complete unless aortography and appropriate selective arteriography are performed. This is particularly so since these lesions may be multiple. This point is best illustrated by 1 of our patients who had seven extraadrenal pheochromocytomas removed. One was in the posterior mediastinum, 1 was in the cervical prevertebral region and 5 were paraaortic in location.

As with arterial evaluation of these patients, appropriate monitoring of pulse rate, blood pressure and medication with phentolamine (regitine) is mandatory. No complications occurred during arteriography or venography in the patients we have studied using these precautions.

5. GYNECOMASTIA

Seventeen men with this finding were studied by venography. No tumors were discovered. This is not surprising since feminizing adrenal tumors are quite rare.[5] One 17 year old boy was thought to have adrenals that were roentgenographically enlarged. All of the remaining patients in this group had normal appearing glands. Five of the patients had Klinefelter's syndrome. Adrenal venous blood samples were

collected and assays of appropriate hormones performed.

6. MISCELLANEOUS

In this group of 11 patients, venography demonstrated a left adrenal tumor in a 60 year old man with hypertension and normal serum electrolytes. This patient was thought to have a nonfunctioning adrenal tumor, but refused further evaluation or surgical exploration. A 36 year old woman with obesity, cyclic edema and hypertension was thought to have roentgenographically enlarged adrenals. The remaining patients in this group included 4 patients with essential hypertension and hypokalemia that proved to be due to diuretic administration. A male with congenital adrenal hyperplasia, 1 patient with Turner's syndrome (XO), and 2 patients with obesity of unknown origin were also studied. One patient with metastatic carcinoma of the breast was studied prior to possible transcatheter adrenal ablation (Table III).

DISCUSSION

Cortical tumors responsible for Cushing's syndrome and virilization as well as pheochromocytomas are often large enough to be demonstrated by conventional roentgenographic techniques. Arteriography and venography are useful in confirming the presence of a lesion, demonstrating its blood supply, and establishing the presence of a contralateral normal gland. We prefer venography in these patients because morbidity has been low and, in addition, blood samples can be obtained for study from normal and abnormal glands as well as from the vena cava and gonadal veins. For these reasons, venography is useful as both a clinical and investigative procedure.

TABLE III

MISCELLANEOUS

Case No.	Sex	Age	Clinical Diagnosis	Venographic Findings	
				Right	Left
1. D.S.	M	60	Hypertension and normal serum electrolytes	NL	Tumor
2. R.Z.	M	40	Essential hypertension	NL	NL
3. A.D.	M	39	Essential hypertension	NL	NL
4. M.J.	M	31	Essential hypertension	NL	NL
5. G.S.	F	43	Essential hypertension	NL	NL
6. J.C.	M	30	Congenital adrenal hyperplasia	Hyperplastic	Hyperplastic
7. P.T.	F	46	Obesity, hypertension, cyclic edema	Hyperplastic	Hyperplastic
8. S.H.	M	45	Obesity	o	NL
9. J.S.	F	22	Obesity, hypertension	NL	NL
10. J.H.	F	17	Turner's syndrome (XO)	NL	NL
11. E.K.	F	66	Metastatic carcinoma of breast; transcatheter ablation not performed	NL	NL

NL = Normal; o = Not successful.

The importance of demonstrating the small adenomas usually encountered with primary hyperaldosteronism is well documented.[6,7,10] Since these lesions are small, they may be difficult to locate at surgery and, in addition, one would want to avoid bilateral exploration if possible. An increased incidence of extravasation associated with aldosteronism has been reported and it has been suggested that there is a possible increased fragility of the veins associated with this condition.[2,7] We had 2 cases of extravasation in 10 patients with aldosteronism studied by this technique (Fig. 9).

It may be difficult to demonstrate these tumors which may be quite small (0.7–2.3 cm. in our series) and thus may be missed unless good reflux throughout the parenchyma is obtained. The challenge to document a lesion is basic to the radiologist, but nonetheless may not be worth the added risk of extravasation, as hormone assay of adrenal blood is more reliable and less dangerous. The adrenal veins in primary hyperaldosteronism may or may not be more fragile, but aggressive angiographic evaluation by the radiologist is not warranted. A good argument can be made for minimal filming and maximal blood sampling in patients with primary aldosteronism. Kahn *et al.*[7] report 95 ger cent accuracy in a combination of adrenal venography, arteriography and assay of adrenal venous blood. Thus far, we have had 100 per cent success in localizing the lesion by adrenal blood assay, when adequate blood samples were obtained. Our plan is to withhold arteriography for cases with negative venograms and equivocal aldosterone assay.

We have had no evidence of hypoadrenalism or regression of clinical or biochemical abnormalities in the 5 patients who had extravasation in this series.[4] Two of these patients had aldosteronomas on the side of the extravasation. The other 3 patients proved to have no demonstrable adrenal pathology. In all 5 cases, flank pain accompanied the leakage of contrast material. This pain was severe and long lasting in only 1 case.

SUMMARY

Adrenal venography in a series of 80 patients was useful in demonstrating the cortical tumors associated with Cushing's syndrome and virilization. Eight of 17 patients with Cushing's syndrome due to adrenocortical hyperfunction had roentgenographically enlarged glands.

This technique was successful in roentgenographically demonstrating 6 of 10 tumors associated with primary aldosteronism. The lesion was correctly lateralized in all patients who had assay of adrenal venous blood performed.

Phenochromocytomas may also be demonstrated by this technique but aortography and selective arteriography must also be performed since these tumors may be extraadrenal.

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DIAGNOSIS OF ADRENAL CYSTS UTILIZING THE PRINCIPLE OF TOTAL BODY OPACIFICATION*

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ADRENAL cysts are rare pathologic entities and are seldom diagnosed preoperatively, unless the cyst wall is calcified.[10] It is important to differentiate adrenal cysts from adrenal neoplasms in the preoperative evaluation of suprarenal masses, because often bilateral or more extensive adrenal gland exploration will be needed when endocrine secreting neoplasms are suspected.[2]

In this communication we wish to report 2 cases of noncalcified adrenal cysts, correctly diagnosed preoperatively utilizing the principle of total body opacification.

TECHNIQUE

A test tomogram is first taken, and this is inspected for position and technique. The contrast material is rapidly infused. We employ 300 cc. Reno-M-Dip (Squibb) which supplies 42.3 grams of iodine. After $\frac{2}{3}$ to $\frac{3}{4}$ of the bottle has been infused, tomography is performed in the anteroposterior projection. Single films rather than book cassettes are employed because of better detail and quality. Oblique and lateral tomograms are taken if indicated.

REPORT OF CASES

CASE I. (Fig. 1, *A–D.*) This 43 year old white female was admitted for the evaluation of a several year history of intermittent hypertension associated with flushing, palpitations and headaches. An excretory urogram at the referring hospital showed a right suprarenal mass. Multiple VMA collections were confusing, being normal 3 times and borderline elevated twice. However, none of the VMA collections were performed while the patient was having a hypertensive paroxysm.

Aortography and renal arteriography revealed an avascular mass superior to the right kidney displacing it inferiorly. No evidence of pheochromocytoma was seen in the abdomen or pelvis. Right adrenal venography was attempted but the right adrenal vein could not be catheterized.

Tomography of the mass during the infusion of meglumine diatrizoate revealed that it was homogeneously radiolucent. A paper thin wall was identified. A diagnosis of right adrenal cyst was made and this was confirmed at surgery.

CASE II. (Fig. 2, *A–C.*) This 60 year old white male was 6 years post laryngectomy for carcinoma of the larynx and was admitted for evaluation of back pain of several months' duration. Rib roentgenograms revealed the presence of a left upper quadrant mass. The excretory urography demonstrated displacement of the left kidney inferiorly. Arteriography was performed and revealed the mass to be avascular. Selective left inferior adrenal arteriography demonstrated splaying of the intra-adrenal branches around a large mass. No abnormal vessels were seen. The total body opacification showed a mass which was homogeneously radiolucent and also demonstrated a thin wall. A diagnosis of left adrenal cyst was made and this was confirmed at surgery.

DISCUSSION

Total body opacification during intravenous urography was first described by O'Connor and Neuhauser in 1963.[9] Since that report, its usefulness in Pediatric Radiology has been well recognized.[6,7] Total body opacification in adults with high dose infusion pyelography was described by Becker[1] in 1967 to visualize hypoplastic and hydronephrotic kidneys. Since that time, there have been only a few reports which indicate the usefulness of this technique in evaluating abdominal masses in adults.[5,8]

Total body opacification is the roentgenologic phenomenon in which the entire

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vascular compartment is opacified with iodine-laden blood following intravenous injection of contrast material. Avascular lesions are therefore rendered radiolucent as compared to the surrounding tissue filled with iodine opacified blood. Lesions in

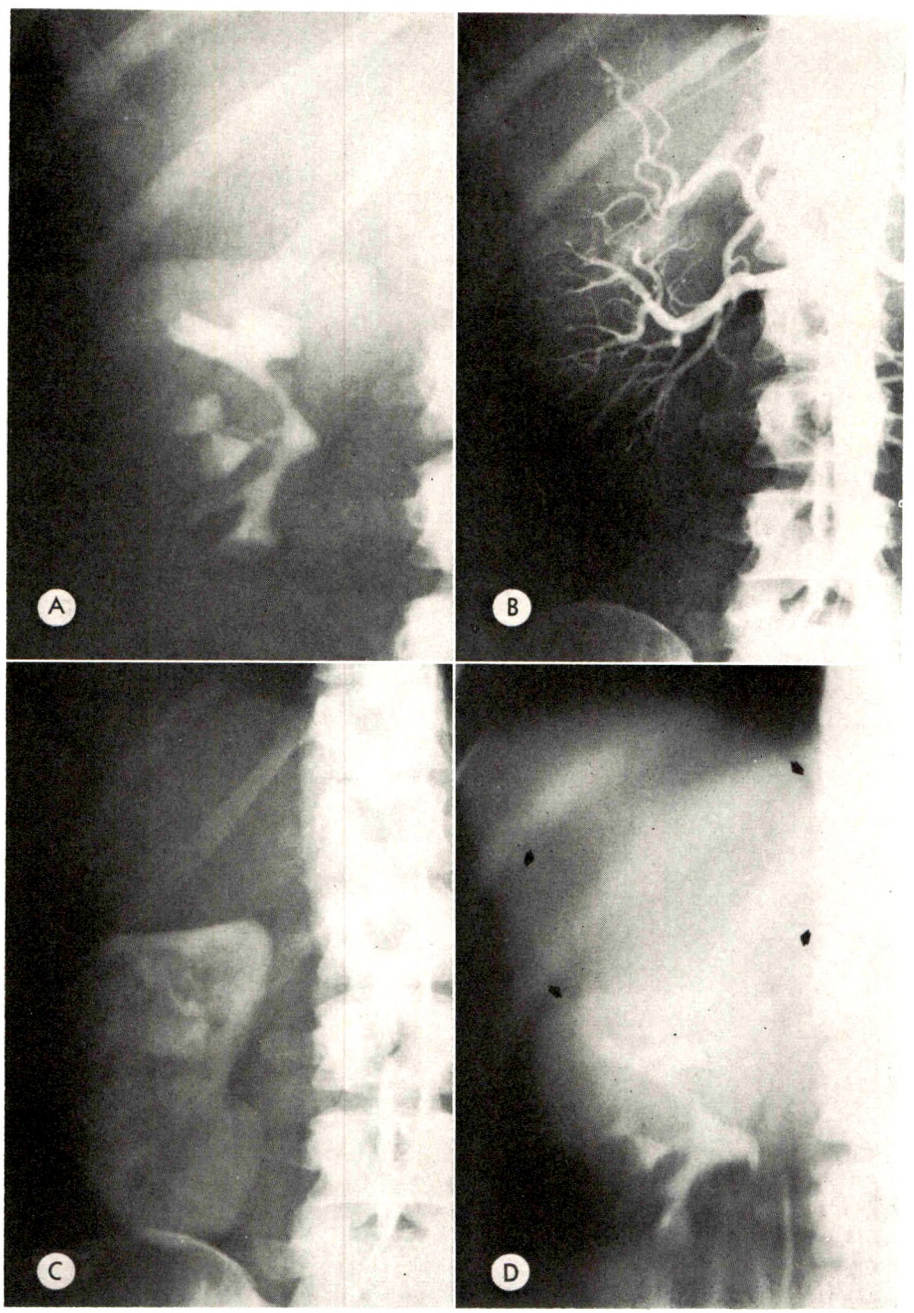


Fig. 1. Case 1. (*A*) Excretory urogram demonstrates a right suprarenal mass with flattening of the upper pole and minimal downward displacement of the right kidney. (*B*) Arterial phase of flush aortography reveals no evidence of tumor vessels in the right suprarenal area. (*C*) Delayed film from aortography shows no staining in the suprarenal mass. The cyst walls cannot be seen. (*D*) Tomogram during the infusion of Reno-M-Dip reveals the suprarenal mass to be homogeneously radiolucent and a thin cyst wall (arrows) is clearly seen.

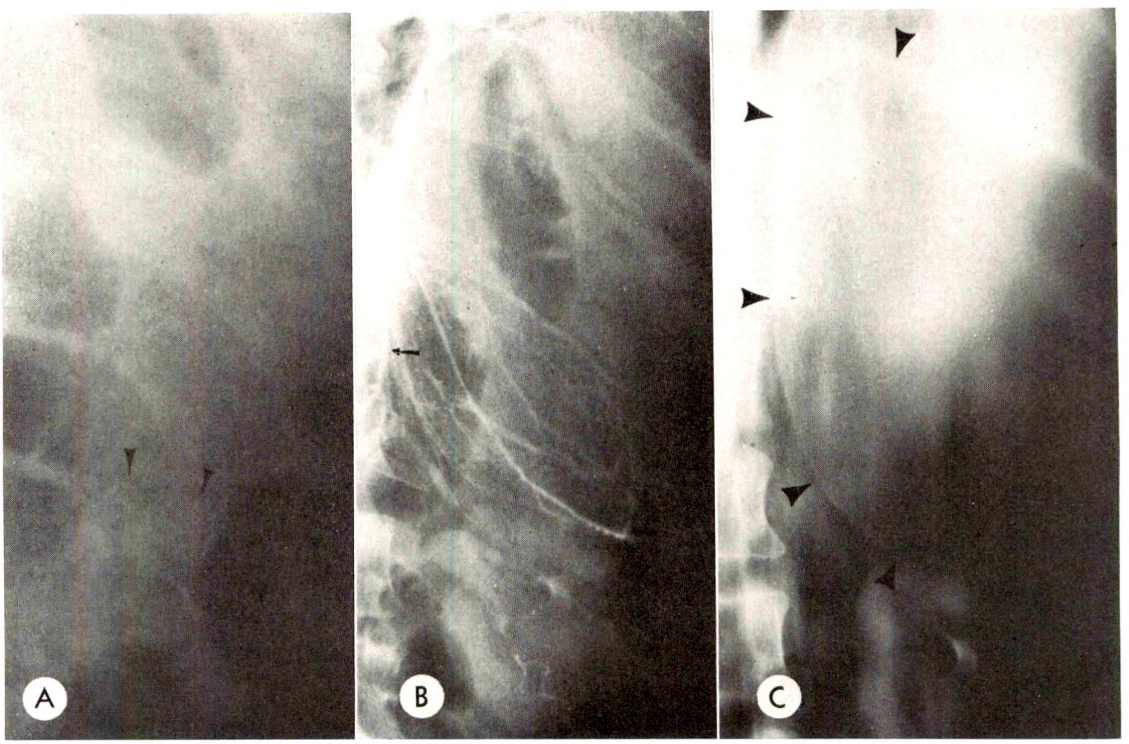


FIG. 2. Case II. (*A*) Preliminary roentgenogram from excretory urography demonstrates displacement of the left kidney inferiorly. The arrows indicate the upper pole of the left kidney. (*B*) Left inferior adrenal arteriogram demonstrates splaying of the intra-adrenal branches by a large mass. Arrow indicates the left inferior adrenal artery. (*C*) Tomogram during total body opacification clearly demonstrates a thin wall (arrows) and homogeneous radiolucency of the mass, proving its cystic nature.

which diminished or absent vascularity enables them to be visualized as lucent areas include cyst and cavity formation, hemorrhage, abscesses, areas of necrosis or infarction and fluid accumulation.

Very often, without tomography, the total body opacification effect can be seen on roentgenograms. Bowel walls and vascular structures such as the liver, spleen, or uterus are often faintly opacified; however, with the use of tomography, excellent detail of these structures may be obtained. This is particularly true of avascular areas which are rendered radiolucent compared to the surrounding tissues.

Preoperative diagnosis of adrenal cysts is often difficult. A recent report cited 220 cases of which only 17 were correctly diagnosed preoperatively; the majority were found incidentally at operation or autopsy.[4]

Roentgenographically, the plain film of the abdomen may demonstrate a soft tissue mass. The presence of a thin curvilinear laminar calcification at the periphery of a soft tissue mass in the suprarenal area is strongly suggestive of a benign adrenal cyst.[10] Unfortunately, this curvilinear calcification is usually absent. On excretory urography, there is downward displacement and rotation of the kidney.

Adrenal angiography should permit a definitive diagnosis in most cases. Arteriography demonstrates absent tumor vessels and the stretching of arteries over an avascular mass. However, because of the variable arterial supply to the adrenals, this examination can be time consuming and difficult. Adrenal venography allows excellent visualization of the architecture of the adrenal glands. Again, there may be considerable difficulty in catheterizing the adrenal veins, particularly on the right.

The technique of total body opacification offers a simple examination with no more morbidity than a routine excretory urography. The wall of the cyst is easily identified. The wall is smooth and regular and usually quite thin. The contents of the mass are homogeneously radiolucent.

SUMMARY

Adrenal cysts are rarely diagnosed preoperatively, unless the cyst wall is calcified.

The technique of total body opacification when combined with tomography is useful in evaluating avascular suprarenal masses. The cyst will appear homogeneously radiolucent and a paper thin cyst wall will be identified.

Two cases of adrenal cysts are reported, where the diagnosis was made preoperatively.

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ARTERIOGRAPHY IN TRAUMA OF THE EXTREMITIES*

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ARTERIOGRAPHY has been used in recent years more frequently in the management of vascular trauma.[5,6,9,11,13,14] Ischemia and gangrene of the extremities due to vascular injury may be recognized clinically; however, the local and peripheral signs of gangrene are occasionally atypical.[8] Clinical differentiation between arterial thrombosis, spasm and laceration may not be possible. Furthermore, pseudoaneurysm and arteriovenous fistula may simulate abscess.[1,11,13] Distal arterial pulses may be present despite arterial laceration.[11]

These facts support the need for arteriography. Demonstration of the lesion and collateral circulation is undoubtedly very helpful for proper treatment.

The purpose of this communication is to describe the arteriographic findings and to discuss their use in the management of vascular trauma of the extremities.

MATERIAL

The material consists of 17 cases studied at Chulalongkorn Hospital during 1968–1971. The patients were divided on the basis of clinical presentation into 2 groups: emergency and non-emergency groups (Table I). Gun-shot wounds and car accidents were the most common causes of trauma, occurring in 7 and 6 cases, respectively. Other causes were cut wounds from glass puncture and dog bites.

There were 10 patients in the emergency group. These patients had clinical signs of ischemia, pulse deficit and threatened gangrene. Clinical differentiation between arterial disruption and compression was difficult in some patients.

In the non-emergency group, either pseudoaneurysm or arteriovenous fistula was suspected as the causative lesion. The patients usually presented with post-traumatic swelling and appeared at the hospital between 9 days and 1 month after the injuries. Bruit was noted in all cases except 2 with very small lesions. The group was comprised of 7 cases.

TABLE I

SUMMARY OF THE ARTERIOGRAPHIC FINDINGS

Emergency Group	Cases
1. Arterial thrombosis	
Secondary to—contusion	1
—intimal tear	1
—subintimal hematoma	1
2. Complete arterial transection	2
3. Arterial ligation	3
4. Arterial compression	1*
5. Pseudoaneurysm, arteriovenous fistula and arterial ligation	1
Total	10

Non-Emergency Group	Cases
1. Pseudoaneurysm	5
2. Arteriovenous fistula	2
Total	7

* The case shown in Figure 2 was not included.

ROENTGENOGRAPHIC FINDINGS AND THEIR ASSESSMENT FOR MANAGEMENT

Trauma to the arteries from various causes may produce contusion of the artery with resultant spasm, intimal and medial injury, or thrombosis. Trauma may cause arterial laceration, complete transection, arteriovenous fistula, pseudoaneurysm or arterial compression by soft tissue edema or hemorrhage.

Soft tissue swelling or mass, fracture or

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bullet fragments may be observed on the plain roentgenogram (Fig. 5*A*).

The arteriographic findings are listed in Table 1.

In 2 cases of complete transection of the arteries, abrupt termination of the contrast medium column was seen on the arteriogram (Fig. 1). At surgery, complete transection and spasm, with a small segment of thrombosis, were found.

In traumatic thrombosis, arteriography demonstrated a blockage of the flow of the contrast medium. The intimal flap, proximal or distal end of the thrombus, was identified in 2 cases where a convex or polypoid filling defect occurred in the contrast medium column (Fig. 2). Intimal tear was noted in 1 case and subintimal hematoma was found in the other case at surgery. In another case thrombosis was assumed to have developed following arterial contusion. Arterial repair and/or graft surgery were done successfully in these patients.

The arteries were ligated in 3 cases as a first aid procedure. Abrupt termination of the contrast medium column was demonstrated; collateral circulation to fill the arteries distally was also seen (Fig. 3). Stationary waves were noted in the proximal arterial segment in 2 cases (Fig. 3). The exact sites of the ligation were demonstrated; without arteriography this could not have been possible.

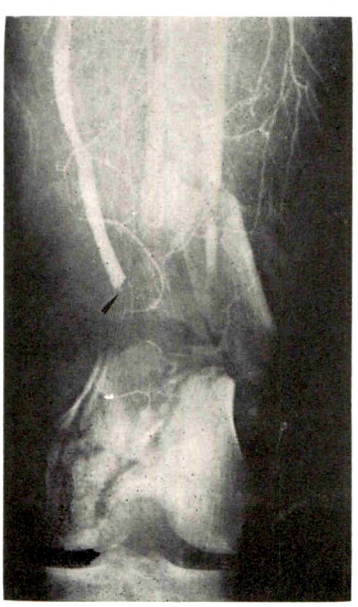


Fig. 1. Complete transection of the popliteal artery (arrowhead), secondary to comminuted fracture of the distal femur. Above knee amputation was performed.



Fig. 2. Traumatic thrombosis of the popliteal artery. Polypoid filling defect (lower arrowhead) in the contrast medium column representing intimal flap, resulting in blockage of the flow and thrombosis distally. Compression of the popliteal artery (upper arrowhead) is noted from soft tissue edema.

Figures 2 and 4, *A* and *B*, show extrinsic pressure effects on the popliteal artery causing arterial compression. Bleeding into the soft tissues was demonstrated by evidence of extravasation of the contrast medium (Fig. 4, *A* and *B*). Clinically suspected arterial disruption could be excluded.

There were 5 cases of pseudoaneurysm, varying from a few centimeters to several centimeters in size. Laceration of the arterial wall was demonstrated (Fig. 5*B*). Extraluminal collection of contrast material was seen, with prolongation of the opacification of the sac (Fig. 6, *A* and *B*). The size of the lesion was estimated by the soft tissue mass and the adjacent vascular displacement.



FIG. 3. Ligation of the femoral (upper arrowhead) and popliteal (lower arrowhead) arteries. Note the collateral circulation filling a ligated segment of the popliteal artery and the stationary waves in the femoral artery.

Arteriovenous fistula was demonstrated on the arteriogram by observing filling of the veins almost at the same time as the arteries. Site, extent and size of the lesion were well depicted. Associated pseudoaneurysm was present in 1 case in our series (Fig. 7, *A* and *B*). In this patient, ligation of the artery was done before admission.

DISCUSSION

In our series, the patients were divided on the basis of clinical presentation, into 2 groups: those with suspected arterial disruption; and those with suspected arteriovenous fistula or pseudoaneurysm.

In the first group all of the patients had signs of ischemia or threatened gangrene. Arteriographies were performed to establish a more accurate diagnosis, since the clinical signs of ischemia are sometimes atypical and do not reflect the true condition.[8] Clinical differentiation of arterial spasm, thrombosis or laceration following skeletal trauma is not always possible.[14]

The sites, size and extent of the lesions were demonstrated as well as the collateral circulation. Differentiation between arterial compression and disruption was made by arteriography in our cases.

Arteriographically, abrupt termination of the contrast medium column in an artery

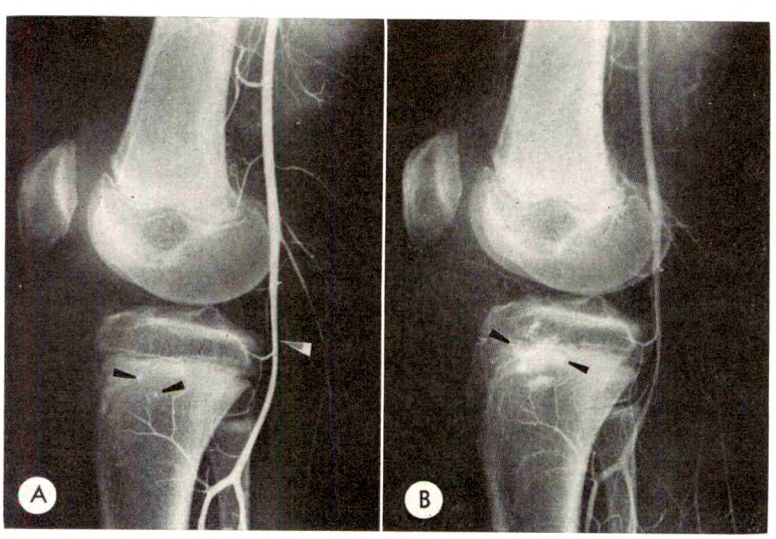


FIG. 4. (*A*) Arterial compression in a case with fracture of the mid tibia, result of a car accident. Compression of the popliteal artery (white upper arrowhead) and extravasation of the contrast medium into the soft tissue (black lower arrowheads) are observed. (*B*) More extravasation of the contrast medium into the soft tissue (black arrowheads) was seen in the late arterial phase. The patient had threatened gangrene of the leg.

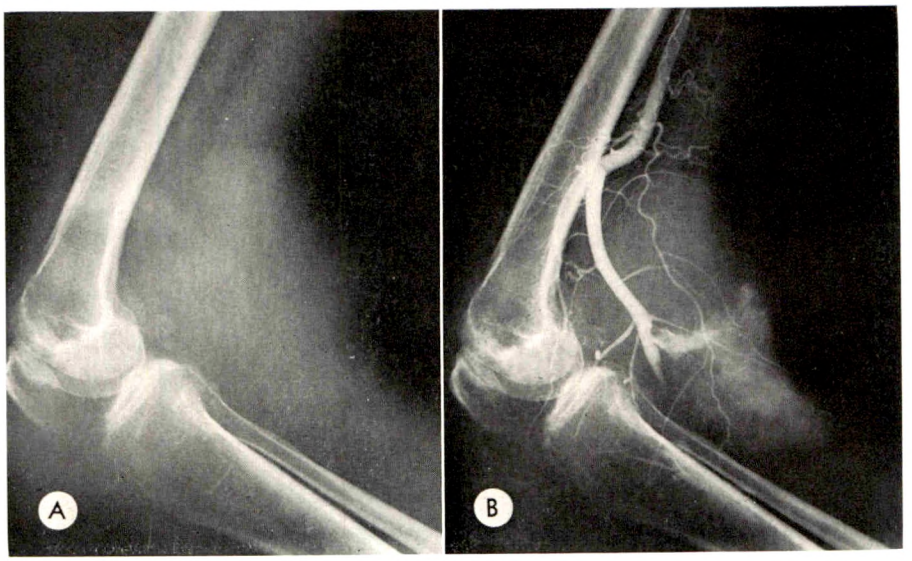


Fig. 5. (*A*) A soft tissue mass is seen on the plain roentgenogram in a case of a pseudoaneurysm of the popliteal artery. (*B*) There is laceration of the popliteal artery with extravasation of the contrast medium and vascular displacement.

may represent arterial ligation, thrombosis, or complete transection. A convex filling defect in the contrast medium column, if present, is characteristic of thrombosis.[11] In 2 cases out of 3, the arterial thrombosis was due to intimal injuries. It is our belief, that intimal tear or subintimal hematoma causes blockage of the flow, followed by thrombosis.

In 2 cases of complete transection, abrupt termination of the contrast medium column was noted. This finding resulted from arterial spasm associated with thrombosis which occurred after complete transection of the arteries.

A crush injury may produce circumferential compression, as reported by Curry and Howland.[5] Bassett and Silver[1] state that edema and hemorrhage within a closed compartment, when of sufficient magnitude, may lead to compression of the arteries. Compression of the popliteal artery

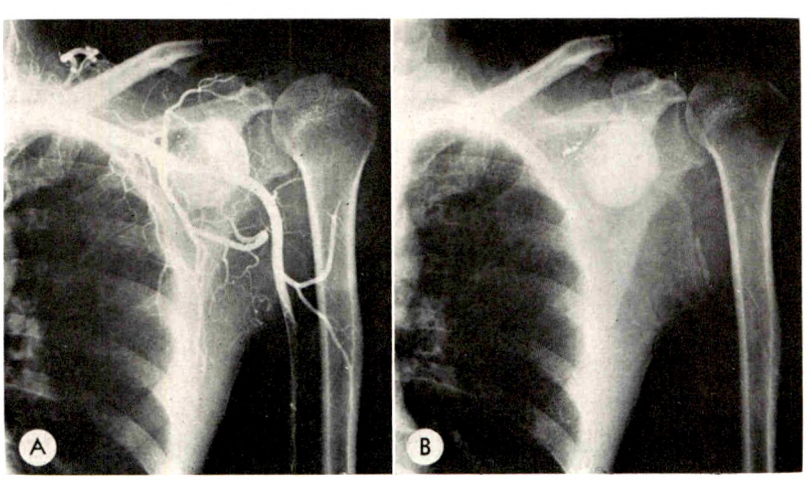


Fig. 6. (*A*) Extraluminal collection of contrast medium in a pseudoaneurysm of the axillary artery is seen, occurring after a bullet wound. (*B*) There is prolongation of opacification of the sac 6 seconds later.

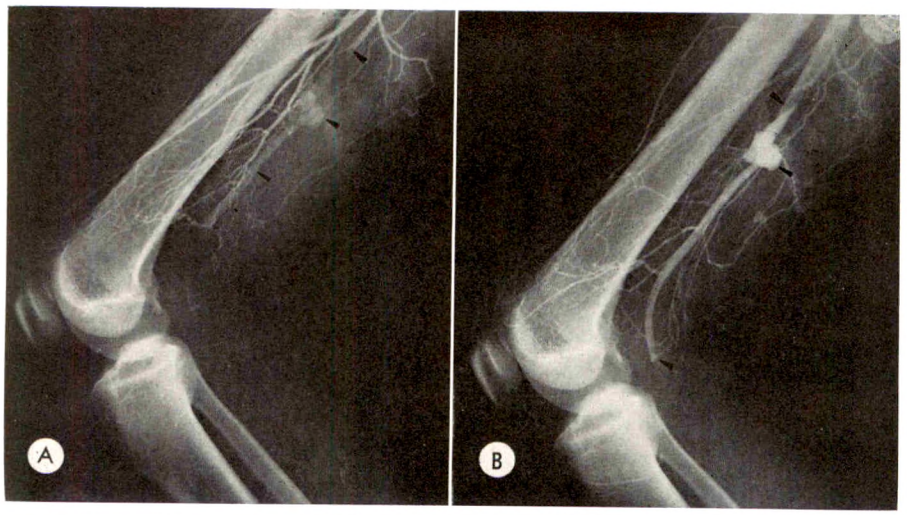


FIG. 7. (*A*) A small pseudoaneurysm (middle arrowhead) originating from the femoral artery after a cut wound, requiring first aid ligation (upper arrowhead). Stationary waves are noted in the femoral artery. Collateral vessels filling the popliteal artery (lower arrowhead) are demonstrated. (*B*) Filling of the femoral vein (upper arrowhead) is seen 1 second later with better opacification of the pseudoaneurysm (middle arrowhead). Ligation of the popliteal artery (lower arrowhead) is noted.

was well demonstrated in our cases; we believe that extension of the leg exaggerated the compression in this patient.

In the second group, pseudoaneurysm or arteriovenous fistula was suspected. The characteristic arteriographic findings of the 2 lesions have been previously described by many authors.

It has been mentioned that pseudoaneurysm or arteriovenous fistula may simulate an abscess. One case of pseudoaneurysm in our series had the appearance of an abscess. Pseudoaneurysm and arteriovenous fistula may present as a late complication of trauma.[12,13] However, Fomon and Warren[7] believe that hematoma leads to the formation of an arteriovenous fistula or pseudoaneurysm immediately, but recognition of the lesion is late. Association of arteriovenous fistula and pseudoaneurysm is not uncommon.[9,11]

The peripheral pulses were normal in 4 out of 7 cases of pseudoaneurysm and arteriovenous fistula. This is in agreement with the findings of Love and Braun,[11] and Sacks.[13]

The indications for arteriography in patients with trauma to the extremities have been amply discussed.[8,11,14] In our experiences, arteriography is indicated in those patients who exhibit signs of ischemia, pulse deficit, suspected arterial disruption, or unarrested bleeding, and in those patients who are suspected of having arteriovenous fistula or pseudoaneurysm.

The clear demonstration of the lesions by arteriography proved to be helpful in the proper management of this group of patients. Vascular reconstruction with restoration of vascular continuity is a safe procedure.[3,10]

CONCLUSION

Arteriography was performed in 17 cases of vascular injuries of the extremities.

The lesions were well demonstrated as well as their site, size and collateral circulation.

Correct diagnoses were made, resulting in proper management.

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TANDEM FILM CHANGERS FOR PERIPHERAL ANGIOGRAPHY*

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MANY approaches have been used to provide an answer to the problem of satisfactory peripheral arteriography that would provide a reliable method of opacifying all vessels from the abdominal aorta to the ankles in a standardized examination. Bi-plane special roentgenographic procedure rooms can be utilized for peripheral angiography by placing the film changers in tandem in the anteroposterior projection as described below.

A bi-plane special procedure room equipped with at least 1 film changer which can be changed from its normal vertical position to a horizontal position, together with 2 tubes able to be centered over the changers in the horizontal position can be utilized. In our suite, two 14×14 inch cut film changers are utilized in conjunction with a tilting roentgenographic table with image intensified fluoroscopy. (A non-tilting, standard catheterization type roentgenographic table may also be used.) In addition to the longitudinal excursion of the motor driven table, a supplementary sliding plastic top provides additional excursion of the patient. Two separate roentgenographic generators and controls are employed in our room so that it is possible to independently vary the roentgenographic technique for each changer. In our particular room the exposure sequence of the power injector and changers is controlled by a punch card system. This provides a greater economy of filming, with excellent timing, than with the usual multisequence timing methods.

METHOD

The 2 changers are placed in tandem 8 inches apart. The 8 inch separation is necessary to provide sufficient overlap when the patient is moved for the second injection. Divergent magnification and consequent lack of overlap can be a serious problem at the junction between pelvis and thigh films because of the large separation between the vessels and the changer when the patient is in the anteroposterior projection, such as with transfemoral aortography and peripheral arteriography. With the translumbar approach, of course, with the patient prone, divergent distortion and lack of overlap is much less of a problem. The basic system consists of filming of the pelvis and the knees simultaneously with a single injection. The patient is then shifted 11 inches cephalad, and a second injection is made filming the thighs and the lower legs. The total coverage with this system is 46½ inches. The first and second injections consist of 45 cc. of renografin 60 per cent injected at a rate of 15 cc. per second. During the first injection the punch card programmer is set to expose 5 films of the pelvis and 7 of the knees, with an appropriate sequence to provide 18 seconds of filming to the level of the trifurcation of the popliteal artery.

During the second injection 7 films of the thighs and 8 films of the lower leg are exposed for a total of 26 seconds of filming from the beginning of the injection. A total of 27 films are therefore obtained during the procedure.

RESULTS

The results so far have indicated that this program provides excellent reproducible coverage without the necessity of making additional injections or change in the program to individualize it for patients

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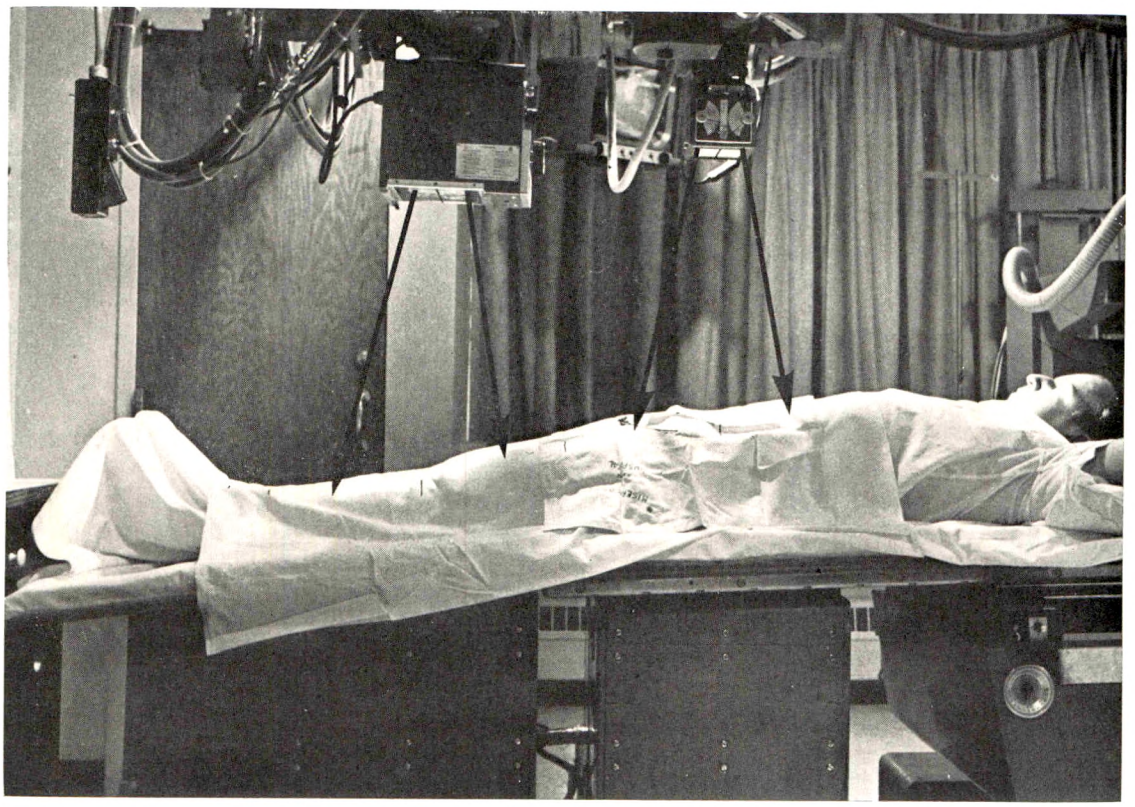


Fig. 1. Patient in place for simultaneous filming of the lower abdominal aorta and arterial tree of pelvis, as well as the popliteal area and trifurcation.

with varying circulation times. It is appropriate to both transfemoral catheter and needle procedures involving retrograde counter current flow to the level of the aortic bifurcation, and to translumbar aortography without modification.

DISCUSSION

Many methods have been used to provide reliable peripheral arterial visualization from the lower abdominal aorta through the lower legs. With an increasing interest in vascular grafting of small vessels, a reliable standardized method with a minimum of repeat injections is desirable.

Briefly the following methods have been used:

1. Single filming long leg cassette: This is the 36 inch or 51 inch variety and does not produce reproducible results with standardized parameters.

2. Moving crane arteriography: A roentgenographic tube with a slit moves longitudinally and exposes each portion of a long leg cassette in succession. By speeding up the tube toward the end of the run, some compensation for decreasing thickness of the extremities occurs at the expense of proper timing of filming.

3. Single film changers, stationary table technique: This usually requires 3 or more injections to perform a satisfactory study of the pelvis, thighs and knees.

4. Intravenous aortography with moving table roentgenography, utilizing a single rapid film changer: This is of historic interest but it provides inadequate visualization of the peripheral vessels in many cases.

5. Moving table arteriography with radioactive triggering: Because of a difference in circulation times between both legs in some cases, this method may not be entirely reliable.

6. Moving table arteriography over a

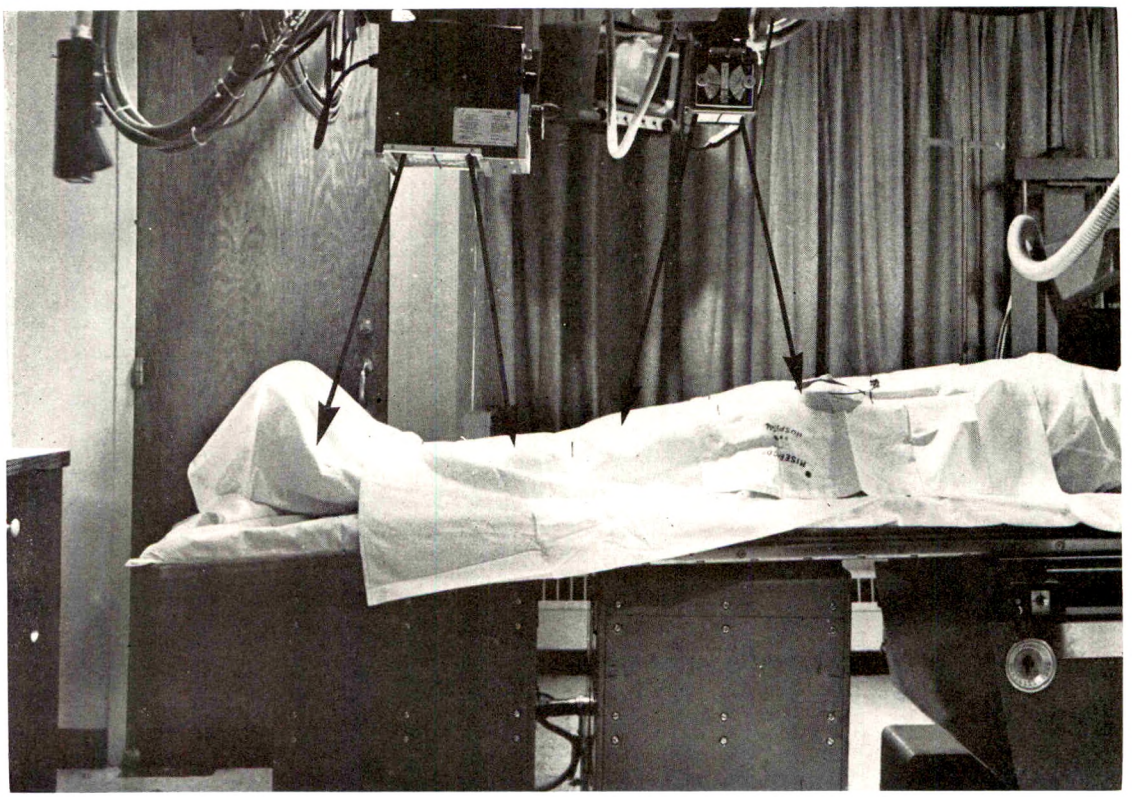


FIG. 2. After an 11 inch cephalad shift of the patient there is simultaneous filming of the arterial tree of the thigh and lower leg.

single film changer: Prolonged injection times and high volume injections have increased the reliability of this method. A compact power injector attached to the table is desirable to prevent the catheter from being pulled out while the table is moving. Fluoroscopic observation of a test bolus at the level of the knees increases reliability.

7. Long leg cassette multiple film changer: This is a fairly reliable single injection method utilizing large volumes of contrast material slowly injected. A soffit in the ceiling or high ceiling mounted tube is necessary for adequate coverage of the cassette. Some changers suffer from magnification distortion on successive films. Often it is not possible to place the changer in such a position that the patient is easily fluoroscoped to check the catheter or needle position with test injections.

Of the above, the motor driven programmed table top over a single film changer is probably the most popular proven method, and the multifilm long leg changer is probably the most reliable in producing a standardized examination with a single injection. The reliability increases with the size and duration of the injection. Quantities of contrast material up to 100 cc. are used by arteriographers for these methods. Even so, reliability rate for producing a complete examination with a single standardized study is somewhere between 70 and 95 per cent. The tandem film changer 2 injection system can at least equal the reliability of the above specialized equipment for a standardized examination.

CONCLUSION

A tandem film changer system for peripheral arteriography represents an adaptation of an existing bi-plane special procedure room to peripheral arteriography without

the need for a separate long leg cassette changer or a programmed motor driven table top system.

The system produces results that are at least as reliable as those obtained with specialized equipment.

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NONSURGICAL TREATMENT OF POSTCATHETERIZATION FEMORAL ARTERY OCCLUSION— A NEW TECHNIQUE*

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ARTERIAL thrombosis following retrograde catheterization procedures has been an infrequent but serious complication in humans over the past several decades.[2,6] In the literature, emphasis has been placed on prompt and adequate surgical correction of the problem. The fact that delay in therapy often occurs in surgical thrombectomy and that surgery may also have its own complications has made necessary a search for other means of approaching the problem.

The increasingly widespread use of catheterization studies in recent years has meant an accompanying increase in the absolute number of complications associated with the procedure. Prevention of thrombosis is the ideal solution and has been approached in many ways by investigators, with partial success.[9] Protective heparinization of patients was introduced by Murray over 30 years ago.[8] Improvement in catheters through the use of new materials and the addition of catheter coatings has had the effect of diminishing thrombosis and embolization.[1,4,5] However, thrombosis of arteries is still an anticipated complication; and the patient must be informed of this possibility. Moore *et al.*[7] cite a 1.2 per cent incidence of thrombosis requiring surgery in retrograde catheter studies.

The purpose of this paper is to describe a new percutaneous nonsurgical thrombectomy method performed in the angiographic suite immediately following the diagnosis of arterial occlusion. The present study is confined to dogs except for 1 human case. It is expected that the value of the method will be further demonstrated in humans in the future.

MATERIAL AND METHOD

Six large mongrel dogs were used in this study, their weights ranging from 15–19 kilograms. All dogs were anesthetized with intravenous sodium pentobarbital (30 mg. per kg.). The animals were placed in the supine position on the fluoroscopic table, and percutaneous transfemoral introduction of an 8 French polyurethane catheter was carried out. The tip of the catheter was placed in the upper descending aorta of each dog initially. At 10 minute intervals, the catheter was withdrawn to the extent that the tip reached the level of the aortic bifurcation. The tip was then advanced again to the upper aorta. This maneuver served the purpose of stripping formed clot from the catheter and depositing it near the arterial puncture site as previously described by Nejad *et al.*[9] Femoral arteriograms were obtained 10 minutes after placement of the polyurethane catheter using a methylglucamine and sodium diatrizoate contrast agent. The contrast material was injected through a short polyethylene catheter which had been introduced percutaneously in the opposite femoral artery with its tip lying at the aortic bifurcation.

Another arteriography was performed in the same manner after occlusion of the femoral artery was obtained as evidenced by lack of pulsation distal to the initial puncture site. This occlusion occurred 40 minutes to 2 hours after the introduction of the polyurethane catheter. When complete

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arterial occlusion was determined by angiography and palpation, another percutaneous arterial puncture was made approximately 2 cm. distal to the site of obstruction at the polyurethane catheter entrance. A 9 French teflon catheter was passed for a short distance into the vessel over a guidewire. The polyurethane catheter was then removed, and the vessel was compressed until arterial flow from the puncture site had stopped. A slightly tapered teflon sheath which fits snugly over the teflon catheter was then advanced into the artery. The device is seen in Figure 1. The teflon catheter and first sheath were then removed; and markedly diminished blood flow was noted through the sheath, apparently due to "back bleeding" from the artery distal to the thrombus. A 5 French Fogarty balloon catheter was then passed through the sheath to the level of the bifurcation. After the balloon was inflated with air, the balloon catheter was withdrawn from the iliac artery; and large clots were retrieved in each case. The procedure is diagrammed in Figure 2. One to three passes of the Fogarty balloon catheter were performed as needed. The sheath was then removed, and compression of the puncture site was maintained until no arterial leakage was evident. An arteriography was then performed via the polyethylene catheter extending over the bifurcation. Follow-up arteriograms were obtained after 4 to 10 days in 4 animals (Fig. 3, *A–D*).

RESULTS

A. ANIMAL STUDY

All 6 dogs in this study showed re-establishment of flow in the femoral artery and its branches following catheter-induced thrombosis with subsequent percutaneous clot removal. Small clots attached to the vessel wall were seen at the puncture sites in the arteriograms immediately following clot removal, but these appeared not to alter distal flow significantly. Follow-up angiograms were performed on 4 of the animals. One animal showed mild narrowing of the vessel at the puncture site 4 days post-thrombectomy (Fig. 3*E*). This was an improvement over the appearance at time of thrombectomy (Fig. 3*D*). Three other dogs had repeat angiograms done 7 to 10 days after the initial procedure, and only minimal narrowing was seen at the puncture site in 3 of the 4 animals. The other animal showed no significant abnormality of the vessel.

B. HUMAN STUDY

A 65 year old white female with metastatic adenocarcinoma to the liver (primary site unknown) underwent hepatic arteriography on October 19, 1972. At the time of arteriography a catheter was left in

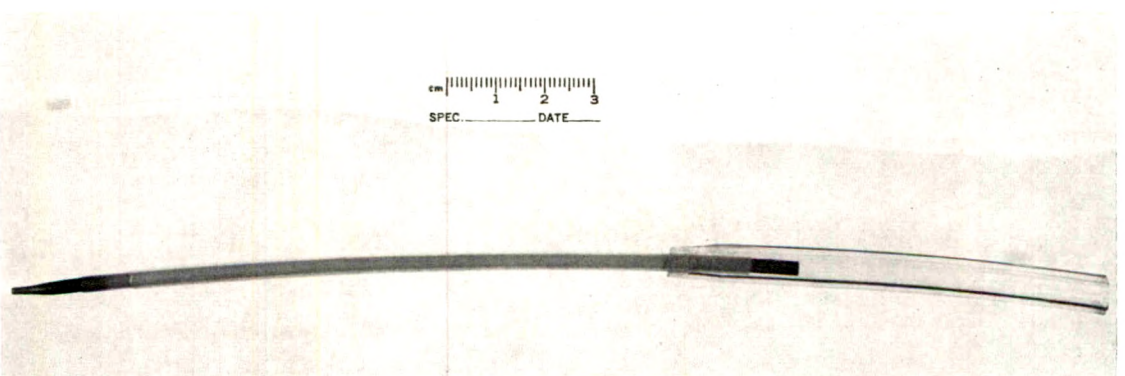


Fig. 1. The clot retrieval device consists of the black 9 French teflon catheter surrounded by 2 teflon sheaths which fit over each other snugly. The tapered vinyl tubing on the right allows one to control blood flow with a clamp across the tubing.

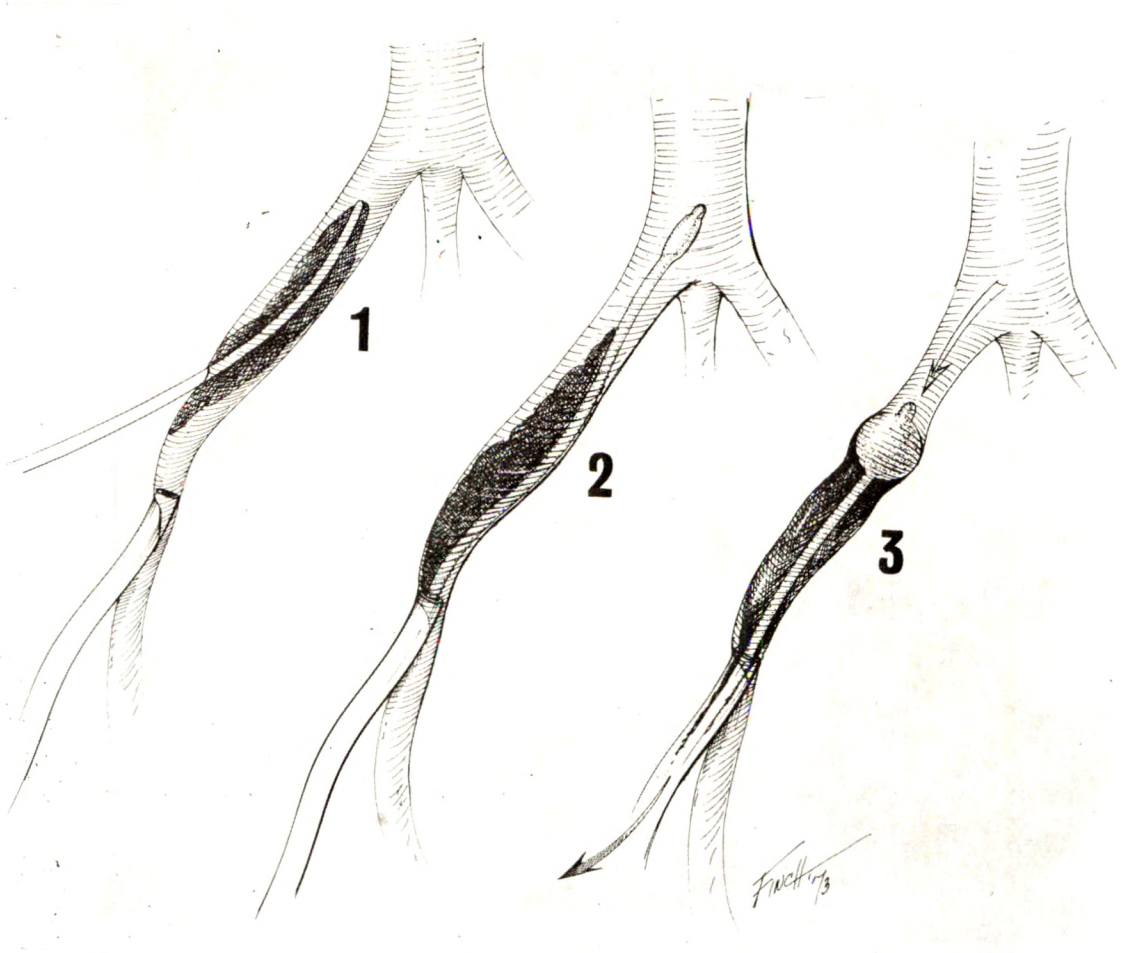


FIG. 2. *Diagram 1.* Clot is noted to surround the upper catheter and obstruct blood flow in the femoral artery. The teflon sheath is in place distal to the clot. *Diagram 2.* The initial upper catheter has been removed and a deflated 5 French Fogarty balloon catheter is advanced through the sheath so that its tip lies above the clot. *Diagram 3.* The Fogarty balloon is inflated with air (or CO_2) and then withdrawn. This maneuver brings the adherent clot into the sheath. The sheath is then withdrawn and pressure applied to the puncture site.

place for long-term hepatic perfusion using a chemotherapeutic agent. Six days later complete occlusion of the right femoral artery was noted on angiography (Fig. 4). No distal pulses were present in the right lower extremity.

A teflon sheath was introduced 3 cm. below the entrance of the perfusion catheter in the same manner used in the animal study (Fig. 5). A large thrombus was removed through the sheath using a Fogarty balloon catheter (Fig. 6). Restoration of blood flow in the femoral artery was obtained, and an excellent peripheral pulse was present in the right posterior tibial artery. Five thousand units of heparin were given intravenously at the beginning of the procedure, and 50 mg. of protamine sulphate was given at the completion of the study as suggested by Nejad and associates.[9]

A follow-up angiogram (Fig. 7) from the opposite femoral artery was obtained 2 days later, and excellent flow was noted in the previously occluded right femoral artery.

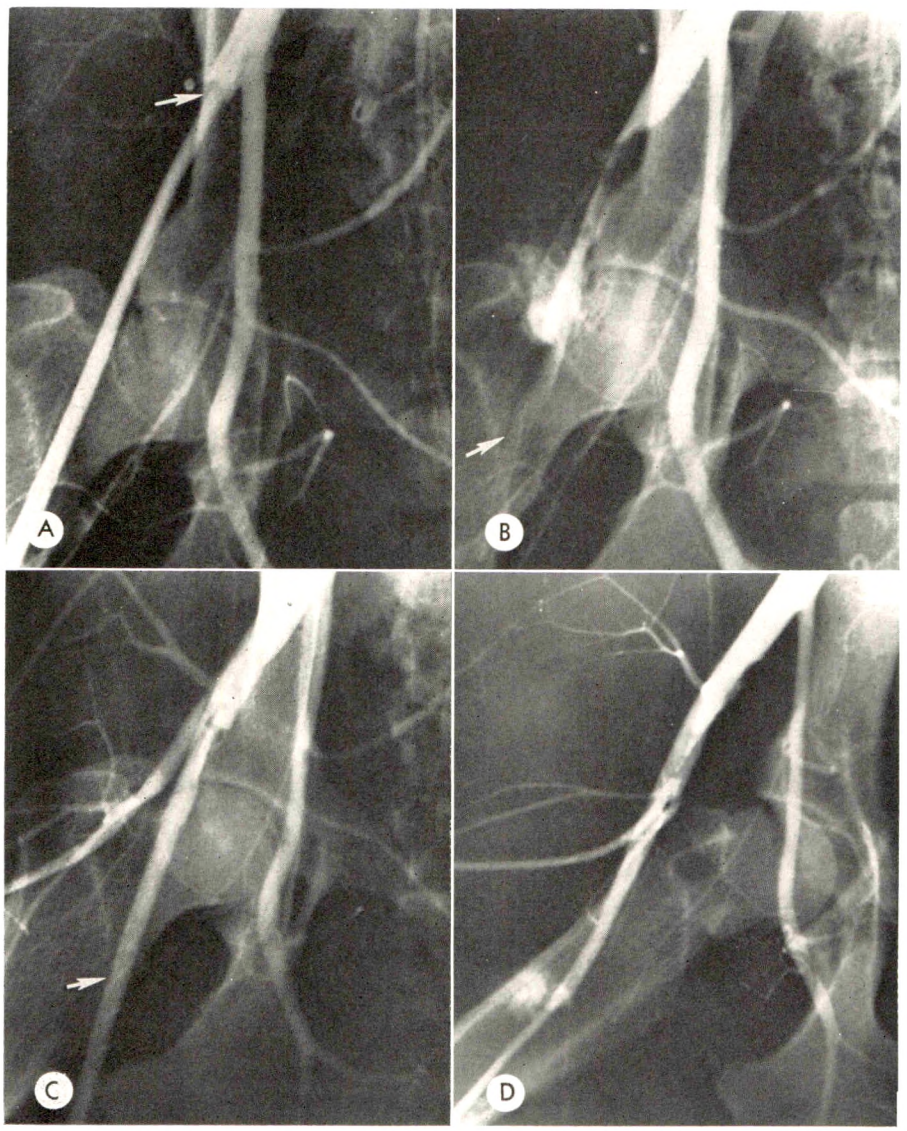


Fig. 3. (*A*) Arteriogram shows contrast material filled polyurethane catheter in right femoral artery of dog with obstruction of the arterial flow by clot (arrow). (*B*) The catheter has been removed with considerable clot obstruction remaining. Mild extravasation of contrast material is noted at the puncture site. The teflon sheath is present in the artery (arrow). (*C*) The teflon sheath (arrow) is now filled with contrast material following use of the Fogarty balloon. Note opacification of the arterial branches not well seen previously. (*D*) Patency of the femoral artery is noted after removal of the sheath. Extravasation and distal arterial spasm are noted at the sheath puncture site. Mild arterial deformity remains near the upper puncture site.

DISCUSSION

Percutaneous thrombectomy promises to offer an improvement over surgical thrombectomy in numerous cases of arterial occlusion related to catheter studies. A principal advantage is immediate therapy in the angiographic room after demonstration of postcatheterization thrombosis. Delay in therapy has been noted as one of the drawbacks of surgical treatment. Prompt therapy provides a clot which is less mature and less adherent to the vessel

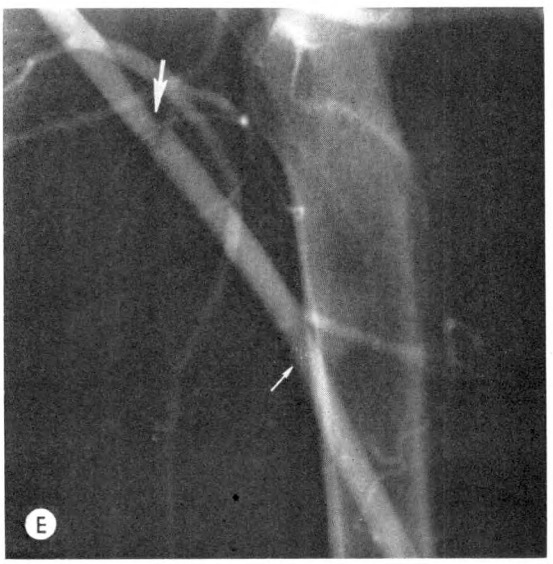


FIG. 3. (*E*) Four days after percutaneous thrombectomy, mild vascular deformity remains at the upper puncture site (large arrow). No significant deformity remains at the lower sheath puncture site (small arrow).

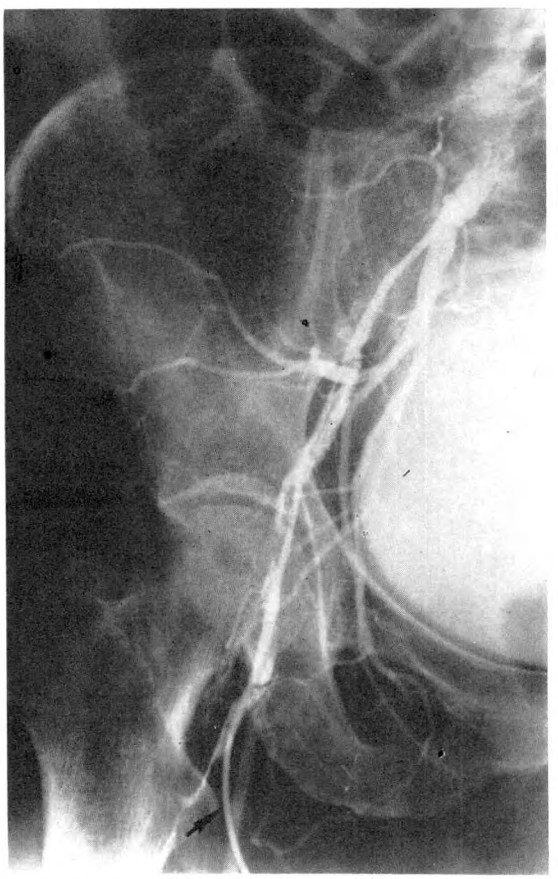


FIG. 4. Complete occlusion of the right femoral artery is noted in a 65 year old female following catheter placement of 6 days' duration. Arteriography is performed via catheter (arrow).

wall. Each of the 6 cases in dogs shows reestablishment of flow distal to the site of thrombosis. Flow is also noted to be maintained on follow-up angiograms. Femoral artery puncture 2–3 cm. below the original catheter position site is not expected to be particularly difficult in spite of an absent palpable pulse. The large experience of Cramer *et al.*[3] has shown that the thrombus usually does not propagate distally because the deep circumflex femoral artery represents the main collateral preserving patency of the superficial femoral artery. Puncture of the occluded artery will, therefore, yield good but reduced arterial blood flow due to "back bleeding."

Another major advantage over the surgical technique is angiographic visualization of the clot, run-off, and post-thrombectomy results. The success of a surgical procedure performed in the operating suite is sometimes difficult to assess because of peripheral spasm.

Our animal studies deal with thrombosis in essentially normal vessels, and it is unlikely that such uniformly good results would be obtained in humans with atherosclerotic vessels or with previous surgical scarring in the area. In case of failure, surgical thrombectomy can still be performed if the percutaneous method does not provide adequate flow. Care should be taken to position the sheath distal to the clot with its tip aimed in a retrograde manner, so that blood flow will direct fragments of thrombus into the sheath if they break loose during catheter maneuvers.

SUMMARY

Arteriotomy has been the treatment of choice in arterial occlusion which occurs in

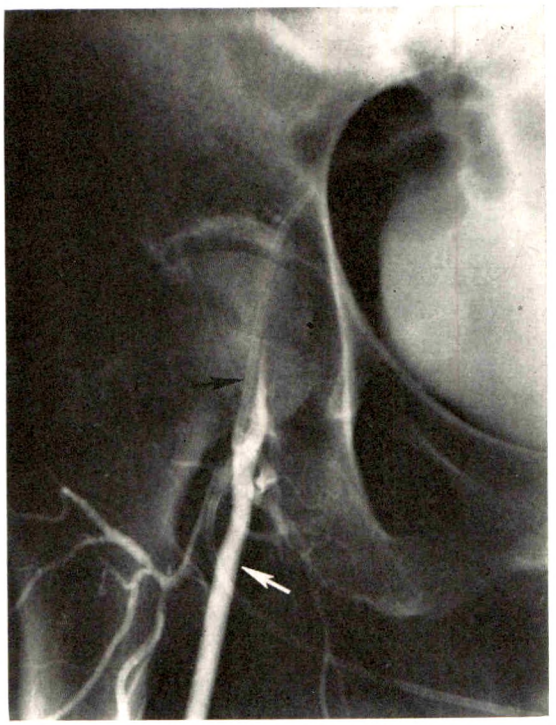


Fig. 5. Teflon sheath (white arrow) is placed about 3 cm. distal to initial catheter (black arrow).

approximately 1 per cent of cases of femoral artery catheterization. However, recent experimentation in femoral artery occlusion in dogs has demonstrated that thrombi can be successfully removed percutaneously.



Fig. 6. Large clots removed from femoral artery.

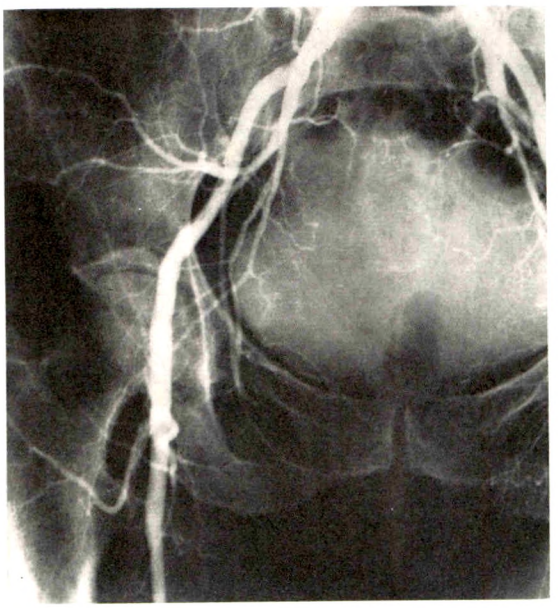


Fig. 7. Two days later, a follow-up angiogram shows excellent flow in the right femoral artery.

A large bore teflon sheath which approximates vessel size is passed into the artery over a catheter which is placed distal to the site of arterial obstruction. The clot is expelled spontaneously by the blood stream or extracted with a Fogarty balloon catheter passed through the sheath.

Blood flow has been restored in 6 animals using this method.

Successful clot removal has been performed in 1 human case. Further human studies are presently planned for retrieval of thrombi associated with percutaneous catheterization.

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ANGIOGRAPHIC IMPLICATIONS IN DIAGNOSIS AND PROGNOSIS OF BASILAR ARTERY OCCLUSION*

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BASILAR artery occlusion often presents a confusing clinical picture and the diagnosis rests upon angiographic findings. Angiographic examinations were made, without complication, on 9 patients who had total occlusion of the basilar artery.

This report compares the angiographic findings with the clinical data and evaluates these findings as prognostic indicators. Complete occlusion of the basilar artery does not necessarily result in death. Six of the 9 patients in this study have survived.

CLINICAL MATERIAL AND OBSERVATIONS

Five of the 9 patients were men, 4 were women. Age range was from 10 to 74 years with an average age of 49 years. The duration of symptoms preceding angiography was from a few hours to 10 years. Five of the patients had symptoms suggesting basilar artery occlusion for over 1 year. In each of these cases the patients had had complete recovery from initial symptoms. Three other patients had transient symptoms for several weeks prior to angiography. One patient underwent angiographic examination after an episode of unconsciousness and died the same day.

The most common symptoms were alteration in consciousness, vertigo, ataxia, dizziness, dysarthria, hemiparesis or hemiplegia, and visual disturbances. All 9 patients were hemiparetic or hemiplegic at some time during the course of their symptoms. Eight patients had speech difficulty and visual dysfunction. Seven patients complained of vertigo or dizziness. According to the clinical records, despite the character of the symptoms, basilar artery occlusion was unsuspected prior to angiography in 7 of the 9 cases.

Through the follow-up period of 3 months to 5 years, 6 of our patients have survived—some show only minor neurologic deficit. One patient, mentioned above, died the day of angiography. Another, whose condition became progressively worse, died 3 weeks after angiography. The third patient died from a brain abscess secondary to meningitis, 2 months after angiography (Fig. 1). In each instance necropsy confirmed the roentgenographic finding of basilar artery occlusion.

ROENTGENOGRAPHIC FINDINGS

Angiography of 1 vertebral artery and both carotid arteries was performed for all but 1 patient. In those instances in which no reflux could be achieved into the opposite vertebral artery, both vertebral arteries were injected. Because of her poor condition, the vertebral study was not performed in 1 patient.

In all but 2 patients (Fig. 2, *A* and *B*), the site of occlusion was distal to the origin of both posterior inferior cerebellar arteries. In these 2 patients, one of the posterior inferior cerebellar arteries was involved in the occlusion. Occlusion was distal to the origin of the anterior inferior cerebellar arteries in 1 patient (Fig. 3). In all instances, the occlusion extended to a site just proximal to the origin of the superior cerebellar arteries.

The posterior cerebral and superior cerebellar arteries were opacified from various pathways in all 9 patients. When carotid arteriography was performed in 7 patients,

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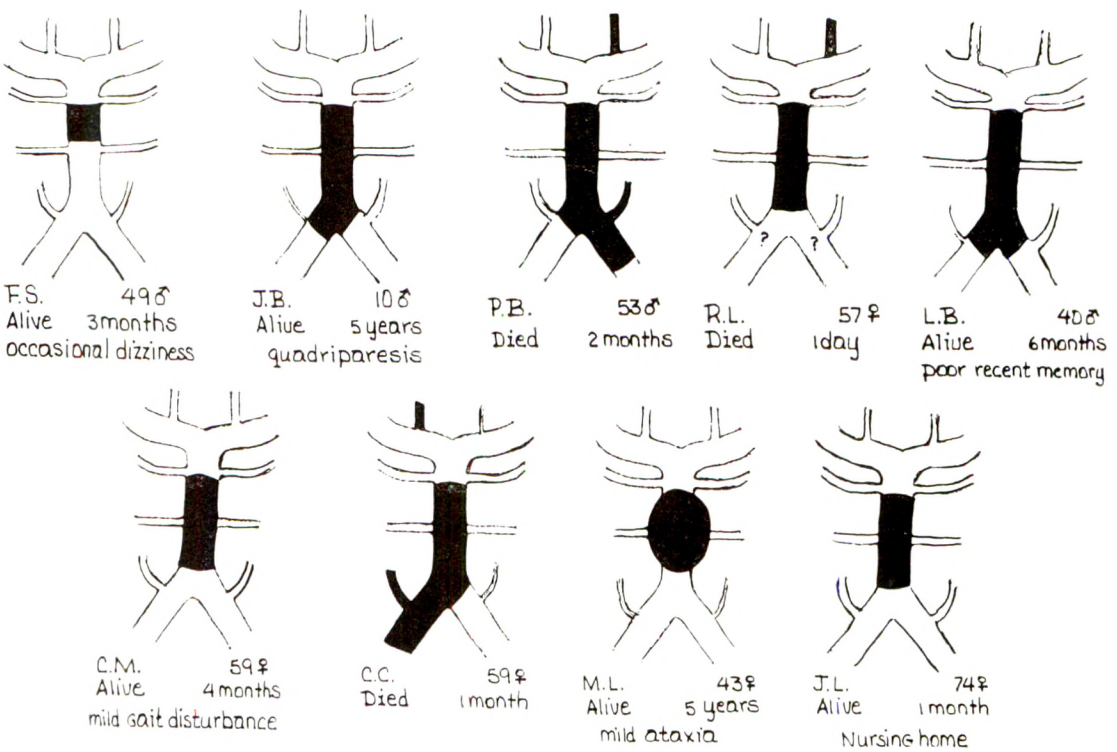


FIG. 1. Schematic drawing indicates the site and extent of occlusions of the basilar artery in 9 patients. Follow-up periods are noted.

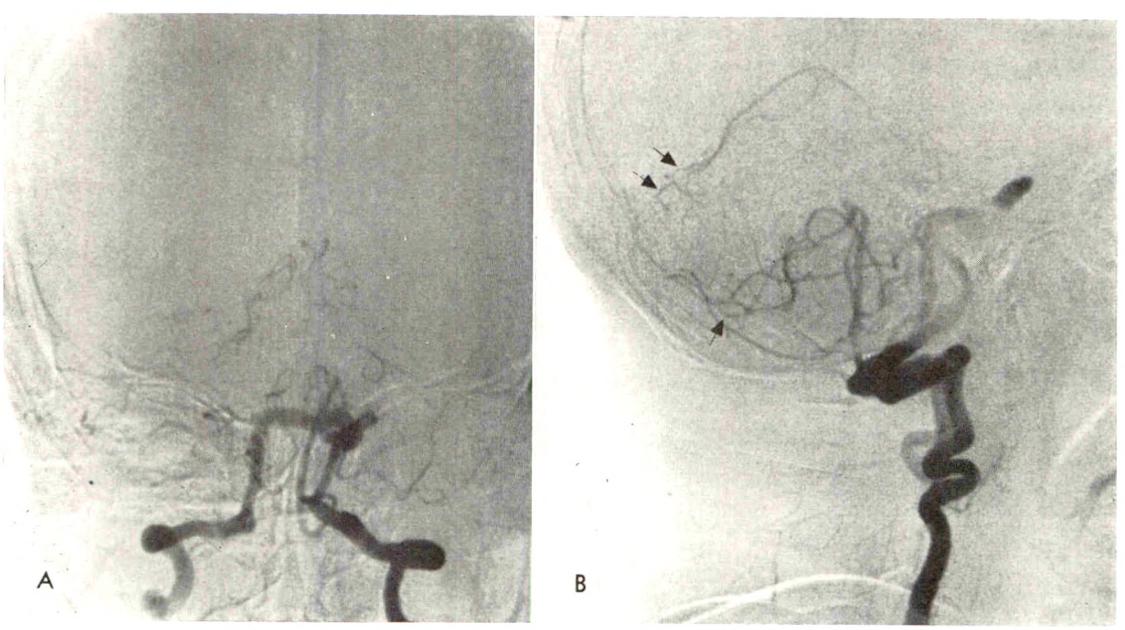


FIG. 2. Left vertebral angiograms. (*A*) Anteroposterior and (*B*) lateral views show reflux into the distal, right vertebral artery. The basilar artery is occluded just distal to the origins of the posterior inferior cerebellar arteries. Note collateral filling of the superior cerebellar artery *via* vermian branches of the posterior inferior cerebellar artery (arrows).

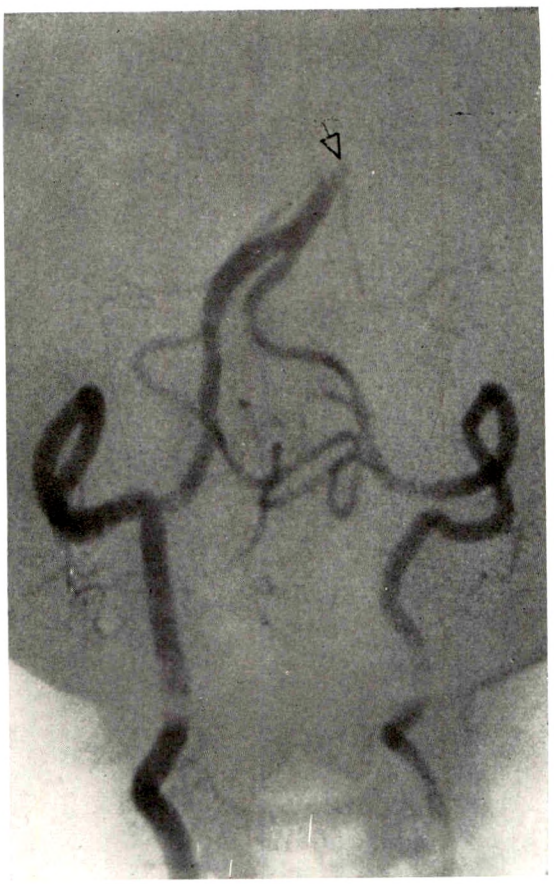


Fig. 3. Right vertebral angiogram shows occlusion (arrow) of the basilar artery immediately distal to the origins of the anterior inferior cerebellar arteries.

both posterior cerebral arteries were filled by patent posterior communicating arteries. The superior cerebellar arteries subsequently filled *via* the tip of the basilar artery (Fig. 4, *A* and *B*). In those 2 patients in whom the posterior communicating arteries carried no flow, the posterior cerebral arteries were filled in a retrograde fashion *via* pial anastomoses between posterior branches of the middle cerebral artery and posterior branches of the posterior cerebral artery (Fig. 5, *A–D*). As in the other 7 patients, the superior cerebellar artery was demonstrated after the contrast medium reached the basilar tip.

In 5 of 9 patients, filling of the superior cerebellar arteries was achieved on the vertebral angiogram as a result of anastomoses between vermian and hemispheric branches of the posterior inferior cerebellar artery and corresponding branches of the superior cerebellar artery (Fig. 2, *A* and *B*). Posterior meningeal branches of the vertebral artery contributed to collateral flow in 3 patients (Fig. 6). In 1 patient (Fig. 7), an hypertrophied tentorial branch of the meningohypophyseal trunk added to the flow. This same patient had an enlarged anterior spinal artery which provided an additional supply of collateral blood (Fig. 8).

Two patients had pneumoencephalography. One showed a large calcified aneurysm in the region of the midportion of the basilar artery (Fig. 9). The thrombosed aneurysm did not fill at angiography. The other patient had enlargement of the prepontine cistern, indicating atrophy of the pons.

REPORT OF CASES

Case I. A 59 year old woman was admitted to the hospital after having been found comatose by her husband. She had been hospitalized twice in the preceding 18 months for episodes of stupor, unsteady gait, and slurred speech. Despite multiple tests no diagnosis had been reached. Since her previous admission, she had done well and had returned to work complaining only of occasional dizzy spells.

At physical examination her eyes were conjugatively deviated to the left. Corneal reflexes were absent. Although the patient was able to open and close her eyes on command, she was unable to move her extremities. Sustained ankle and knee clonus were elicited. Babinski's reflex was exhibited bilaterally. The face, trunk, and extremities showed reaction to noxious stimuli.

Cerebral angiography indicated complete occlusion of the basilar artery.

While in the hospital the patient showed progressive improvement. Follow-up examination 4 months after angiography showed only minor disturbance in gait. The patient was otherwise asymptomatic and neurologically normal.

Case II. A 49 year old man was admitted to the hospital with right hemiparesis and right-sided numbness, dizziness—with falling to the right, and dysarthria. He had been well until

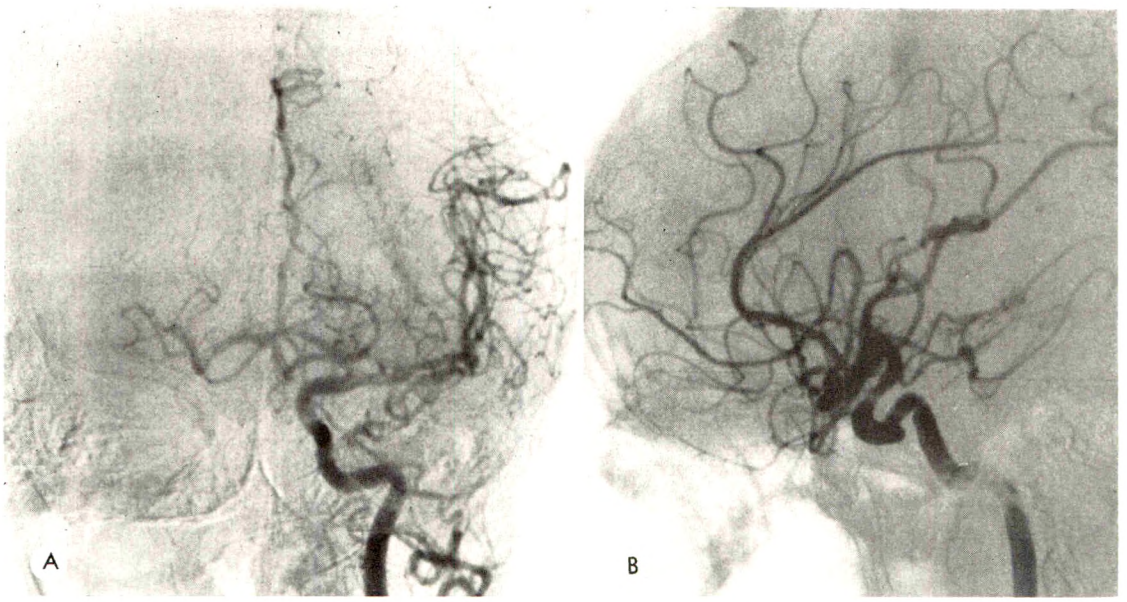


FIG. 4. Left carotid angiograms. (*A*) Anteroposterior and (*B*) lateral views demonstrate filling of posterior cerebral artery and superior cerebellar artery from the posterior communicating artery.

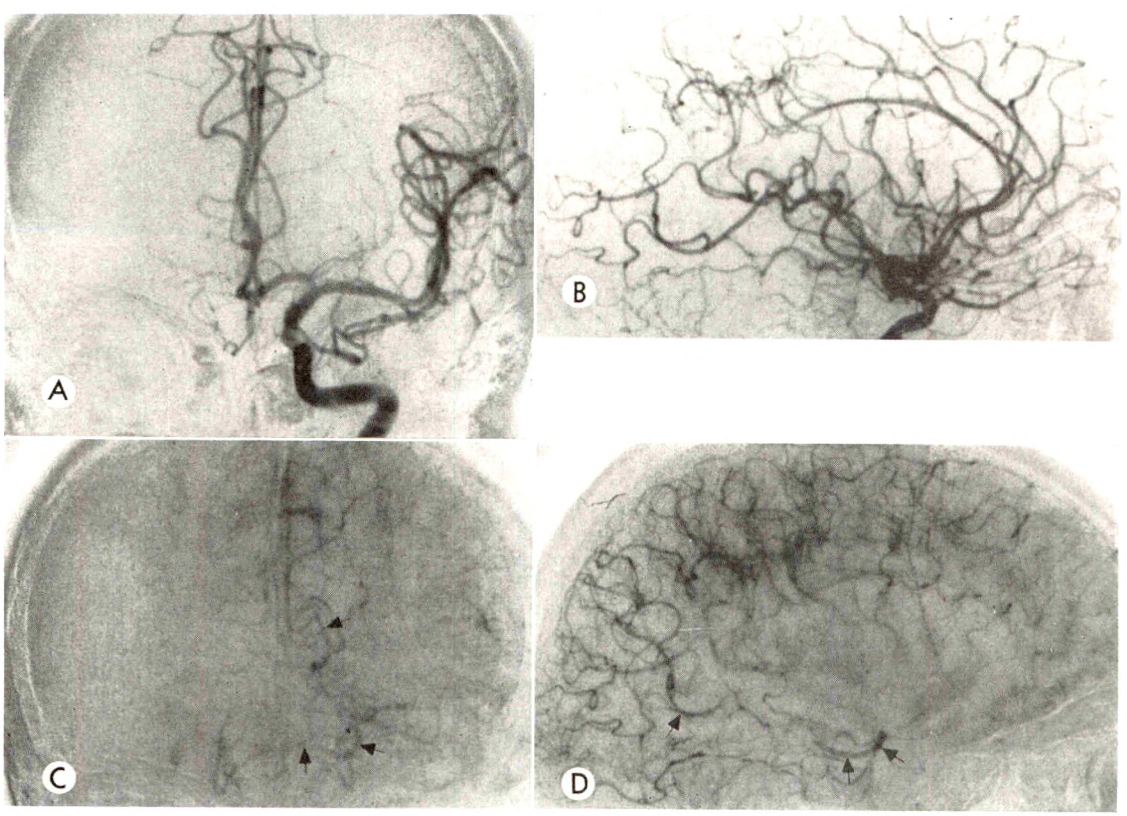


FIG. 5. (*A*) Anteroposterior and (*B*) lateral left carotid angiograms in early arterial phase show no filling of posterior cerebral artery from the injection of the carotid artery. (*C*) Anteroposterior and (*D*) lateral left carotid angiograms, late arterial phase, indicate retrograde filling of posterior cerebral artery (arrows) from posterior branches of the middle cerebral artery. The superior cerebellar artery fills after opacification of the basilar tip.

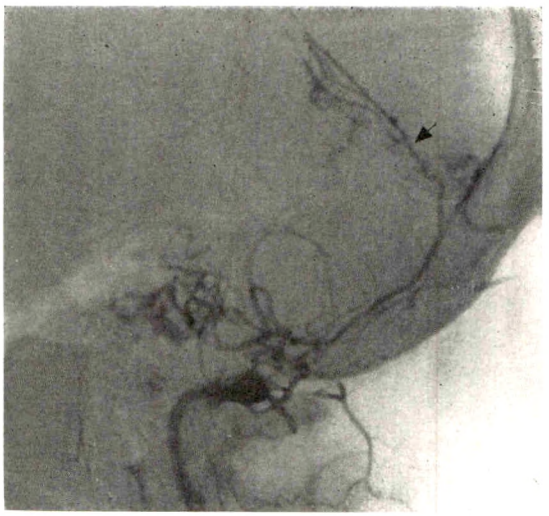


Fig. 6. Right vertebral angiogram shows proximal occlusion of the basilar artery. There is poor filling of the posterior inferior cerebellar artery. Collateral supply to the superior vermian branch of the superior cerebellar artery by means of the posterior meningeal branch of the vertebral artery is demonstrated (arrow).

3 weeks before admission when he had a sudden onset of dizziness, nausea, vomiting, diplopia, and left-sided weakness. This episode lasted only a few minutes, then completely cleared. In the interval between the onset of symptoms and admission, he had had several such transient episodes with alternating hemiparesis.

Physical examination showed no abnormality. Neurologic examination revealed central facial weakness on the right and a mild weak-

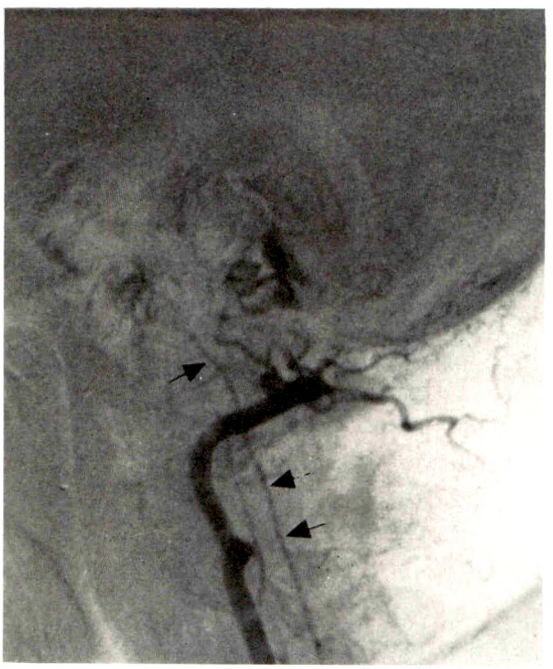


Fig. 8. Left vertebral arteriogram shows an enlarged anterior spinal artery (arrows) serving as collateral blood supply to the brain stem.



Fig. 7. Left internal carotid arteriogram shows a hypertrophied tentorial branch (arrow) of the meningohypophyseal trunk adding to the collateral flow.

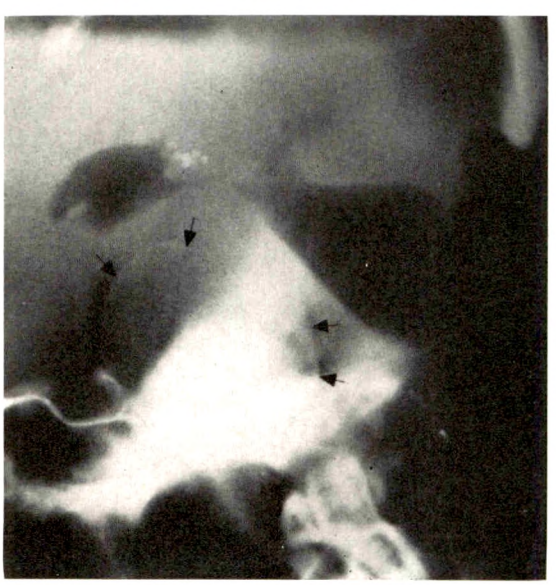


Fig. 9. Lateral pneumoencephalogram demonstrates a large calcified aneurysm of the basilar artery (arrows), which was totally occluded at angiography.

ness of the right extremities. The patient walked with a wide-based gait and tended to drift toward the right. Cerebral angiography disclosed complete occlusion of the basilar artery.

During his hospitalization the patient's symptoms cleared; he was discharged. Examination 6 months after angiography showed a mild, right hemiparesis. No other abnormalities were noted.

CASE III. A 47 year old man was admitted to the hospital for evaluation after a "blackout spell" occurred while he was driving his car. Seven years before he had been evaluated following 2 transient episodes of light-headedness and falling to the right side. No diagnosis was reached at that time.

Physical examination at the time of admittance indicated mild, left hemiparesis; no other neurologic abnormality was noted. Cerebral angiography indicated complete basilar artery occlusion.

Six months after angiography, the patient complained of slight clumsiness with his left arm and occasional dizziness. Physical examination was unrevealing.

DISCUSSION

Basilar artery occlusion and its accompanying clinical picture has been recognized for more than 100 years.[8,11] Before 1946, basilar artery occlusion was a retrospective diagnosis. Of 22 cases reported by Kubik and Adams in 1946,[10] 18 were of basilar artery occlusion with detailed correlation between symptoms and pathologic findings. The other 4 patients demonstrated characteristic symptoms; these patients survived. To our knowledge, however, the diagnosis was not confirmed. In 1956, Haugsted[7] made the angiographic diagnosis of occlusion of the basilar artery. Shortly thereafter (late 1950s) arteriographic findings for this condition were reported.[5,9,13] Until that time, it was generally accepted that basilar artery occlusion was catastrophic—usually fatal—and, if not fatal, capable of causing severe neurologic changes. In 1966 Fields and his coworkers[6] described 8 patients who survived basilar artery occlusion (follow-up period was 18 months).

Occlusion of the basilar artery is not rare. It was noted to appear in 1 of every 300 necropsy specimens at the Boston City and Massachusetts General Hospitals.[10] Of 4,834 necropsy examinations conducted at Bellevue Hospital in New York from 1938–1958, 0.2 per cent of the patients had had complete occlusion of the basilar artery.[3] Over a 5 year period at the Mayo Clinic, 1 of about every 450 necropsies revealed basilar artery occlusion.[15]

Although basilar artery occlusion has been reported to occur in all age groups, the majority of patients are over the age of 50. Men are afflicted slightly more often than women. The youngest recorded case is that of a 6 year old boy who had typical symptoms; the cause was undetermined.[4] We have demonstrated basilar artery occlusion in a 10 year old child. Schechter and Zingesser[14] reported basilar artery occlusion to have occurred in a 16 year old person.

The great majority of occlusions of the basilar artery are due to atherosclerosis. Occurrence of basilar artery occlusion due to embolus has been reported in various percentages. Kubik and Adams[10] reported 7 of 18 cases of basilar artery occlusion to be due to embolus; Cravioto *et al.*[3] reported 2 of 14. Many early-reported cases were caused by syphilitic endarteritis.[13] Other causes such as trauma and infections have been noted as well. One case of phycomycotic thrombosis of the basilar artery in a 23 year old patient with lymphoblastic leukemia has been reported.[2] Upon the death of one of our patients, necropsy disclosed thrombosis secondary to meningitis. Loop and his co-workers[12] described a basilar artery trapped and occluded in a fracture of the clivus. Thrombosis in a patient with polycythemia has been described, as well as has accidental ligation during a surgical procedure.[2]

Aneurysms of the basilar artery are not uncommon. No case could be found in the literature, however, of survival following complete occlusion of the basilar artery secondary to thrombosis of a basilar artery aneurysm. One of our patients, a 43 year

old woman, has survived for 5 years after the diagnosis of thrombosed aneurysm of the basilar artery.

Patients with basilar artery occlusion fall basically into 2 categories: (1) those with abrupt onset of coma and rapid progression to death; and (2) those with transient symptoms of brain stem ischemia. Patients in the latter group often present a problem in diagnosis and are of particular concern to radiologists.

The acuteness with which occlusion occurs is directly reflected in the outcome of the condition. In the case reported to be due to accidental ligation, the patient died within hours after the surgical operation.[1] In the instance in which the basilar artery was trapped within a fractured clivus, the patient quickly died.[12] The 7 patients of Kubik and Adams[10] who had embolic occlusion died much sooner than those who had atherosclerotic thrombosis. One of our patients who had suddenly become unconscious without antecedent symptoms died just hours after angiography.

To our knowledge no relation between angiographic findings and prognosis has been previously reported. Well established collaterals through the circle of Willis and anastomosis between branches of the basilar artery, however, certainly favor survival. In our series, the 2 patients in whom the occlusion involved one of the posterior inferior cerebellar arteries died within 2 months after angiography. Significantly, these 2 patients also had no flow through one of their posterior communicating arteries; this absence of flow necessitated retrograde flow from the middle cerebral artery to fill the posterior cerebral and superior cerebellar arteries. Our third patient who died had no vertebral angiogram; she too, however, had only one, small patent posterior communicating artery. The surviving 6 patients, had all major collateral pathways available. These 9 cases support the contention that a direct relationship exists between the availability of collateral pathways and survival.

SUMMARY

Angiographic examinations were made of 9 patients who had complete occlusion of the basilar artery. Six are alive 3 months to 5 years after diagnosis.

In 7 of the patients the diagnosis was not suspected prior to angiography. The commonest site of occlusion was just distal to the origins of the posterior inferior cerebellar arteries. Various patterns of collateral flow were observed.

A direct relationship is indicated between patency of collateral pathways and prognosis.

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ANGIOGRAPHIC APPROACH TO THE DIFFICULT AORTIC ARCH: A NEW TECHNIQUE FOR TRANSFEMORAL CEREBRAL ANGIOGRAPHY IN THE AGED*

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CEREBRAL angiography and brachiocephalic catheter angiography have become widely employed,[1,2,4,9–12,15,16,18] particularly for relatively young patients in whom the procedure is quite simple using any of a variety of catheters. An occasional exception to this simplicity is posed by the origin of the left common carotid artery from the brachiocephalic trunk. Elderly patients, on the other hand, present unique problems which may seriously hinder selective catheterization. These include: (1) tortuosity or stenosis of the iliac arteries; (2) dilatation and elongation of the aortic arch (Fig. 1*A*); (3) sharply angled brachiocephalic vessel origins (Fig. 1*B*); and (4) tortuosity of their cervical courses (Fig. 1*C*). Despite these problems, it is often necessary to selectively study such vessels. The method of study must be judged not only by its diagnostic quality but also by its relative ease and safety.

Some angiographers have preferred to forego selective cerebral angiography in the elderly in favor of diagnostically substandard expedients; *i.e.*, aortic arch injections with[5] or without[6,23,24,26] subtraction technique. Others[10,22] have filled in the quality gap by supplementary multiple approach studies.

This paper describes the use of a new catheter technique (Fig. 2), specifically designed for the elderly but also useful in any adult-sized patient for catheterizing the left common carotid artery which arises from (Fig. 3*A*) or is contiguous with the brachiocephalic artery (Fig. 3*B*). Most selective catheter techniques,[4,12,15,16] when used to deal with the manifold problems of aged vessels, share the drawback that the catheter must be pushed to its final placement, a maneuver which often snaps the catheter from the vessel of interest back into the aorta. Methods that modestly place the catheter tip at the orifice of the target artery are not sufficiently secure in their position to prevent catheter recoil, particularly if the aorta is large, and they are characterized by excessive contrast reflux and dilution for adequate delineation of the intracranial circulation.

MATERIAL

From February, 1972 to December, 1972, 213 of 615 cerebral angiographies at Loma Linda University Hospital were performed with the long open-loop catheter to be described. The more difficult presbyaortas and the less accessible left common carotid arteries in patients of any age were the indications for the use of the new catheter.

TECHNIQUE

With infrequent exceptions, the transfemoral approach is used; a No. 7 French Cordis* polyurethane catheter is preferred because of its superior torque control. Should the transaxillary route appear more prudent in a given case, No. 5–6 French polyethylene or No. 6–7 French polyurethane catheters may be used. Generally, a compatible selective-nonselective catheter size combination is chosen which will allow opacification of the aortic arch prior to selective catheterization. The technique is suitable for either the common carotid artery, the left vertebral artery, or

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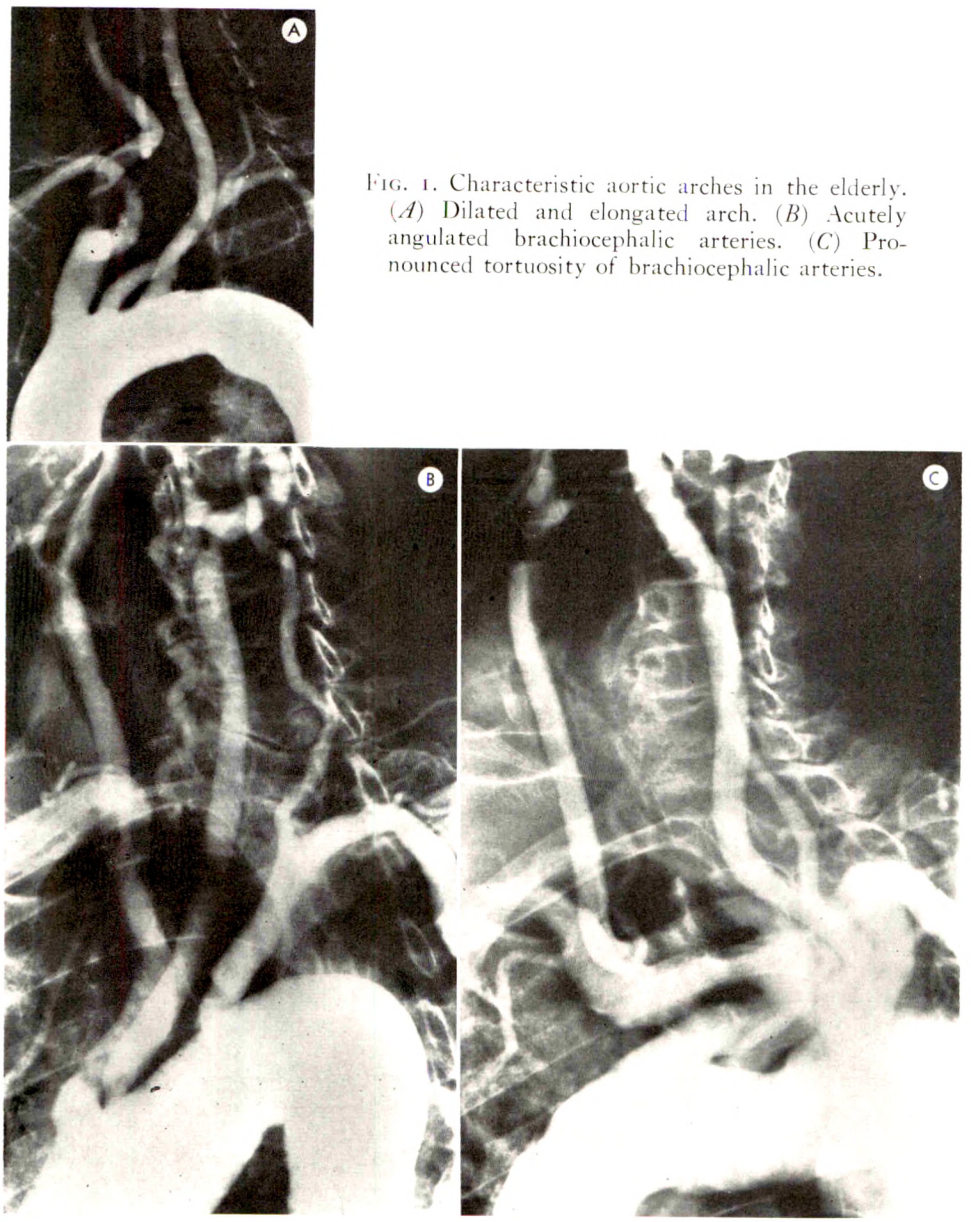


FIG. 1. Characteristic aortic arches in the elderly. (*A*) Dilated and elongated arch. (*B*) Acutely angulated brachiocephalic arteries. (*C*) Pronounced tortuosity of brachiocephalic arteries.

the brachiocephalic and subclavian arteries. It is unsuitable for selective catheterization of the internal or external carotid arteries. The right vertebral artery is catheterized with ease only from the right transaxillary approach.

Peculiar to the catheter described herein is dependence upon a withdrawal motion to enter and properly position the catheter within the brachiocephalic vessels—a step which both minimizes trauma to these arteries and circumvents the vector difficulties inherent in catheterizing acutely angled arch vessels. It also minimizes the unfavorable effect of aorto-iliac tortuosity on torque control, by maximally straightening the catheter within the patient.

TRANSFEMORAL APPROACH

(Fig. 4, *A–D*)

Usually the catheter is inserted into the right common femoral artery under local anesthesia, using the percutaneous Sel-

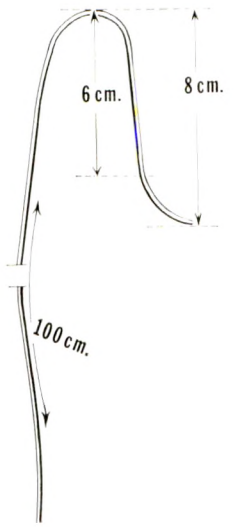
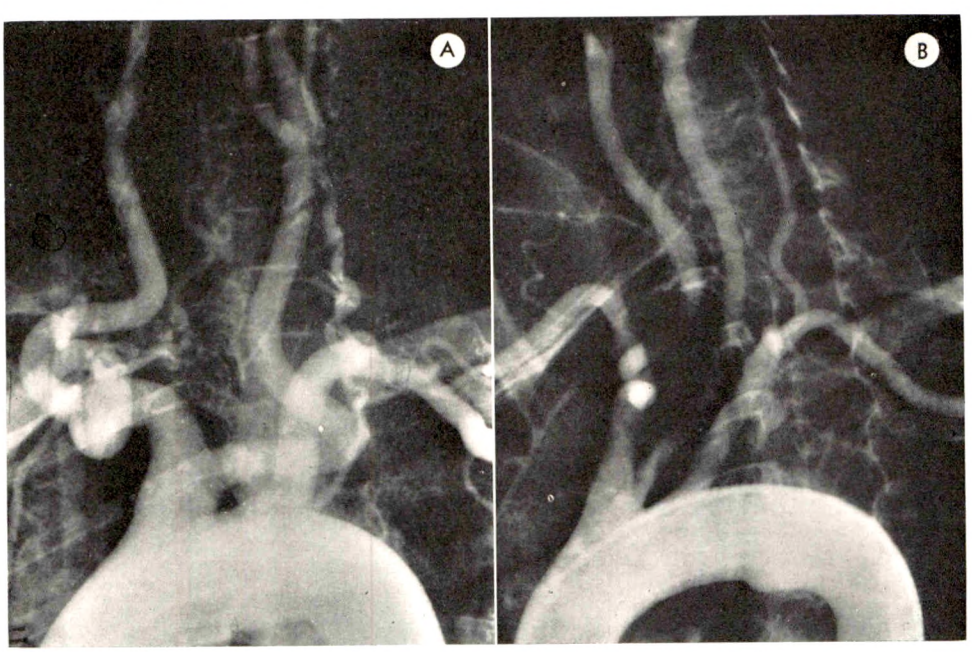


Fig. 2. Sketch of catheter.

dinger method.[21] The catheter is advanced over a movable core safety J-guide†[13,14] to the proximal descending aorta (#1). The guide-wire is withdrawn slightly and the tip of the catheter advanced into the left subclavian artery (#2). Led by the guide-wire to avoid vertebral artery entry (#3), the catheter is then advanced to a point just distal to the left vertebral origin, and the guide-wire is withdrawn to the aortic arch. Further advancement of the catheter causes the preformed open loop to develop in the ascending aorta (#4), whereupon the guide-wire is removed and the catheter flushed. The catheter is withdrawn, its tip engages the brachiocephalic artery (#5), and then advances within it to a position of stable maximal penetration in the right carotid artery as withdrawal continues (#6). Overwithdrawal results in loss of the open loop configuration.

When the right common carotid artery or the brachiocephalic artery is studied first, catheterization of the left carotid or the left vertebral arteries simply involves catheter advancement out of the brachiocephalic trunk and into the aorta to allow the loop to reform. The catheter is rotated slightly during withdrawal in order to bypass the brachiocephalic trunk and then brought back into line to be engaged in the vessel of interest. The guide-wire generally need not be reinserted, once the open loop has been formed for the first selective

† Safety and Safety J-Guides. Cook, Inc., Bloomington, Ind.



IG. 3. (*A*) Left common carotid artery originating from the brachiocephalic trunk. (*B*) Origin of the left common carotid artery contiguous with the brachiocephalic artery.

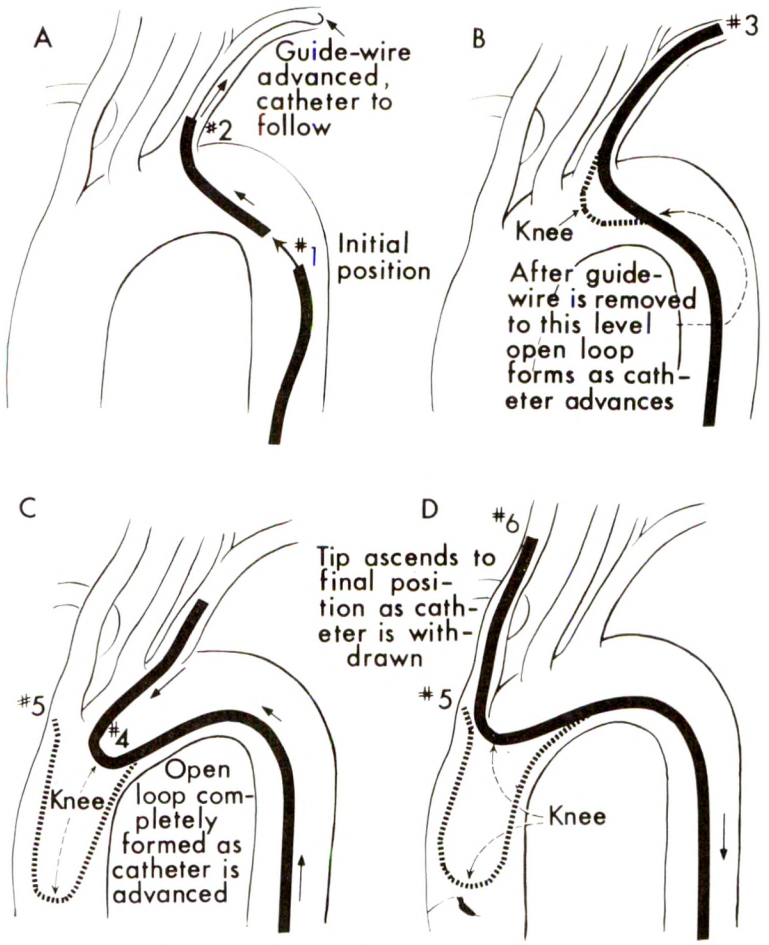


FIG. 4. (*A–D*) Catheter manipulation in a typical transfemoral approach.

catheterization. Infrequently, the open loop is not easily formed in the left subclavian artery, in which instance the catheter loop can be formed by advancing the catheter tip against the aortic valve, the J-guide well in the lead. In the nonelongated arch, the left common carotid or brachiocephalic arteries may also be used to form the open catheter loop. Should very tortuous right subclavian or right carotid arteries resist advancement of the catheter, a 0.025 inch teflon-coated guide-wire can be briefly directed into the desired artery, merely to lead the catheter into the proper orifice.

ORIGIN OF LEFT COMMON CAROTID ARTERY FROM THE BRACHIOCEPHALIC TRUNK

For study of this vascular configuration (Fig. 3*A*), the loop is narrowed by clockwise or counterclockwise rotation of the catheter (Fig. 5*A*, #1), directing the catheter tip toward the origin of the left common carotid artery (#2), which can be entered by slowly withdrawing the catheter (Fig. 5*B*). Reverse slight rotation during withdrawal will facilitate maximal advancement of the catheter.

TRANSAXILLARY CATHETERIZATION

The advantages and hazards of this approach are well defined;[8,20,25] it circumvents troublesome atherosclerotic aorto-iliac changes and substitutes a short catheter course for a long one. Subclavian arteriosclerosis is usually the limiting factor in this approach, but if the artery is not severely stenosed, it can usually be negotiated.

TABLE I

COMPLICATIONS

Patient	Age (yr.)	Diagnosis	Complications
1. F.H.	72	Intermittent visual blurring, bilateral carotid stenosis	Right upper extremity weakness, complete recovery in 4 days
2. R.M.	62	Tinnitus, vertigo, unstable gait, basilar artery occlusion	Paralysis of upward gaze, nearly complete recovery in 3 days
3. M.Y.	70	Drowsiness and lightheadedness, right carotid stenosis	Confusion, beginning 4 hours postangiography, complete recovery in 12 hours
4. W.A.	71	Left homonymous hemianopsia, dizziness and incoordination, left carotid stenosis	Dysphasia with minimal residual left hemiparesis, with complete recovery in 2 days
5. D.M.	82	Bilateral carotid stenosis	Partial expressive aphasia, complete recovery in 24 hours
6. E.T.	60	Advanced diffuse cerebral atherosclerosis	Transient dysphasia with complete recovery

The left axillary approach is preferred because catheter mechanics are similar to the transfemoral approach. Two methods are available for forming the open loop in the aortic arch:

1. *Polyurethane Catheter.* An initial maneuver with a simple curved catheter to aid passage of the J-guide into the descending aorta is usually necessary (Fig. 6*A*). With the guide-wire in the descending aorta, the long open loop catheter is exchanged and catheter advancement started into the descending aorta until the knee of the preformed loop is in the aorta (Fig. 6*B*, #1). The guide-wire is withdrawn and the catheter is further advanced to form the open loop (#2). Maneuvers to complete the procedure are the same as with the transfemoral approach.

2. *Polyethylene Catheter.* A Cook tip deflector may be used. The catheter is advanced over the curved tip in the aortic arch to the descending aorta, thus forming the loop.

COMPLICATIONS

The neurologic complications are summarized in Table I. Two patients had mild residual neurologic deficits persisting for more than 4 days. Four other patients with neurologic complications had complete return of function. There were no deaths.

Except for several small and moderate-sized hematomas at the puncture site, there were no non-neurologic complications.

RESULTS

The technique was successful in 97 per cent of the selected group. As seen from Table II, almost 90 per cent of the patients were over 60 years of age. The 5 failures were in the transfemoral category. Analysis of the cause for failure showed predominantly inability to form the open loop due to aorto-iliac disease and arch changes. This included 1 patient who had both a thoracic and an abdominal aneurysm. Several of the failures occurred early in the survey. With increasing finesse and minor modification

TABLE II

AGE DISTRIBUTION

Age (yr.)	40–49	50–59	60–69	70–79	Over 80
No. of Patients	12	28	82	78	13

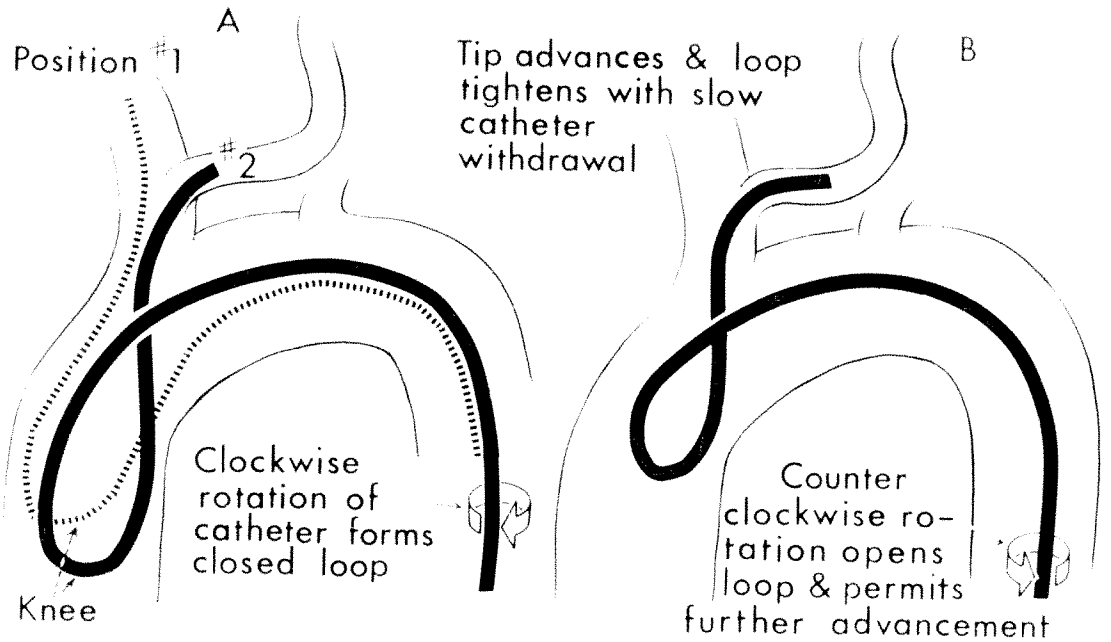


FIG. 5. Transfemoral approach. (*A* and *B*) Maneuvers for catheterization of left carotid artery originating from brachiocephalic trunk.

of the open loop catheter, the failure rate rapidly diminished.

DISCUSSION

The dimensions of the catheter can be individualized. We maintain a stock of 3 loop lengths: 5, 6.5, and 8 cm. The diameter of the loop must be less than the width of the aortic arch. The catheter configuration is usually chosen after visualization of the

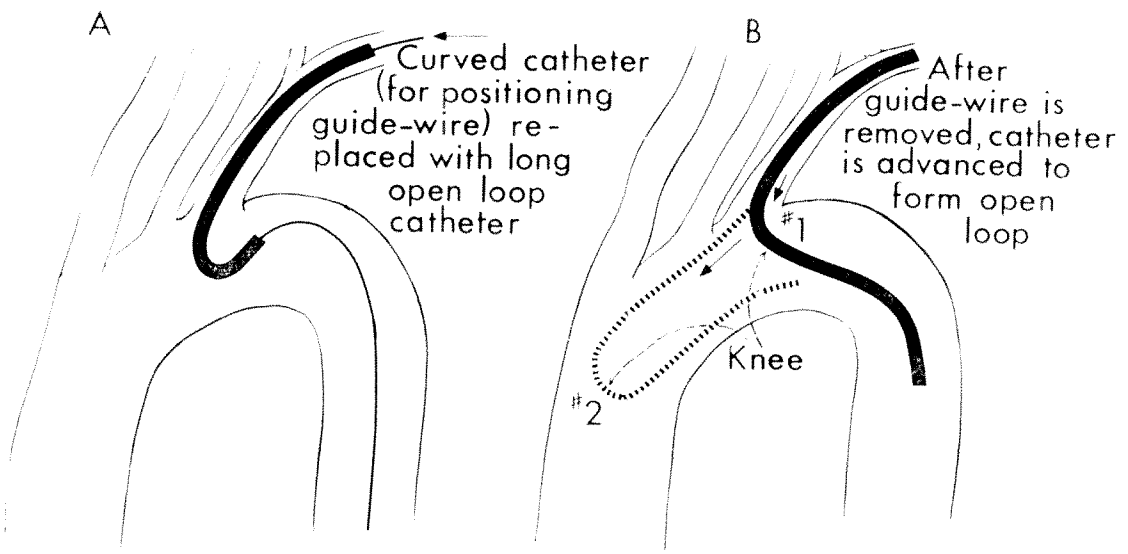


FIG. 6. (*A*) Left axillary approach. Curved catheter for introduction of guide-wire into descending aorta. (*B*) Simple curved catheter exchanged for the selective catheter which is advanced into the descending aorta and arch to reform the loop.

arch anatomy. In cases where origin of the left subclavian artery is remote from the apex of the arch and subtends an acute angle, the length and angle of the catheter tip may be increased to make for easier engagement into the subclavian artery for formation of the open loop. With experience, one soon can categorize the different types of arches, and selection of the catheters is simplified.

If the prescribed standard steps are not followed, attempts to form the open loop directly in the arch may result in formation of a closed loop with a pseudoknot appearance. This is easily overcome by insertion of a guide-wire to straighten the catheter. Knot formation which has occurred with certain other types of catheters[3,7,27] is not a problem.

SUMMARY

A new catheter technique for selective study of the branches of the difficult aortic arch has been used in 213 patients. Designed for the femoral approach, it is also applicable to either axillary approach.

Ninety-seven per cent of attempted selective catheterizations were successful. The complication rate was minimal, reflecting the relatively short procedure time and economy of manipulation.

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THROMBUS FORMATION DURING DIRECT PUNCTURE CEREBRAL ANGIOGRAPHY*

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FOLLOWING the initial report of Nejad *et al.*,[14] in 1968, there has been an increasing awareness of thrombus formation on the surface of angiographic catheters and guide wires.[3,4,8,9,17] The deposition of platelets, fibrin, and red blood cells often occurs within 10 minutes exposure of the angiographic materials to the blood stream which serve as a source of emboli by fragmentation of the thrombus. During withdrawal of the guide wire and catheter, the thrombi are stripped from the surface, accumulate at the site of puncture and may produce local vascular occlusion or emboli. When catheters are exchanged, the thrombus gathered at the puncture site from the first catheter may attach to the second catheter and may be deposited into other vessels, including the coronary and carotid arteries.[12,18]

Previous publications have shown that thrombus formation may be effectively minimized by coating catheters and guide wires with benzalkonium chloride-heparin complex (BH).[2] In addition, the use of systemic heparinization has been shown to decrease clot deposition and reduce thromboembolic complications during angiography.[17] Aspirin, because of its action in diminishing platelet adhesiveness, has also been suggested as an agent for diminishing clot formation.[12]

CLINICAL OBSERVATION

The present investigation was undertaken to determine the presence and extent of thrombus formation in association with direct puncture of the carotid artery for cerebral angiography. To demonstrate the clot, contrast material must opacify the carotid artery immediately surrounding the shaft of the stainless steel needle or teflon sheath. This was accomplished by retrograde puncture and injection of the carotid artery. An irregular radiolucency along the shaft of the needle or teflon sheath indicated the presence of thrombus.

Carotid arteriography was performed in patients following retrograde puncture of the carotid artery with an 18 gauge teflon sheathed needle. As a control and to demonstrate changes due to trauma to the arterial wall, contrast material was injected soon after the teflon sheath was in place in the carotid artery. Just prior to the end of the puncture, *i.e.*, 30 to 60 minutes after puncture, the teflon sheath was partially withdrawn to allow the thrombus to accordion and the vessel was reinjected. The 2 studies were compared to establish the presence of thrombi. This sequence was carried out in 6 carotid arteriograms and radiolucent irregularities along the sheath most prominent at the puncture site suggestive of thrombi were seen in 3 (Fig. 1; and 2).

LABORATORY INVESTIGATION

In order to study these clinical observations in greater detail, similar investigations were undertaken in laboratory animals.

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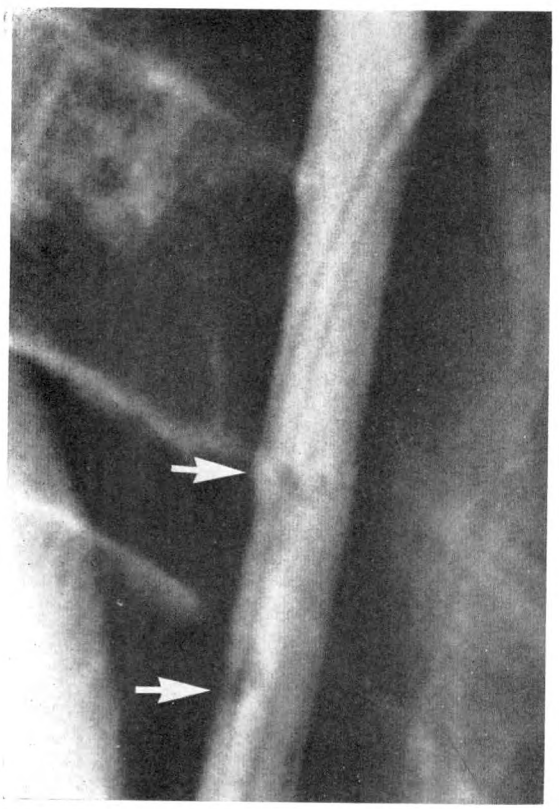


FIG. 1. Thrombus deposition on teflon sheath in human carotid artery; 30 minutes after insertion.

MATERIAL AND METHOD

Eleven mongrel dogs, weighing between 20 and 25 kg. were anesthetized with intravenous injection of nembutal (30 mg./kg. body weight). An endotracheal tube was passed orally and the animals were allowed to breathe spontaneously.

Group 1. In 3 animals the right carotid artery was punctured by an 18 gauge teflon sheath needle while an 18 gauge stainless steel needle was inserted into the left carotid artery. Catheterization was then performed *via* the right femoral artery using a No. 7 French polyethylene catheter. Selective carotid arteriography and aortography were then done at varying intervals up to 120 minutes.

Group 2. In the other 8 dogs a midline incision was made in the neck from the thyroid cartilage to within 2 cm. of the suprasternal notch. The carotid arteries and jugular veins were isolated. Eighteen and 20 gauge stainless steel and teflon sheathed needles were the materials tested for their thrombogenic properties. The needles or sheaths were inserted into the artery in the direction of blood flow and exposed to the blood stream for varying periods of time up to 90 minutes. A 21 gauge "butterfly" needle was inserted into the vessel proximal to the point of entry of the test materials. Periodically during the experiment, an occlusal film was placed under the dissected vessel and the vessel was temporarily occluded by a "bulldog" clamp proximal to the butterfly needle. Ten milliliters of contrast material was injected slowly through the butterfly needle while the roentgenogram was exposed. The period of time of occlusion of the vessels was less than 1 minute. The "bulldog" clamp was then released, re-establishing blood flow. This technique resulted in high resolution roentgenograms of thrombus formation at intervals throughout the time

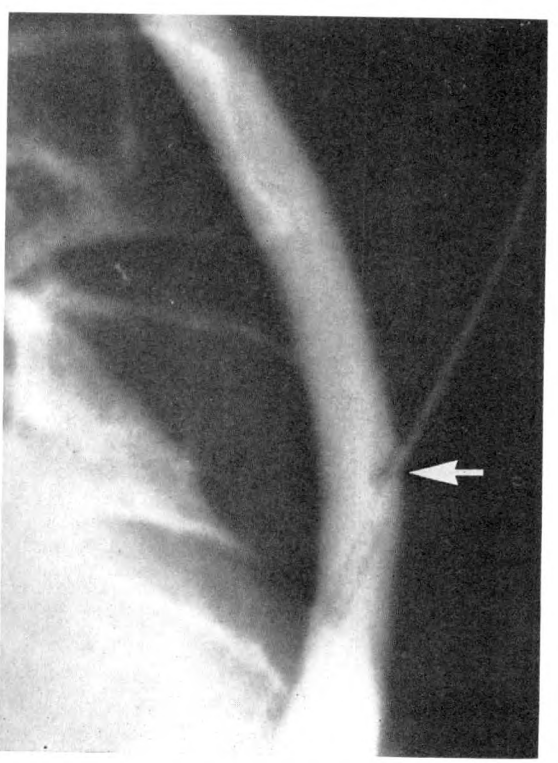


FIG. 2. Thrombus gathered at site of puncture of human carotid artery after partial withdrawal of plastic tubing 30 minutes after insertion.

the test materials were exposed to the blood stream. Similar experiments were performed with the jugular veins.

At the end of the test period, the portions of the vessels containing the test materials were excised, opened, and placed in 2 per cent cacodylic acid buffered glutaraldehyde (pH 7.4) and preserved for photographic recording.

Thrombus formation on the needle and teflon sheath was studied under the following experimental conditions:

(1) Without anticoagulant protection
(2) With anticoagulant protection
  (a) coating the test materials with benzalkonium chloride-heparin compound (BH)
  (b) administration of systemic heparinization.

Systemic heparinization consisted of an initial intravascular injection of 45 units of aqueous heparin per kg. of body weight followed by intravascular infusions of 3–5 cc. of a solution containing 1,000 units of heparin in 500 cc. of normal saline. The infusions were given every 2 to 3 minutes throughout the experimental period.

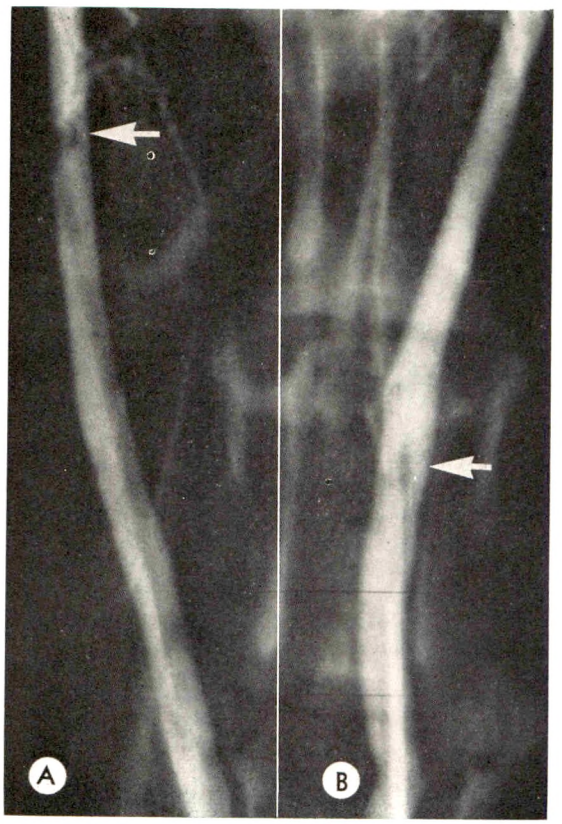


Fig. 3. Thrombus adhering to (*A*) teflon sheath and (*B*) steel needle in the carotid arteries of dogs 90 minutes after insertion.

RESULTS

Roentgenographic evidence of thrombus formation appeared as a zone of radiolucency along the outer surface of the test material. In view of the radiolucency of the teflon sheath, an additional thickened and irregular zone was considered to be thrombus deposition.

The findings in these experiments must be evaluated with the knowledge that the activated clotting time for dogs' blood is approximately one-half that of humans.

In Group I, thrombus formation did not become recognizable on the test materials until after 45 minutes of exposure to the blood stream because of the masking effect of the cervical musculature (Fig. 3, *A* and *B*). In Group II the higher roentgenographic resolution obtained by placing the film in direct contact with the vessel allowed the demonstration of clot deposition within 15 minutes of the puncture (Fig. 4; and 5, *A* and *B*). This early clot deposition was confirmed by visual inspection of the excised vessel (Fig. 6). Thrombus formation in each group increased with longer exposure to the blood stream.

In many instances, the initial observable thrombus was found at the point where the needle or tubing abutted against the intimal surface (Fig. 7). When this needle or tubing was partially withdrawn from the vessel, the thrombus sleeve was stripped from the surface, accumulating at the site of puncture. In one instance, a thrombus was seen at the tip of the needle in the carotid artery but was not observed 5 minutes later, indicating its detachment and embolization.

Clot deposition occurred earlier and to a greater extent on test materials in the jug-

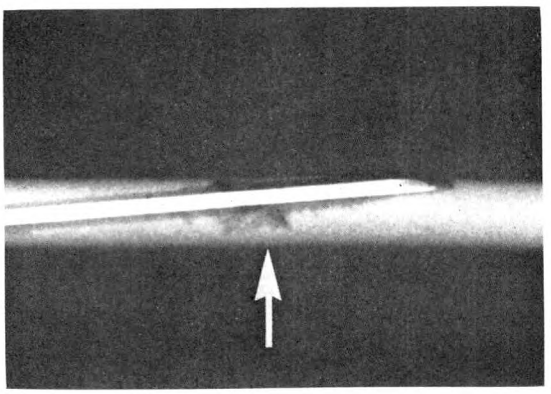


FIG. 4. Clots on teflon sleeve needle 15 minutes after insertion.

ular vein when compared to those in the carotid artery. No significant difference was noted in the relative thrombogenicity of stainless steel and teflon.

The use of anticoagulation by systemic heparinization or coating the materials with the BH prior to insertion minimized or eliminated the clot formation (Fig. 8, *A–C*). Systemic heparinization provided sufficient anticoagulant protection as long as the coagulation time was prolonged to 1.5 to 3 times normal. The BH coated materials remained free of thrombi even after

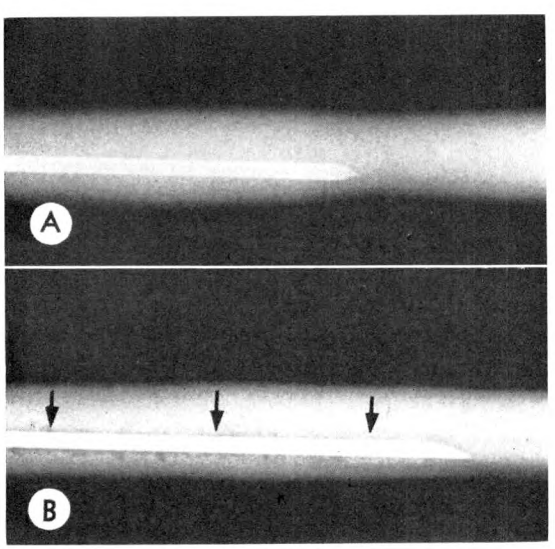


FIG. 5. (*A*) Steel needle soon after insertion in carotid artery of a dog. (*B*) Same needle 30 minutes later. Note thrombus deposition evenly distributed along shaft of needle.

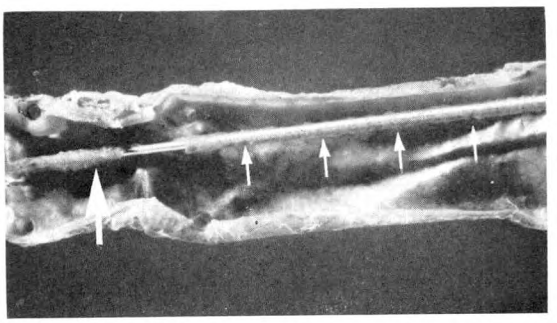


FIG. 6. Light micrograph of same artery as shown in Figure 5*B* after excision. Note the fibrin sleeve along the shaft of the needle.

6 hours of exposure to the arterial blood stream.

DISCUSSION

Although reports of clinically significant emboli during cerebral angiography are few, this complication is of vital importance.[1,6,13,20] The findings of this investigation suggest that emboli are frequent even though not clinically obvious; the brain must adequately handle most emboli.[10,15,18]

Undoubtedly, the speed and technical proficiency of the physician performing the procedure are primary factors in minimizing complications. However, our studies show that thrombi will form on all needles and catheters presently utilized for angiography. Until nonthrombogenic materials are available, the protection by anticoagulant measures should be considered as long as this protection can be obtained without

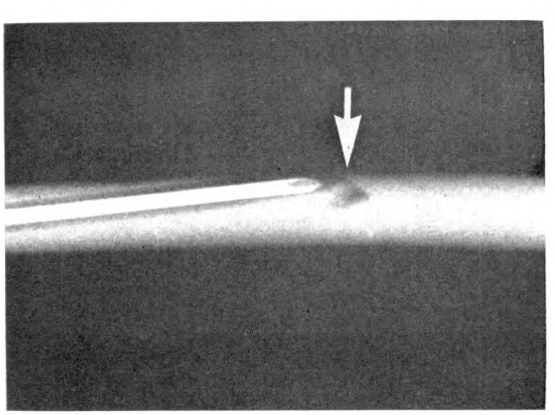


FIG. 7. Teflon sleeve needle with clot at its tip 15 minutes after insertion in carotid artery of a dog.

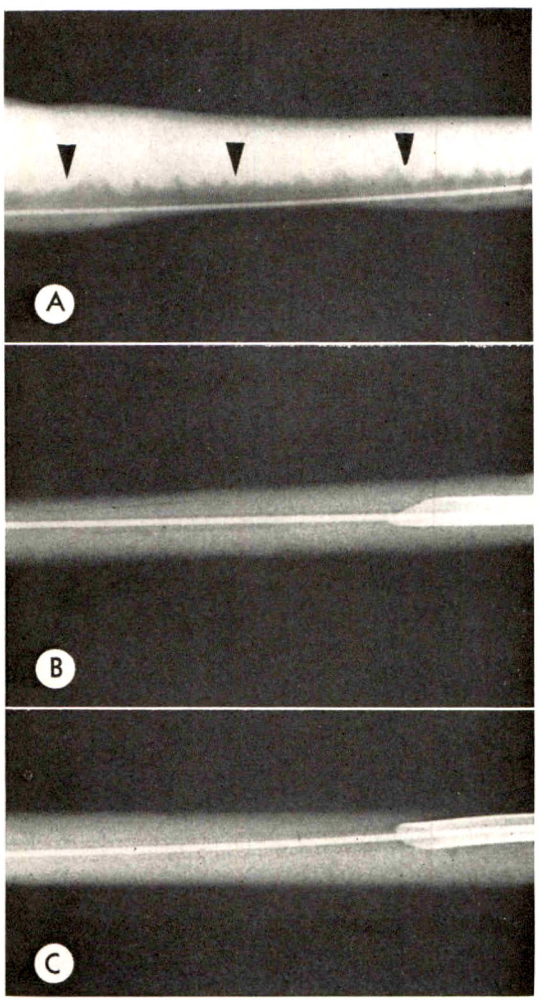


Fig. 8. (*A*) Plastic tubing 75 minutes after insertion in the jugular vein of a dog. Note transparent clot adhering. (*B*) Similar tubing protected by systemic heparinization 75 minutes after insertion. (*C*) Similar tubing coated with BH compound 75 minutes after insertion.

increasing the risk of bleeding in patients with normal clotting mechanisms.

In over 1,500 cases of angiography performed (primarily *via* the femoral arterial route) with systemic heparinization, there was no significant incidence of increased bleeding at the site of puncture.[19] Excessive bleeding has not been reported as a significant complication with the use of BH coating.[7] Anticoagulant measures have not been employed in patients with active bleeding.

SUMMARY

Thrombus formation occurs frequently on stainless steel and teflon sheath needles utilized for direct puncture cerebral angiography.

Although embolization is not a frequently reported complication of carotid arteriography, measures for minimizing thrombus formation are suggested.

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ANGIOGRAPHY IN POST-TRAUMATIC PRIAPISM*

A CASE REPORT

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PRIAPISM secondary to trauma is an uncommon occurrence. Thrombosis or hemorrhage causing obstruction or compression of the venous drainage of the corpora cavernosa are the most common pathogenetic events. Priapism may also result from aorto-vena caval shunt due to trauma,[1] surgery, or spontaneous aneurysmal rupture. Burt *et al.*[3] reported aspirating a large volume of bright red blood from the corpora cavernosa in one patient with idiopathic priapism. In this case, the etiology was felt to be excess arterial flow, and priapism disappeared following right internal pudendal artery ligation.

Arteriovenous fistulae have been reported in all areas of the body.[2,4,5] However, to our knowledge, angiographic demonstration of a local arteriovenous fistula to the corpora cavernosa causing priapism has not yet been reported.

REPORT OF A CASE

A 25 year old male tripped and fell after which he was immediately struck in the pubic area by a loaded shopping cart. The following morning he awakened with a persistent erection. He was placed on fibrinolytic enzymes and bed rest by his physician with no relief of his priapism.

Seven days after his initial injury, he was admitted to Loma Linda University Medical Center with priapism. Physical examination showed a large, firm, erect penis which was pink in color and warm to the touch. There was no cyanosis. The remainder of the physical examination and laboratory studies were within normal limits.

The patient was taken to surgery where aspiration of the corpora cavernosa resulted in copious flow of bright red blood. There was no reduction in size of the penis following the aspiration of over 100 cc. of blood. It was decided not to proceed with the contemplated cavernovenous shunt. After review of the case, an arteriovenous fistula was considered the most likely possibility, and angiography was requested.

A right femoral percutaneous catheterization was done. A preformed Kifa catheter was placed selectively in each hypogastric artery. Contrast material pooled abnormally in the corpora cavernosa from terminal branches of the right internal pudendal artery (Fig. 1) and the left injection showed a normal arterial pattern extending into the proximal portion of the body of the penis, without extravasation of contrast agent.

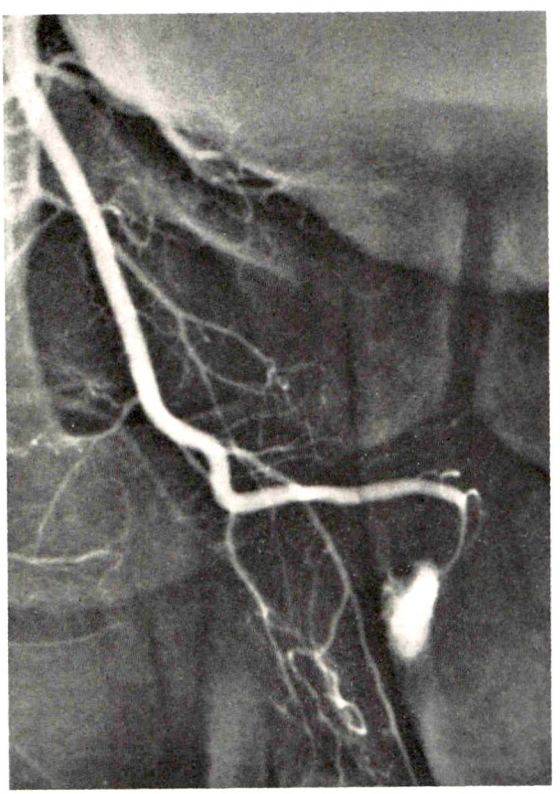


FIG. 1. Right internal iliac angiogram showing laking of contrast material near the termination of the right internal pudendal artery.

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Surgical ligation of the terminal branches of the right pudendal artery was performed. The penis gradually decreased in size and was normal at the time of discharge 8 days later. The patient has had no recurrence of priapism and has had no erections since surgery.

DISCUSSION

Angiography is a most useful method of diagnosis of vascular abnormalities. Priapism can easily be studied angiographically and this procedure should be particularly useful in those cases in which trauma or other vascular abnormality is the suspected etiology. Selective catheterization of the internal iliac arteries offers visualization of the arterial supply of the penis. Selectivity is facilitated by catheter insertion from the femoral artery opposite the pudendal artery of interest.

In retrospect, it might have been beneficial to try infusion of a vasoconstrictive agent *via* the arterial catheter.

SUMMARY

A case of traumatic arteriovenous fistula involving the internal pudendal artery and corpora cavernosa causing priapism is reported.

The fistula was demonstrated by percutaneous selective angiography.

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A SINGLE PLANE CHANGER SPECIAL PROCEDURE ROOM WITH THE AID OF A SIMPLE TURNING DEVICE*

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CORONARY artery atherosclerosis accounts for more than 50 per cent of the deaths in the United States each year, and indications are that its incidence is increasing. It is now estimated that approximately 25 million Americans have coronary artery disease.[5]

With the recent advances in the surgical treatment of coronary artery disease utilizing the saphenous vein bypass graft, an increasing number of patients with suspected coronary artery disease are being evaluated for possible operative intervention. Since coronary angiography is considered essential in the evaluation of every patient suspected of coronary insufficiency and is helpful in evaluating postoperative results,[2] the number of studies performed is ever increasing. Within the past year the number of patients undergoing coronary arteriography at the University of Minnesota Hospitals has doubled. Therefore, a more efficient means of studying patients must be devised.

The purpose of this communication is to present various modifications of existing equipment and describe a simple patient turning device that expedites coronary angiography.

Figure 1 depicts the appearance of our angiographic room prior to modification. Note the cluttered appearance with numerous obstacles impeding easy access to the equipment as well as the patient.

Figure 2 shows the room after modification. The biplane Schoenander film changers have been replaced by a single plane see-through changer,[1,3] thus eliminating one film changer, one generator, and 2 x-ray tubes. The pressure transducers, power injector, and electrocardiographic leads have been permanently mounted to the table. All electrical cables for this equipment are placed beneath the floor. The oscilloscopes for pressure and electrocardiographic display are mounted to the wall of the angiographic room for ease of viewing. The pressure recording equipment has been placed behind the control panel allowing easy access to the equipment and better protection for the recording technician. The small pressure injector has been mounted to the roentgenographic table. A simple patient turning device has been constructed that can be added to any angiographic table and has proved useful for various procedures, particularly coronary angiography.

Figure 3 shows the construction principle of the turning device. For his comfort, the patient rests on a 1 inch thick Mylar covered polyurethane mattress. A motor (A) rotates the 2 shafts (B) which are permanently mounted alongside the edges of the table. A Mylar belt (D) is firmly attached to the drive shafts (B), thus allowing both transport across the table top and rotation of the patient. In order to prevent slippage of the patient, radiolucent friction strips (E) are cemented to the transport belt. The apparatus can be foot operated, thus leaving both hands free for patient manipulation. To facilitate rotation and immobilization of the patient in various oblique positions, the use of a cloth harness proved very helpful (Fig. 4). Straps (S) allow rotation of the patient and immobilization through table attached

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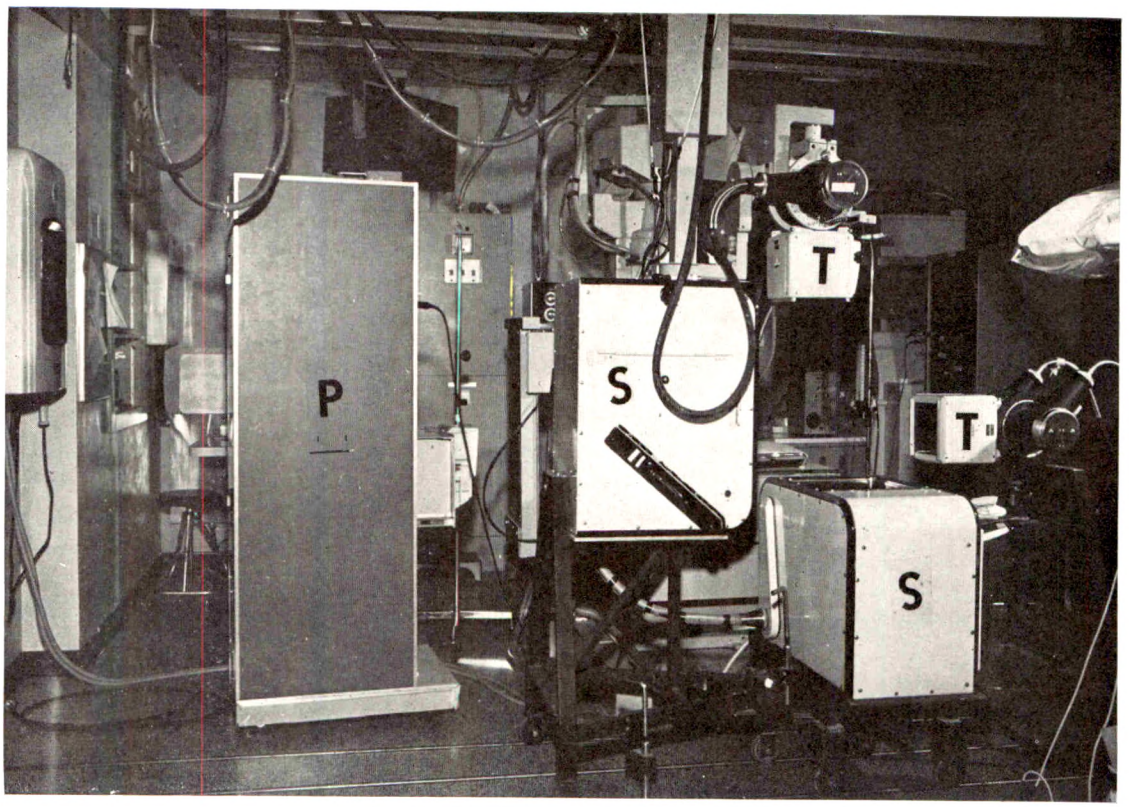


FIG. 1. Special procedure Room A with biplane Schoenander film changers (S), physiologic recording equipment (P), and 3 roentgenographic tubes (T) prior to modification. "Birdnest design" makes angiographic procedures cumbersome. (Pressure injector not shown.)

pulleys in various oblique positions. Cloth loops (L) allow insertion of a plexiglass turning rod facilitating rotation.

In order to prevent shifting of the drape in relation to the patient, a sterile stainless steel tube is threaded over the acrylic rod which has been inserted into the loops of flap (L). This can be accomplished under sterile conditions and provides sterile dressing in any position (Fig. 5, *A* and *B*).

The device allows rapid positioning and immobilization of the patient in the lateral (Fig. 6), left posterior oblique (Fig. 7), right or left anterior oblique positions (Fig. 8).

DISCUSSION

In general, the simplest way to rotate a conscious patient is to ask him to "roll up on his side" as is common for gastrointestinal roentgenography and coronary arteriography on standard roentgenographic tables. With slight assistance by the radiologist, this is easily accomplished but the patient is no longer centered in the fluoroscopic beam. The patient has to be asked to "slide towards the examiner" in order to bring the examination field into the central beam. Turning of patients by this simple technique may be tiresome for both patients and radiologists, particularly in the heavily sedated patients undergoing angiography. The patient can usually roll up on his side with some manual assistance, but the command "slide towards me" commonly cannot be carried out. Consequently, the patient together with the mattress must be moved into the central beam. This procedure complicates angiography. Therefore, various mechanical means of rotating patients have been advocated.

The most popular turning devices are

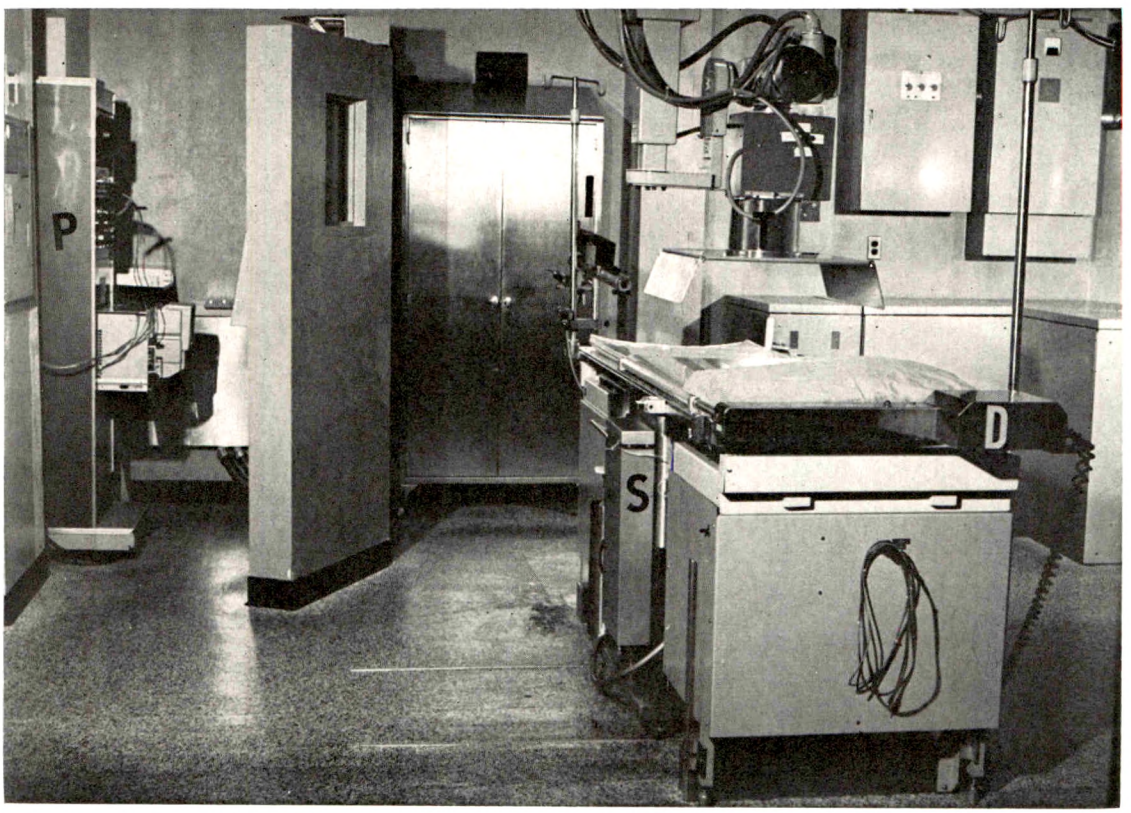


Fig. 2. Biplane film changers replaced by see-through changer (S). Anteroposterior, lateral, and fluoroscopic tube replaced by single overhead tube (T). Portable pressure injector (I) permanently mounted on roentgenographic table. Physiologic recording device (P) mounted behind roentgen-ray control booth. Patient turning device (D) eliminates biplane changers and tubes.

cradles in which the patient is strapped into a radiolucent cradle which is turned by a motor. This approach has several drawbacks, particularly in coronary roentgenography: (1) The fairly heavy device has to be fastened to the roentgenographic table and must be removed if the table is used for other procedures not requiring rotation of the patient. (2) Placing the sedated patient into the cradle is cumbersome. (3) The patient is uncomfortably immobilized and strapped down for a long period of time, particularly if the catheterization procedure is complicated. (4) Many cradles do not allow rotation into 90° position, a view that is very important for coronary arteriography. (5) Additional filtration of the roentgen-ray beam is unavoidable with a cradle. (6) With rotation the patient is moved away from the film changer, increasing target-film distance and distortion. These factors are increased with heavily padded cradles. (7) The edges of the cradle cast a shadow on the roentgenogram which is visible even with low density beveled aluminum or plastic materials. (8) The patient cannot be overrotated, thus bringing the heart close to the roentgenographic film in undertable roentgenography.

The simple patient turning device as described has the following advantages over all previously constructed devices: (1) It can be attached to any roentgenographic table. (2) It does not have to be removed and can remain permanently in place. (3) Placing the patient on the device is as simple as placing him on a standard table. (4) Added filtration is eliminated, and visualization of the cradle edges is

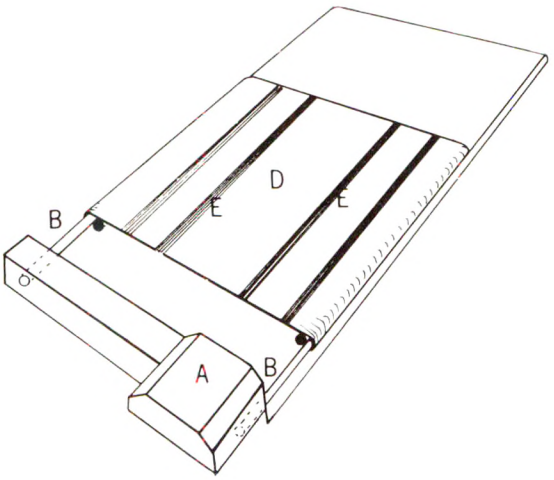


FIG. 3. Construction principle of the turning device. (Available from Nedmac, Inc., Minneapolis, Minnesota.)

eliminated. (5) Object-film distance is not increased. (6) Rotation is simply accomplished. (7) Rotation in 90° positions in both directions is feasible. (8) Rotation beyond 90° is feasible, thus bringing the heart close to the roentgenographic film eliminating a lateral film changer (Fig. 8).

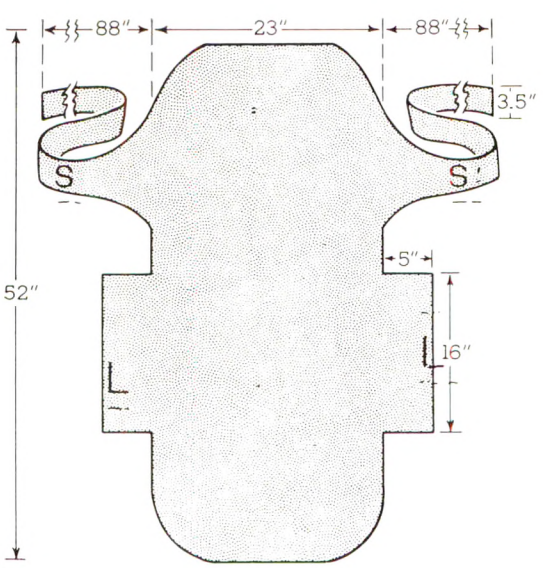


FIG. 4. Cloth harness allowing rotation and immobilization of the patient in various positions. Rotation is facilitated by straps (S) and flaps (L) containing plexiglass rods for rotation.

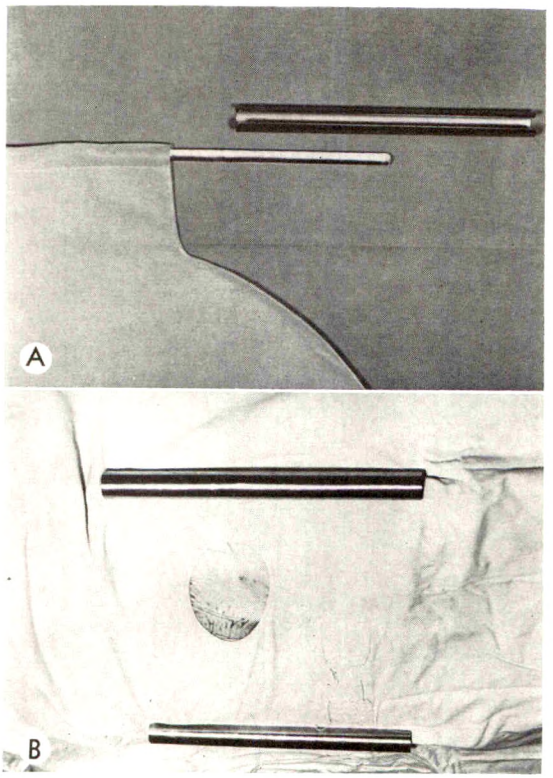


FIG. 5. (*A*) The acrylic rod inserts into the loops of the cloth harness allowing the slotted sterile stainless steel tube to be placed over the rod and drape. (*B*) Patient's groin with drape in place. Stainless steel tubes serve as handles to help rotate the patient and eliminate shifting of drape.

(9) The apparatus is equally useful for undertable roentgenography as for cine roentgenography. (10) Activation by a foot switch leaves both hands free for catheter



FIG. 6. Patient in the 90° lateral position.

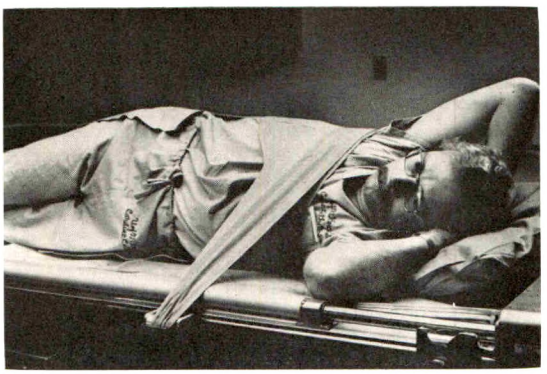


Fig. 7. Patient in the left posterior oblique position.



Fig. 8. Patient in the left anterior oblique position. (Right anterior oblique position not shown.)

manipulation. (11) The device should be useful not only for all types of single plane angiocardiography, but also for gastrointestinal roentgenography.

CONCLUSION

Various ways of expediting coronary angiography are shown.

The modification of existing equipment is easily made and improves the functional arrangement of an angiographic room.

The turning device is readily adapted to any roentgenographic table and is useful for all types of vascular roentgenography as well as gastrointestinal roentgenology.

The use of a see-through changer expedites all types of angiography, particularly coronary roentgenography.

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PHYSICAL CHARACTERISTICS OF MODERN MICROFOCUS X-RAY TUBES*

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IN view of the growing popularity of direct magnification techniques using commercially available microfocus x-ray tubes, we recently undertook a comparative study of the physical characteristics of several of these tubes made by different manufacturers.

The following is a report of the findings of this study. A critical, theoretical and practical analysis of the value of radiographic magnification itself and its limitations has recently been published elsewhere.[4]

TUBE DESIGN

Four x-ray tubes, each one made by a different manufacturer, were included in this study. Their pertinent design characteristics are shown in Table 1. According to the manufacturer's statement, each of them had a nominal focal spot size of 0.3 mm. ×0.3 mm. As can be seen from Table 1, the design characteristics of all the 4 tubes tested were more or less similar. As the power ratings are more or less the same, the maximum permissible exposure time at a given KVP and mA is almost the same for all the tubes. The target angle of Tubes 3 and 4 is slightly larger so that the diameter of the useful x-ray beam at a given distance will be slightly larger also.

PIN-HOLE IMAGES

Pin-hole images of the focal spot at various values of KVP and mA were made using a 30 micron pin-hole made by Nuclear

TABLE I

DESIGN CHARACTERISTICS OF MICROFOCUS X-RAY TUBES

	Tube 1 (manuf. 1)	Tube 2 (manuf. 2)	Tube 3 (manuf. 3)	Tube 4 (manuf. 4)
Stated nominal size of focal spot	0.3 mm.	0.3 mm.	0.3 mm.	0.3 mm.
Minimum size measured	0.34 mm.	0.31 mm.	0.34 mm.	0.34 mm.
Maximum size measured	0.80 mm.	0.52 mm.	1.00 mm.	0.75 mm.
Rotor speed	10,000 RPM	10,000 RPM	10,000 RPM	10,000 RPM
Stator frequency	180°	180°	180°	180°
Generator used	3-phase 12 Pls.	3-phase 12 Pls.	3-phase 12 Pls.	3-phase 12 Pls.
Power rating at 1/10 sec.	12.8 KW	12 KW	12.5 KW	12.5 KW
Maximum permissible exposure at 100 KVP, 100 mA	1 sec.	1 sec.	1.3 sec.	1.3 sec.
Target angle	10°	10°	11°	11°
Diameter of anode disk	100 mm.	100 mm.	100 mm.	100 mm.

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Associates. A large magnification factor (approximately 10) was selected to demonstrate clearly the intensity distribution across the length and breadth of the focal spot. Typical images, obtained with one of the tubes tested, are shown in Figure 1. Notice that the size and shape change drastically with tube current. At the lower tube current, the focal spot may be seen to be smaller and the intensity distribution more uniform. At the larger tube currents, the temperature of the filament goes up and the space charge distribution is substantially altered. This results in a larger focal spot and, in addition, the emission of x rays is confined essentially to 2 narrow edge bands. Figure 1 also shows that the variation with kilovoltage is not as great as with tube current. In this case, the size does not seem to change substantially; only the shape or the intensity distribution is somewhat different. These features were common to all the 4 tubes tested. Another general finding was that the focal spot was often rectangular in shape, indicating that its resolution capability is different in different directions across the film plane.

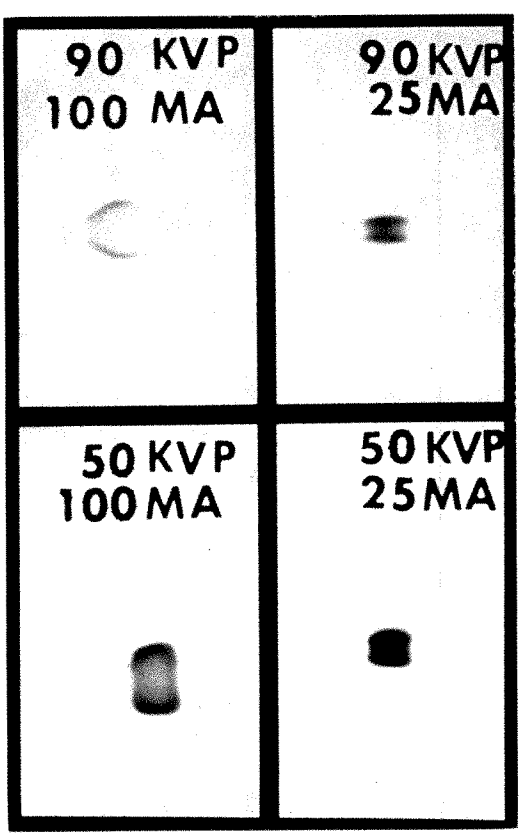


FIG. 1. Pin-hole images of the focal spot of a typical microfocus x-ray tube. Notice the drastic change in size and shape with tube current.

While pin-hole images do provide an impressive visual demonstration of the shape of x-ray focal spots, they are not adequate for quantitative measurements. This is because, as shown in Figure 2, the resolution capability or the Modulation Transfer Function (MTF) of an x-ray focal spot is a complex function of its size, shape and the intensity distribution. For example, it can be shown using the data in Figure 2 that with a homogeneous square focal spot of a given size, the frequency at which the MTF goes to zero is exactly twice as great as when the x-ray emission is confined to 2 infinitely narrow bands at the edges. Another difficulty with pin-hole images is that they are usually blurred and unsharp at the edges, thus making the measurements difficult and arbitrary. Furthermore, the estimated dimensions are also influenced by the average density of the pin-hole image. The darker the image, the larger will be the estimated size. It is precisely in view of these uncertainties that the International Commission on Radiological Units and Measurement (ICRU) has established a number of arbitrary standards under which pin-hole images should be measured.[1]

Apart from these drawbacks, the use of the pin-hole method is highly impractical for routine work. To get a sharp image with a 0.3 mm. focal spot, the pin-hole has to be of the order of 30 microns or so in diameter. A minute pin-hole such as this allows very little radiation to pass through. Furthermore, the film has to be placed sufficiently away from the pin-hole so that the image will be magnified at least 5 times. Otherwise the measurement becomes very difficult. Intensifying screens cannot be used as these will tend to further blur the image. The net result of all these stringent require-

SHAPE OF LSF	Line Spread Function $L(\xi)$ (NORMALIZED AT $\xi = 0$)	Modulation Transfer Function $M(f)$ (NORMALIZED AT $f = 0$)
IMPULSE	$L(\xi) = 1$ at $\xi = 0$ $L(\xi) = 0$ elsewhere	1
GAUSSIAN	$e^{-K^2\xi^2}$	$e^{-\frac{\pi^2 f^2}{K^2}}$
EXPONENTIAL	$e^{-\lvert K\rvert \xi}$	$\frac{1}{1 + 4\pi^2 f^2/K^2}$
LORENTZIAN	$\frac{1}{1 + K^2\xi^2}$	$e^{-2\pi f/K}$
HOMOGENEOUS SQUARE	$L(\xi) = 1$ from $\xi = -\frac{a}{2}$ to $+\frac{a}{2}$ $L(\xi) = 0$ elsewhere	$\frac{\sin \pi a f}{\pi a f}$
STEPLADDER	$L(\xi) = 1$ from $-a/2$ to $+a/2$ $L(\xi) = K$ from $\frac{-(a+r)}{2}$ to $-a/2$ and from $a/2$ to $\frac{(a+r)}{2}$ $L(\xi) = 0$ elsewhere	$\frac{K \sin \pi f(a+r) + (1-K)\sin \pi a f}{(a+Kr)\pi f}$
TRIANGULAR	$L(\xi) = (1 + \frac{2\xi}{a})$ from $\xi = \frac{-a}{2}$ to 0 $L(\xi) = (1 - \frac{2\xi}{a})$ from $\xi = 0$ to $a/2$ $L(\xi) = 0$ elsewhere	$\left[\frac{\sin \frac{\pi a f}{2}}{\frac{\pi a f}{2}}\right]^2$
TWO EDGE BANDS	$L(\xi) = 1$ from $\frac{-a}{2}$ to $\frac{-p}{2}$ and from $\frac{+p}{2}$ to $\frac{+a}{2}$ $L(\xi) = 0$ elsewhere	$\frac{\sin \frac{\pi f(a-p)}{2} \cos \frac{\pi f(a+p)}{2}}{\pi f(a-p)/2}$
TWO INFINITELY NARROW BANDS	$L(\xi) = 1$ at $\xi = -a/2$ and at $\xi = +a/2$ $L(\xi) = 0$ elsewhere	$\cos(\pi a f)$
FOUR EDGE BANDS	$L(\xi) = 1$ from $-a/2$ to $-r/2$ from $-q/2$ to $-p/2$ from $+p/2$ to $+q/2$ from $+r/2$ to $+a/2$ $L(\xi) = 0$ elsewhere	$\frac{\sin \pi f q - \sin \pi f p + \sin \pi f a - \sin \pi f r}{\pi f(q - p + a - r)}$

FIG. 2. Analytic expressions for the Modulation Transfer Function (MTF) of x-ray focal spots of various configurations.

ments is that the exposure time required in making a good pin-hole image of a 0.3 mm. focal spot turns out to be extremely long, being of the order of half a minute or so. As such long exposures are far beyond the rating characteristics of these tubes, one often resorts to a number of multiple exposures of much shorter duration and allows enough time for the target to cool off between exposures. The procedure is thus extremely tedious and time consuming.

MEASUREMENT OF FOCAL SPOT SIZE BY RESOLUTION METHOD

In view of the foregoing difficulties, many investigators[2,5,7,8] are now turning to the

so-called resolution method of measuring focal spot size. The theoretical basis for this method has been discussed at length by one of us in a previous publication.[5] In its practical aspect, it simply consists in making a radiographic image using a non-screen film of a test object of the type shown in Figure 3. The test object is placed at some intermediate position between the x-ray tube and the film. Its exact distance from the film is not important. A careful inspection of the processed film will reveal that the patterns in the test object become progressively blurred up to a certain point and thereafter reappear with a reversal of the dark and the bright lines (Fig. 3). Knowing m, the magnification factor and f, the number of line pairs per mm. in that pattern in which the contrast is minimal, the focal spot size is calculated using the equation

$$a = \frac{m}{(m-1)f}. \qquad (1)$$

The value of m can be determined simply by dividing the image length of the entire test object by its actual length (Fig. 3).

The measured focal spot size corresponds to the direction in which the test object is held with respect to the anode-cathode axis of the x-ray tube. In our laboratory we routinely make two measurements, one parallel to the A-C axis and the other perpendicular to the A-C axis.

The main attraction of the resolution method is that it is based on the observance of a type of "null" effect rather than direct measurement. For this reason, errors inherent in the traditional pin-hole method are largely avoided. The numerical value of the focal spot size as estimated by this method does not correspond to the physical size alone. Rather, it gives us that size of a homogeneously irradiating focal spot that has the same resolution capability as the one under measurement. For this reason it is appropriate to call it the Equivalent Focal spot size.[3,6]

Some investigators prefer the use of a bar pattern with continuously varying spatial frequencies. Still others prefer the use of a star pattern. While conceptually it does not matter which of these is used, we feel that the use of discrete bar patterns is the most convenient from a practical point of view, because it eliminates the need for any precise measurements on the image. Also, with the star pattern one places the center of the star along the central axis and hence the disappearance will always occur at a

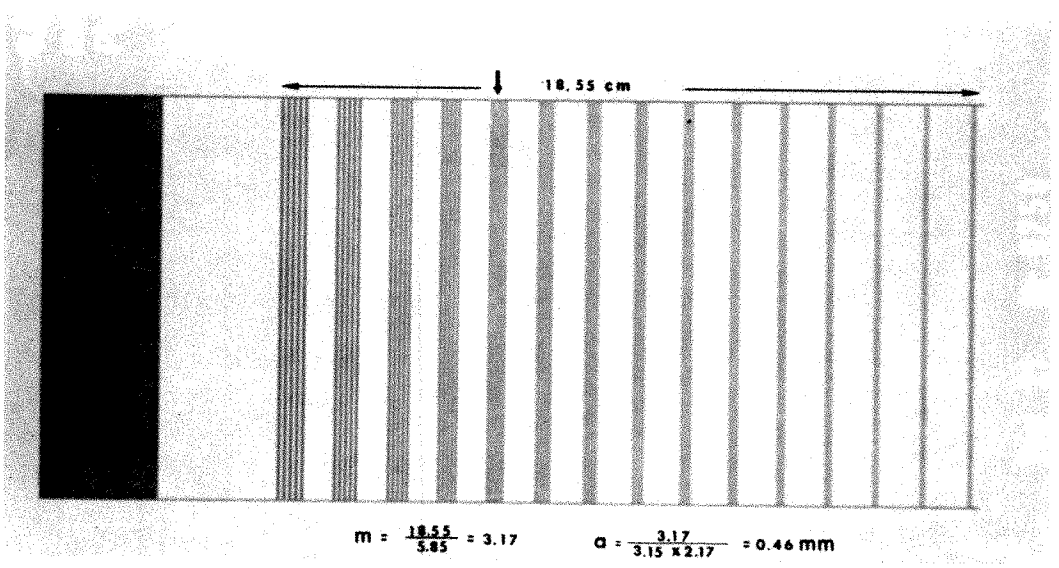


FIG. 3. Test pattern used for the measurement of focal spot size by the resolution method.

TABLE II

MEASURED FOCAL SPOT SIZE OF FOUR COMMERCIALLY AVAILABLE X-RAY TUBES

(nominal size 0.3 mm.×0.3 mm.)

	Parallel to A-C Axis			Perpendicular to A-C Axis		
	50 KVP	90 KVP	120 KVP	50 KVP	90 KVP	120 KVP
Tube 1						
25 mA	0.36	0.35	0.34	0.39	0.36	0.36
50 mA	0.40	0.40	0.40	0.62	0.50	0.44
100 mA	0.51	0.51	0.45	0.80	0.71	0.71
Tube 2						
15 mA	0.35	0.34	0.33	0.36	0.36	0.36
50 mA	0.34	0.33	0.32	0.46	0.41	0.41
100 mA	0.33	0.32	0.31	0.52	0.41	0.41
Tube 3						
25 mA	0.36	0.35	0.34	0.50	0.49	0.49
50 mA	0.37	0.36	0.34	0.62	0.51	0.50
100 mA	0.37	0.37	0.34	0.83	0.54	0.52
150 mA	0.39	0.37	*	1.00	0.63	*
Tube 4						
25 mA	0.38	0.36	0.34	0.38	0.36	0.34
50 mA	0.43	0.41	0.38	0.48	0.44	0.40
100 mA	0.47	0.43	*	0.67	0.50	*
150 mA	0.51	0.46	*	0.75	0.65	*

* Tube rating did not permit measurement.

point away from the central axis. Therefore, one does not really measure the focal spot size along the central axis with this method. If the star is moved to get a better estimate of the focal spot size along the central axis, the symmetry of the arrangement will be destroyed.

Table II shows the measured values of the focal spot size of the 4 tubes under test, using the discrete bar pattern method described above. All the tubes tested obviously have focal spots whose dimensions are substantially larger than the nominal size quoted by the manufacturers. Of all the tubes tested, Tube 2 was found to be the closest to the stated dimensions. Tube 2 is superior also in the sense that it has a focal spot which is more nearly a square than the others. Parallel to the A-C axis, the equivalent focal spot size for this tube did not change appreciably with KVP or mA. Perpendicular to the A-C axis, there is some change with KVP and mA, but even here the variation is much less than in the case of the other tubes. The remaining tubes were all more or less equally unsatisfactory.

DETERMINATION OF EQUIVALENT FOCAL SPOT SIZE WHEN THERE IS NO CROSS OVER

Occasionally, the disappearance phenomenon cannot be observed. This will be the case if the intensity distribution from the focal spot happens to be close to a Gaussian or exponential rather than to a homogeneous square or to 2 edge bands. In the present study we encountered this difficulty with only one tube and that too only in a direction parallel to the A-C axis. In

this case we initially tried to look for the highest number of line pairs per mm. at which the pattern was still visible and use Equation 1 with this number for f. However, this method was soon found to yield erroneous results. For one thing, the estimated size turned out to be of the order of 0.10 mm., almost a third of the nominal size of 0.3 mm. Secondly, when the films were exposed at various levels of magnification, the answers were all different. In order to investigate the reasons for this anomaly, all the patterns in the image were scanned by a microdensitometer and the entire square wave response curve plotted. This is shown in Figure 4. The dotted curve in the same Figure shows the theoretical square wave response or a 0.2 mm. homogeneous focal spot. The procedure used in obtaining this theoretical curve has been described elsewhere.[5] Notice that while the theoretical curve is seen to fall sharply to zero, the experimental curve shows a rather asymptotic fall-off. This suggested that a more realistic value for the equivalent focal spot size would be obtained if the exponential curve is extrapolated linearly to intercept the frequency scale as shown in Figure 4. The x-intercept would then give the value of f to be used in Equation 1. Employing this procedure, the focal spot size came out to be independent of the magnification factor involved in the imaging process, as it should be.

Satisfying as this procedure is, it is not practical for routine use. Microdensitometers are expensive and are not readily available. For this reason we proceeded to investigate if the square wave response could be obtained using an ordinary laboratory densitometer. A special aperture measuring 0.25 mm. in diameter was made. Using this aperture the maximum(Imax) and the minimum(Imin) densitometer readings in each of the bar patterns were measured. The ratio

$$\frac{\text{Imax} - \text{Imin}}{\text{Imax} + \text{Imin}}$$

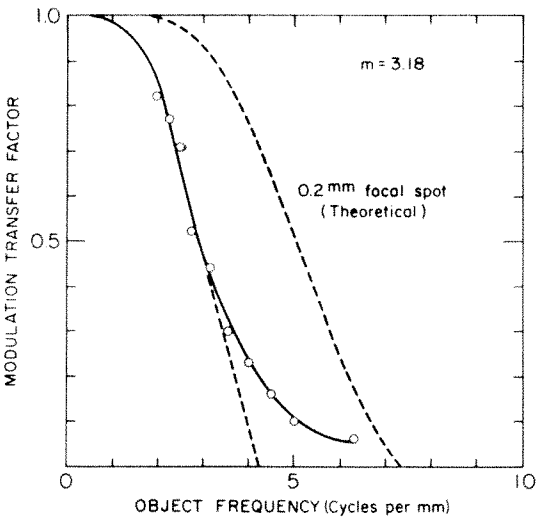


FIG. 4. Square wave response for a roentgenogram focal spot (see text for explanation).

yielded the square wave response in each case. Of course, the reading corresponding to the base fog of the film was subtracted from Imax and Imin before this ratio was determined. As the film was exposed to densities under 1.0 and as intensifying screens were not used, it was possible to assume that the densitometer readings were linearly proportional to the radiation exposure. It was gratifying to note that this method also yielded a square wave response curve closely in agreement with that obtained using a sophisticated microdensitometer and shown in Figure 4.

DISCUSSION

One of the most important findings of this study was that the equivalent focal spot size can change drastically with tube current.

A series of phantom experiments were carried out to see if these changes were large enough to be clinically important. Figure 5 shows 2, one made at 50 KVP, 25 mA (focal spot size 0.36 mm.×0.39 mm.) and the other at 50 KVP, 100 mA (focal spot size 0.51 mm.×0.80 mm.). The total mAs was the same in each case. At $m = 2.5$, obviously the difference is conspicuous. However, at $m = 1.5$, the difference still exists, but is

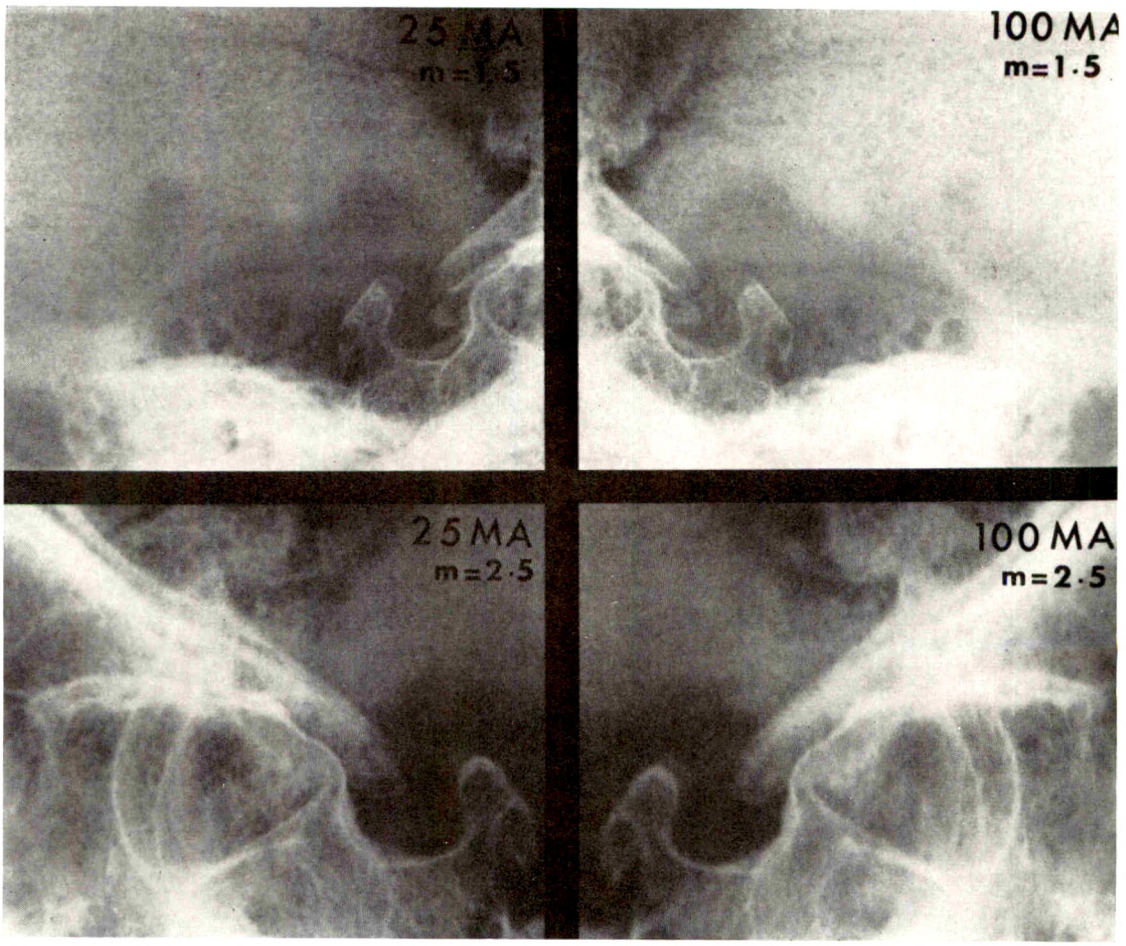


FIG. 5. Roentgenograms demonstrating the effect of changes in focal spot size with tube current. All the roentgenograms shown here were made at 50 KVP and 50 mAs. The only variables are the tube current and the magnification factor. The measured focal spot size was 0.36 mm.×0.39 mm. at 25 mA and 0.51 mm.×0.80 mm. at 100 mA.

much less. If the roentgenograms are made at $m \simeq 1$, the difference between them would be even less. These results are of course quite as expected, since the smaller the magnification factor, the less the importance of the focal spot. Another point to be remembered is that the faster the screens, the less the importance of focal spot size at any given magnification factor. This is illustrated in Figure 6 which shows the composite Modulation Transfer Functions of different screens used at different magnification factors. Notice that at $m = 1.5$, with fast screens, the resolution is not limited seriously by the size of the focal spot, whereas with slow screens it is. This means that if one uses fast screens to avoid motion unsharpness and does not use magnification factors in excess of 1.5 or so, one need not be too concerned about the variation of focal spot size with technical factors. Indeed, one may be able to use even a 0.6 mm. tube and still get roentgenograms of comparable quality. Another conclusion that may be drawn from this study is that if one has a tube which shows a drastic change in focal spot size with tube current, it is preferable to use a high KV and a low tube cur-

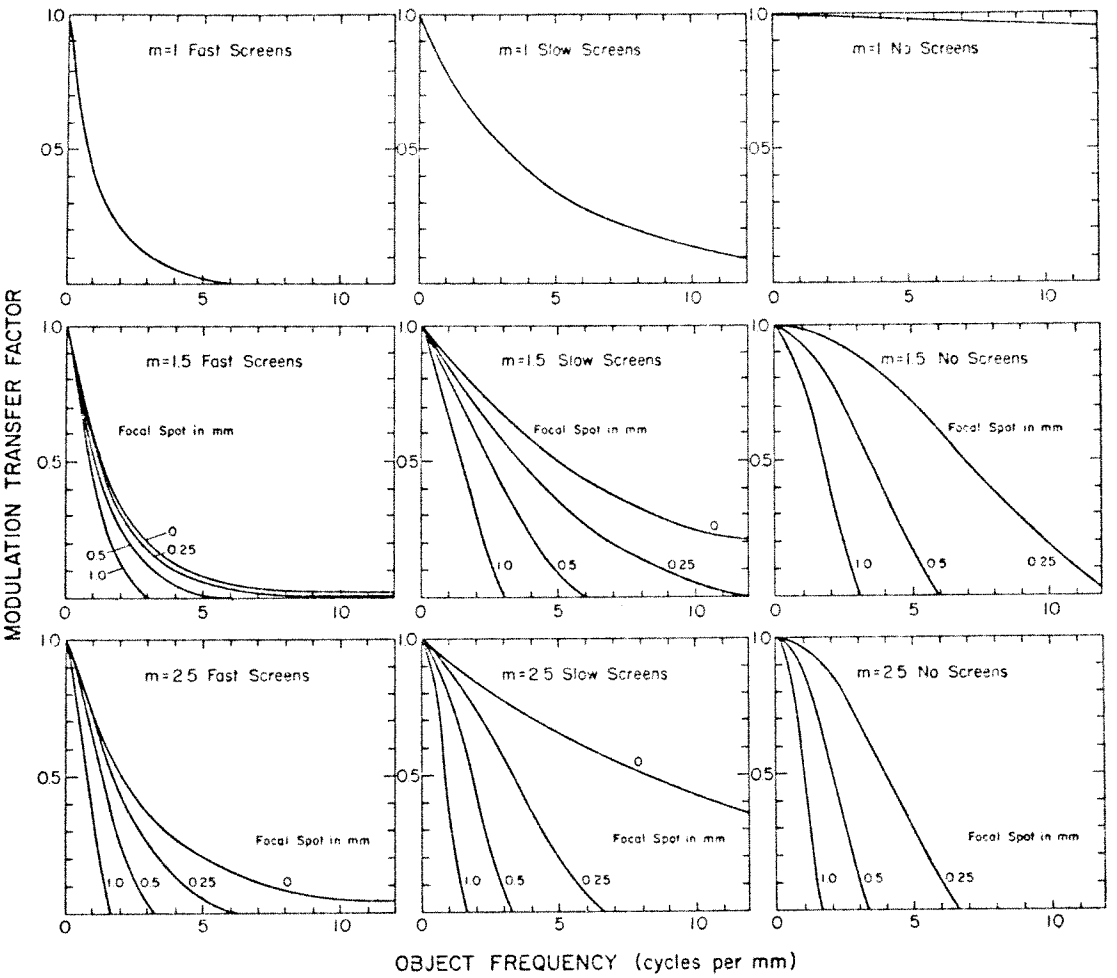


Fig. 6. Composite Modulation Transfer Functions of different radiographic imaging systems (see text for explanation).

rent. However, unfortunately, the lower the tube current, the larger the exposure time and this might introduce some motion unsharpness, if the object motion is excessive. A possible escape from this is the use of faster screens, but then the screen unsharpness may be excessive. Thus, in routine magnification radiography, one has to carefully evaluate the relative importance of focal spot size, screen speed, object motion and the magnitude of scattered radiation present in order to establish optimal routines. Several examples of making this type of calculations have been reported elsewhere.[4]

SUMMARY

The focal spot size of four 0.3 mm. microfocus commercial x-ray tubes was measured at various kilovoltages and tube currents using a resolution method.

The implications of the variations observed are discussed in relation to clinical magnification radiography.

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EDITORIAL

CARDIOVASCULAR RADIOLOGY—CURRENT STATUS

THIS issue of the JOURNAL is devoted primarily to Cardiovascular Radiology and it seems appropriate to examine the current status of this subspecialty of diagnostic radiology. The discipline of Cardiovascular Radiology is both a study of acquired and congenital heart disease and of peripheral vascular disorders. In this context perhaps the word is best hyphenated cardio-vascular radiology. Current trends in internal medicine and surgery include subspecialization and it is increasingly apparent that this trend is also developing in diagnostic radiology. Subspecialization in neuro and pediatric radiology leading to special competence in these areas has enabled the radiologist to relate more effectively than the "generalist" with his medical and surgical counterparts.

Before beginning their training in cardiovascular radiology, most residents ask us one or more of the following questions: (1) How much training do I have to have before I become competent? (2) How can I get training in coronary and cardiac catheterization? or more commonly (3) Can I just do abdominal and peripheral angiography instead of cardiac radiology? It is clear to us that a three month rotation through cardio-vascular radiology does not constitute sufficient training to become competent in this subspecialty. It is also apparent that each resident rotating through cardio-vascular radiology should have some experience in the radiology of the heart and not just the peripheral vessels. Finally, if a radiology resident wishes to achieve special competence in cardiac techniques, an extensive training period beyond the introductory rotation in cardiovascular radiology is required.

Learning percutaneous vascular techniques is really the simplest part of cardiovascular radiology. Of course, there are some inherent differences in manual dexterity among trainees which allow some to progress faster than others in gaining confidence with these techniques, but after a three month rotation on an active angiography service, most trainees have gained some facility in performing procedures.

Cardio-vascular radiology actually begins with consultation between the referring physician and the radiologist. This aspect includes: (1) should the patient be studied? and (2) what type of radiographic procedure is most likely to furnish the desired information? The cardio-vascular radiologist must share in this decision since he is responsible for the patient during and immediately after the study. Interviewing the patient, talking to the patient's physician and reviewing the clinical record as well as all previous roentgenographic data is part of the pre-study evaluation. If the cardio-vascular radiologist feels that the requested procedure is not indicated or that it should be modified, it is his responsibility to convey his opinion to the patient's physician in order to achieve an optimal diagnostic plan for each patient. It is not possible to develop this type of judgment by simply reading texts or articles, but instead it requires personal experience and training to achieve this goal. It also takes time to become skilled and confident in the roentgenographic interpretation of complex cardio-vascular abnormalities and hemodynamic flow patterns, to learn the optimal application of the equipment utilized in cardio-vascular radiology, and to gain an awareness of the types of complications that occur as a

result of cardio-vascular procedures. The cardio-vascular radiologist is often the first physician to recognize a complication and he should be thoroughly conversant with the alternatives and the techniques of proper treatment. He should be trained to initiate corrective therapy in an emergency situation or at least know the proper medical or surgical consultant to assist him in managing the complication. These four aspects of cardio-vascular radiology (judgment, roentgenographic interpretation, understanding of equipment, and management of complications) require more than a three month training period. Training should also be provided in cardio-vascular pathology, research techniques and related pharmacology.

For the individual seeking competence only in peripheral angiography whether it be in private practice or an academic setting, there should be a minimum requirement of one year of training in vascular radiology. During this year there should be some exposure to cardiac procedures since the vascular radiologist, particularly in a community hospital, will of necessity be expected to have some knowledge of these examinations.

It is also clear to us that to have special competence in *cardiac* and *peripheral* vascular procedures will require a minimum training period of two years (Table 1). This does not mean that the cardio-vascular radiologist has to be equally accomplished in cardiac as well as in peripheral vascular catheterization, but after two years he should have enough experience and training to effectively direct a laboratory performing both types of procedures. More importantly he will be able to relate well with cardiologists, cardiovascular surgeons, and vascular surgeons who have had a similar two year training experience in a subspecialty beyond their general medical and surgical residency. It is anticipated that among the cardio-vascular radiology staff, some members will be more interested in cardiac procedures and others in peripheral vascular studies, but all should be competent in both of these disciplines. This two year period of experience would seem appropriate for the trainee remaining in an academic department or entering private practice in a hospital where he is expected to perform both categories of examinations.

Table 1

Cardio-Vascular Radiology Training	Competence
3 month rotation during first 2 years of residency	Introduction to range of available techniques and methodology
1 year rotation during 3rd and 4th years of residency	Abdominal and peripheral angiography
2 year rotation during 3rd and 4th years of residency	Abdominal, cardiac and peripheral angiography

It might be argued that since cardiologists in this country perform most cardiac and coronary catheterizations, there is little opportunity for the radiologist to contribute to or participate in these procedures. This sentiment is often expressed as an explanation to justify abbreviated cardio-vascular training for radiology residents. Our experience, however, has been that the well trained cardio-vascular radiologist has a great deal to contribute and need not assume a passive role among his peers in cardiology. After a two year training period he is more familiar with radiographic and catheterization techniques and the use and capability of equipment than the medical resident after a two year cardiac fellowship. The cardio-vascular radiology trainee spends two years in the laboratory, while the cardiac fellow must divide his time between the cardiac catheterization laboratory, outpatient and inpatient cardiology and noninvasive procedures such as electrocardiography and phonocardiography.

"Competition" for the performance of catheterization procedures is counter-productive and only adds to interdepartmental

friction. Both the cardiologist and the radiologist can contribute to the information content of procedures by virtue of their special orientation in the catheterization laboratory. The responsibility for "pushing the catheter" should be shared among those in the laboratory, who have had proper clinical training irrespective of their specialty orientation.

One particular trend developing in diagnostic radiology requires special scrutiny: It is the concept that cardio-vascular radiology be divided by systems; *i.e.*, so that the pulmonary, renal and visceral angiographies are performed by those members of the Radiology Department with special interest in these areas. Arguments can be mustered both *pro* and *con* for this arrangement. Part of the discussion on this point should relate to some of the fundamental questions considered earlier. Angiographic pathology not always simply relates to just one system, and so the angiographer must be thoroughly conversant with all types of vascular radiology. This means that should one only want to be a "renal angiographer," he should first achieve thorough training in vascular radiology, so he won't end up having to call a "thoracic angiographer" when searching for a thoracic pheochromocytoma etc. This fragmentation in cardio-vascular radiology, in our opinion, has some undesirable features. It would seem illogical to have a vascular surgeon request a urologist to perform the operation when he encounters an abdominal aortic aneurysm that requires a vascular repair on the renal artery. Vascular radiology like vascular surgery consists of a variety of techniques which can be applied to many systems in the body. Since there is definite morbidity and, rarely, mortality associated with vascular techniques, it is important that those individuals performing vascular radiologic techniques be thoroughly conversant with this subject.

What does all this mean for the resident in training in diagnostic radiology? What it means in our opinion is that all residents in diagnostic radiology should rotate through the subspecialties during the first two years of their training just as their counterparts in medicine and surgery do. At the end of this two year period, some residents may elect to go into a training program in cardio-vascular radiology or others may elect to continue with a third year of diagnostic radiology and then perhaps pursue a training period in one of the subspecialties. Should there be an excess of trainees in any one of the subspecialties, the law of supply and demand in radiology should act as a control.

In addition to the Eastern and Western Societies of Angiography, there is a newly formed *Council on Cardiovascular Radiology* of the American Heart Association and a newly formed *North American Society of Cardiac Radiology*. It is our opinion that these organizations in conjunction with the American Board of Radiology and the American College of Radiology should address themselves to a policy which will outline training in cardio-vascular radiology and establish uniform criteria for special competence in this area.

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THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Seventy-fourth Annual Meeting of The American Roentgen Ray Society held at the Queen Elizabeth Hotel, Montreal, Canada, September 25–28, 1973, were again numerically large and scientifically the most educational in the history of the Society.

The secret Scientific Awards Committee, considering the excellent quality and splendid display of all exhibits, after much study and deliberation awarded 16 certificates of Merit and selected 3 exhibits for the great honor of the Society's *Gold*, *Silver* and *Bronze Medals*.

Gold Medal

Stridor in Infants and Children. Pliny C. Smith, M.D., Leonard E. Swischuk, M.D., and Charles J. Fagan, M.D., University of Texas Medical Branch, Galveston, Texas.

Many lesions cause stridor in infants and children, and most are demonstrable with plain film roentgenographic examination. Barium studies are also occasionally helpful, but endoscopy and bronchoscopy can usually be avoided.

To substantiate this premise, a concise review of normal anatomy and numerous lesions, from the nasopharynx to the trachea and upper esophagus, were illustrated and discussed.

Plain film roentgenographic diagnosis was stressed and special emphasis was given the upper esophageal foreign body whose clinical presentation (stridor, respiratory distress) can completely mislead the physician and cause extensive, potentially hazardous, examination of the larynx and trachea.

Other case presentations included retropharyngeal masses, pharyngeal lesions, vascular anomalies, and laryngomalacia.

There was a scientific essay on the same subject.

Silver Medal

Nerve Tumors and Neurofibromatosis. R. Ethier, M.D., D. Melancon, M.D., G. Belanger, M.D., P. C. Milette, M.D., and M. Jacoby, M.D., Montreal Neurological Hospital and Institute, Montreal, P.Q., Canada.

This exhibit dealt with the presentation of the roentgenologic features of nerve tumors and neurofibromatosis. These features were illustrated with cases of various nerve tumors and neurofibromatosis, as well as a self-teaching kit of approximately 70 slides which could be selected at random.

Special emphasis was placed on plain film roentgenographic examination. Anatomic specimens were shown.

Bronze Medal

Anomalous Pulmonary Venous Connection. Rogelio Moncada, M.D., Maurice Lev, M.D., Robert Miller, M.D., David Wasserman, M.D., Saroja Bharati, M.D., and Harvey White, M.D., Loyola University of Chicago, Maywood, Illinois.

Anomalous pulmonary venous connection is a developmental defect characterized by the lack of anatomic continuity between the pulmonary veins and the left atrium. These veins in turn must enter either a systemic vein or the right atrium.

The broad spectrum of bronchopulmonary abnormalities that are usually present in partial A.P.V.C. to the S.V.C., R. A. left vertical vein, and right and left-sided "scimitars" was presented. The supracardiac, cardiac and infradiaphragmatic varieties of total A.P.V.C. with their remarkably different clinical and hemodynamic alterations were shown, with emphasis on the gross pathologic anatomy and angiographic findings.

Certificates of Merit

Diagnosis and Detection of Arteriovenous Malformation of the Bowel. R. J. Alfidi, M.D., T. H. Hunter, M.D., W. Hawk, M.D., E. Winkleman, M.D., R. Turnbull, M.D., C. Esselstyn, M.D., and R. Herman, M.D., Cleveland Clinic Foundation, Cleveland, Ohio.

Isolated arteriovenous malformations (AVM) of the bowel are a seldom recognized cause of chronic gastrointestinal tract bleeding. They are rarely visible on conventional barium studies and at laparotomy. Pathologists may not be able to detect them in surgical specimens.

Selective arteriography is the only method currently available for diagnosis and reliable localization at laparotomy and in surgical specimens. Corrosion casting of plastic injected arteriovenous malformations provides a logical and consistent method of detecting small lesions in surgical specimens. Surgical correlation with arteriograms is good, when the lesions are located in the colon. For isolated lesions located in the small intestine, concomitant arteriography with laparotomy should be used for localization.

This was amply demonstrated in the exhibit.

Anomalous Arterial Communications in the Chest. Edmund A. Franken, Jr., M.D., and Roger A. Hurwitz, M.D., Indiana University Medical Center, Indianapolis, Indiana.

Many uncommon anomalous arterial communications in the chest have been described which mimic the more well known lesions (example—patent ductus arteriosus).

In the exhibit these vascular anomalies were classified in anatomic and hemodynamic fashion and their cineangiographic characteristics described. They included:

I. Direct Aortic—Pulmonary Communications
II. Systemic Artery
  A. Fistula—to vein, lung, pulmonary artery
  B. Anomalous origins—subclavian and coronary arteries
III. Pulmonary Artery
  A. Fistula—to vein, left atrium
  B. Anomalous origin—from ascending or descending aorta.

The Importance of Liver-Spleen Spatial Relationship in Scan Diagnosis. Raymundo Tiu Go, M.D., Norihisa Tonami, M.D., Ching Liang Chiu, M.D., Rolf L. Schapiro, M.D., and James Henry Christie, M.D., Department of Radiology, University of Iowa, Iowa City, Iowa.

The purpose of the exhibit was to show liver-spleen height disparity and flattening or depression of the liver-spleen dome as important signs in scan diagnosis.

These signs, as seen in the routine technetium 99m sulfur colloid liver-spleen scan, lead the Exhibitors to suspect and detect supradiaphragmatic, diaphragmatic or subdiaphragmatic abnormalities. Correlation with routine or inspiratory chest roentgenograms is important and may reveal the suspected abnormality. If the chest roentgen finding does not explain the scan signs, then a transmission-emission scan is obtained. This will often delineate the presence and extent of the abnormality and will usually help to decide what further studies to pursue.

Liver-spleen height disparity is observed in abnormal conditions such as cardiomegaly, pericardial effusion and/or unilateral pleural effusion, unilateral abnormally high hemidiaphragm, subdiaphragmatic abscess and intrasplenic pathology. Flattening or depression of the liver-spleen dome is seen in pulmonary emphysema and bilateral pleural effusion.

Lymphoproliferative Disorders of the Thorax. E. R. Heitzman, M.D., B. Markarian, M.D., and C. T. DeLise, M.D., State University of New York, Upstate Medical Center, Syracuse, New York.

This exhibit illustrated those diseases of lung characterized by lymphocytic infiltration of the pulmonary parenchyma and mediastinal structures.

Lymphocytic interstitial pneumonia, pseudolymphoma and lymphoma were considered and their inter-relationship discussed; their roentgenographic appearance and clinical course were reviewed in detail.

Newly recognized information concerning the immunoglobulin abnormalities associated with these diseases was included. Possible associations with other entities such as Sjögren's syndrome, Mikulicz' syndrome, inflammatory pseudotumor of lung, Waldenström's disease and angiofollicular lymphoid hyperplasia were explored.

Renal Microcirculation following Papillary Damage. Denis Lyonnet, M.D., Harry Z. Mellins, M.D., Peter G. Herman, M.D., and Itaru Yamamoto, M.D., Department of Radiology, Harvard Medical School, Boston, Massachusetts.

Silicone rubber perfusion studies in rabbits support the view that cortical changes which follow papillary injury are not the result of vascular damage but may be the sequelae of collecting tubule lesions.

This was demonstrated in the exhibit.

Pitfalls in the Radiographic Diagnosis of Trauma. Robert D. MacIntyre, M.D., Norma Fallis Harris, M.D., and Gene C. Cunningham, Jr., M.D., Department of Radiology, Baylor College of Medicine, Houston, Texas.

Twenty cases of unusual fractures or dislocations or those difficult to diagnose were presented.

They represented entities normally encountered in the Emergency Room requiring no special techniques for demonstration. The presentation was such that viewers could test their diagnostic acumen, if they chose.

All of the cases were collected at the Baylor Affiliated Hospitals over a 4 year period.

Haustral Anatomy and Pathology: A New Look. Morton A. Meyers, M.D., Frank Volberg, M.D., Barry Katzen, M.D., and Gerald Abbott, M.D., The New York Hospital-Cornell University Medical Center, New York, New York.

The distinctive haustral contour of the large intestine is provided by 3 bands of longitudinal muscle, the tenia mesocolica (TM), tenia omentalis (TO), and tenia libera (TL). The haustral sacculations are thus organized into 3 easily identifiable rows: (1) TM-TO; (2) TO-TL; and (3) TL-TM. Each row of haustra has characteristic anatomic relationships.

The purpose of this report was four-fold: (1) to clarify the role of the tenia in the colonic contour; (2) to establish the anatomic definition and relationships of the rows of haustral sacculations and their roentgenographic identification; (3) to determine the fascial planes of spread from adjacent organs to specific rows of haustra first involved; and (4) to document the roentgenologic localization and diagnosis of intraabdominal disease states by their effect on haustral sacculations.

These observations were based on postmortem studies involving contrast examination of the colon with identification of anatomic relationships, injection of the mesenteric attachments of the colon to determine the planes of spread, and selected clinical cases.

Hypotonic Radiography with Glucagon. Roscoe E. Miller, M.D., Stanley M. Chernish, M.D., Jovitas Skucas, M.D., Bernard D. Rosenak, M.D., and Bruce E. Rodda, Ph.D., Indiana University School of Medicine, Indianapolis, Indiana.

The effect of glucagon, atropine sulfate, and propantheline bromide on the stomach, duodenum, and small bowel was compared to placebo in 72 roentgenographic examinations. This double blind crossover study showed that in 10 and 30 minutes there was a significant decrease in gastric, duodenal, and small bowel motility and tonicity with all the active drugs. Glucagon, however, was significantly better than the anticholinergics, had little action at 60 minutes, and had few side effects.

Atropine sulfate and propantheline bromide were still effective at 60 minutes, and the intensity of side effects was much more distressing with propantheline bromide.

There was a scientific essay on the same subject.

Paths of Progress. Charles E. Mullins, M.D., John T. Dawson, Jr., M.D., and Dan G. McNamara, M.D., Baylor College of Medicine and the Texas Children's Hospital, Houston, Texas.

This display was designed to present spot film roentgenograms showing "characteristic" and unusual catheter courses in a variety of cardiac malformations including: cervical aortic arch; total anomalous venous return; L-transposition; D-transposition; Pott's anastomosis; patent ductus arteriosus; double outlet right ventricle; asplenia syndrome; as well as normal hearts.

The corresponding diagrams revealing the catheter courses and anatomy were available to the viewer by pushbutton. The display was challenging and instructive to the viewer as to his knowledge of possible catheter courses.

The complicated patterns created by the catheters were also intriguing.

The Meconium Plug Syndrome. Roentgen Evaluation and Differentiation from Hirschsprung's Disease and other Pathologic States. Rubem Pochaczevsky, M.D., and John C. Leonidas, M.D., City Hospital Center at Elmhurst and The Mount Sinai School of Medicine, New York, New York.

The authors' experience with 14 cases of the meconium plug syndrome and 13 cases of Hirschsprung's disease was presented and the roentgenographic findings reviewed. Criteria for differentiation of meconium plug syndrome from Hirschsprung's disease as well as from other pathologic processes such as necrotizing enterocolitis of infancy and meconium ileus were formulated and illustrated.

Infusion Tomography of the Gallbladder. Sanford E. Rabushka, M.D., Leon Love, M.D., and Rogelio Moncada, M.D., Loyola University Medical Center, Maywood, Illinois.

It is possible by whole body opacification effect to visualize the wall of an abnormal gallbladder. This is accomplished by the administration of high doses of meglumine diatrizoate intravenously as an infusion technique similar to the one used for nephrotomography.

Tomographic sections at the gallbladder level may visualize the gallbladder after 15 minutes. The technique has been successful in: (1) nonvisualizing gallbladders following oral or intravenous cholangiography; (2) jaundice; and (3) acute cholecystitis.

The exhibit included gross specimens as well as microsections demonstrating wall thickness.

Hysterosalpingography. John A. Richmond, M.D., and Victor G. Mikity, M.D., Womens Division, LAC-USC Medical Center, Los Angeles, California.

Some common and unusual cases of OB-GYN pathology were shown. These cases were selected from 1,000 consecutive hysterosalpingograms obtained at the Womens Division, LAC-USC Medical Center.

The clinical and pathologic correlation of inflammatory, granulomatous and neoplastic diseases as well as of some rare congenital anomalies was demonstrated. The added combined use of pelvic pneumonography in selective cases was emphasized as a useful and valuable adjunct.

"Clipping Injury" Fracture of the Epiphysis in the Adolescent Football Player: An Occult Lesion of the Knee. Lee Frank Rogers, M.D., Stanley C. Jones, M.D., and A. Ross Davis, M.D., Department of Radiology and Department of Orthopedic Surgery, Hermann Hospital, The University of Texas Medical School, Houston, Texas.

The athlete's knee is quite vulnerable to ligamentous injury. This is particularly true of mature athletes. The high school athlete's or adolescent knee may react differently to the same stresses. The major difference in the osseous structure between these age groups is the presence of the growth plate and epiphysis in the adolescent skeleton. Forces that would result in a ligamentous injury in an adult are likely to

result in an epiphyseal injury in a child or adolescent.

Clipping injuries of the knee in the adolescent football player may result in occult, undisplaced epiphyseal separations. Clinically, these are commonly mistaken for ruptures of the medial collateral ligament. The latter require immediate surgery, whereas an epiphyseal injury requires only closed reduction and casting. This crucial distinction can be made roentgenographically. The findings are subtle and easily overlooked. Stress views may be necessary.

Seven cases were presented. The injury was reproduced in young rabbits. These specimens were utilized to demonstrate the significant anatomic relationships and the pathophysiology of injury.

There was a scientific essay on the same subject.

The Axial Pancreatic View: A New Approach for Recognizing Enlargement of the Body and Tail of Pancreas. Philip A. Sorabella, M.D., William L. Campbell, M.D., and William B. Seaman, M.D., Department of Radiology, Columbia-Presbyterian Medical Center, New York, New York.

The axial pancreatic view in the upper gastrointestinal series was designed to permit more accurate assessment of the cross-sectional area of the body and tail of the pancreas than is possible with conventional lateral views.

The patient is oriented such that a horizontal beam is directed 25° toward the patient's head and 20° posteriorly as it passes from right to left, thus aligning the beam with the axis and the body and tail of the pancreas. Diagrammatic and postmortem studies showed the theoretic basis for the sensitivity of this view in demonstrating enlargement of the body and tail of pancreas. Clinical cases were illustrated where the enlargement was shown only on the axial pancreatic view. The cases presented included carcinoma and acute pancreatitis.

The Role of Angiography in the Management of Hemorrhage following Pelvic Trauma. Arthur C. Waltman, M.D., Christos A. Athanasoulis, M.D., W. Robert Courey, M.D., Ernest J. Ring, M.D., and Stanley Baum, M.D., Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts.

Arteriography was performed on 6 patients massively hemorrhaging following pelvic trauma and 2 patients following hip surgery.

The bleeding points were localized and hemostasis was produced by intraarterial techniques (balloon and blood clot).

Hysterosalpingography in Infertility. Heun Y. Yune, M.D., Robert E. Cleary, M.D., Loren Petersen, M.D., and Eugene C. Klatte, M.D., Indiana University Hospital, Indianapolis, Indiana.

Contributions of hysterosalpingography in the work-up and treatment of female infertility have been considered to be anywhere between indispensable by some and unnecessary by others.

The Exhibitors have reviewed all case records of patients who came to Indiana University Medical Center Hospital in the past 5 years. The cases were classified into correctable and uncorrectable causes of infertility; the role the hysterosalpingography played towards diagnosis and establishment of therapeutic measures in each patient was re-evaluated.

The results indicated areas of positive and often definitive diagnostic contribution and sometimes a therapeutic effect of this examination.

There was a scientific essay on the same subject.

Other Excellent Exhibits Were:

Early Diagnosis of Breast Cancer. American College of Radiology in cooperation with the National Cancer Institute and the American Cancer Society.

This beautifully displayed and authoritatively documented exhibit was a descriptive presentation of various techniques of detecting early carcinoma of the breast and announcing centers for reorientation of

physicians and technologists in the different modalities currently used.

Congenital Diaphragmatic Defects in the Pediatric Patient. Ronald B. Addlestone, M.D., Arvin E. Robinson, M.D., and Herman Grossman, M.D., Department of Radiology, Duke University Medical Center, Durham, North Carolina.

While emphasis has been on understanding the persistent pleuroperitoneal type of posterior diaphragmatic defect, other abnormalities of the diaphragm which present in the pediatric age group are often poorly defined, and consequently managed inconsistently.

These defects include: anteromedial, posterolateral and esophageal hiatal hernias; and partial and complete eventrations. With proper recognition of the development and presentation of these various abnormalities, differences in the proper management and prognosis become apparent.

An understanding of the development of these abnormalities and an evaluation of clinical limitations will allow a better determination of the most effective method of management than immediate concern with abnormal roentgenograms.

Pediatric Case of the Day. Walter J. Alexander, M.D., and Barry D. Fletcher, M.D., Department of Radiology, The Montreal Children's Hospital and McGill University, Montreal, P.Q., Canada.

This exhibit consisted of the daily exposition of roentgenograms depicting the findings in typical pediatric conditions. Related historical and clinical information was given, where indicated. For each daily case, the observer was afforded the opportunity to indicate his or her interpretation and diagnosis.

The documented answer was displayed on the following day.

Retroperitoneal Fibrosis. Peter H. Arger, M.D., Jonathan L. Stolz, M.D., and Wallace T. Miller, M.D., Hospital of University of Pennsylvania, Philadelphia, Pennsylvania.

Retroperitoneal fibrosis presents with a clinical and roentgenographic spectrum involving the ureters, inferior vena cava, iliac vein and rectum. The clinical and roentgenographic aspects of this spectrum were shown and demonstrated.

Medial deviation of tapered obstructed ureters in the lower lumbar region was described as a "classic sign" of retroperitoneal fibrosis.

A comparative statistical analysis of the position of the ureters relative to the lower lumbar spine and sacrum was done, comparing 19 cases of retroperitoneal fibrosis with 60 normal cases. This analysis was shown and the lack of significant difference between the 2 groups demonstrated.

Pseudotruncus Arteriosus: The Importance of Selective Bronchial Angiography for the Planning of Total Surgical Repair. Harold A. Baltaxe, M.D., David C. Levin, M.D., Aaron R. Levin, M.D., Henry P. Goldberg, M.D., Mary Allen Engle, M.D., Thomas A. Sos, M.D., and Paul A. Ebert, M.D., Cornell University Medical Center, New York, New York.

Pseudotruncus is now amenable to total surgical repair by the use of aortic homografts. In order to perform this procedure, the blood supply to the lungs must be fully appreciated.

The anatomic arrangement of the pulmonary and bronchial arteries is greatly variable in pseudotruncus and must be outlined by selective angiography.

This exhibit discussed in detail the various anatomic arrangements found in pseudotruncus. Examples of various types of blood supply to the lungs were demonstrated, and the importance of their visualization was stressed in the light of a possible surgical correction.

Experimental Diphenylhydantoin Induced Rickets. A. Bar, Ph.D., W. Bergstrom, M.D., R. Chiroff, M.D., L. V. Gould, M.B., Ch.B., M. Villareal, B.S., and R. H. Wasserman, Ph.D., Cornell University, Ithaca, New York, and Upstate Medical Center, Syracuse, New York.

That rickets and osteomalacia occur in patients treated with anticonvulsant ther-

apy was first reported in the late 1960s and is now widely recognized. Most reports involve patients treated with a combination of phenobarbitone and diphenylhydantoin (DPH).

Clark *et al.* (*Invest. Radiol.*, 1971, *6*, 152–154) found no evidence of rickets in rats treated with DPH, suggesting that DPH was not responsible for clinical rickets or osteomalacia.

In other experimental animals, the Exhibitors have produced definite evidence of rickets related to dosage with DPH. This is found to be associated with proportional changes of calcium absorption and calcium binding protein. This observation incriminates DPH as an agent altering calcium metabolism.

Pulmonary Manifestations of Sarcoidosis. Yahya Berkmen, M.D., and Robert Bard, M.D., Department of Radiology, New York Medical College, New York, New York.

The 4 stages of pulmonary sarcoidosis are: Stage I—hilar lymphadenopathy–asymptomatic; Stage II—parenchymal lesions–subacute or acute symptoms; Stage III—parenchymal lesions with lymphadenopathy–symptomatic; and Stage IV—fibrosis–respiratory insufficiency.

Cases typical of these 4 stages were presented in the exhibit.

Case of the Day. Alfred S. Berne, M.D., Richard D. Gerle, M.D., George E. Mitchell, M.D., John C. Sanborn, M.D., Bernard Schneider, M.D., and Howard R. Wilkov, M.D., Crouse-Irving Memorial Hospital, Syracuse, New York.

Unusual radiologic cases of educational value were shown for analysis.

The following day the correct answer, and explanation and the appropriate bibliography were demonstrated.

Endoscopic Cholangiopancreatography. M. K. Bilbao, M.D., T. Lee, M.D., R. M. Katon, M.D., J. Rösch, M.D., and C. T. Dotter, M.D., University of Oregon Medical School, Portland, Oregon.

A method for peroral transpapillary retrograde cholangiopancreatography using fluoroscopically controlled endoscopy and radiography was described.

The method has yielded a high rate of successful papillary cannulation even in inexperienced hands (97 per cent of the first 35 cases), and therefore appears to be an improvement over previously published methods.

The radiologic findings, indications, and role in patient management were illustrated.

Metastatic Lesions from Common Urinary Tract Malignancies. Robert C. Brown, M.D., C. M. Kinloch Nelson, M.D., James A. Matter, M.D., Raymundo T. Go, M.D., Petronio T. Lerona, M.D., and Rubin H. Flocks, M.D., University of Iowa Hospitals, Iowa City, Iowa.

A correlation of the gross pathologic, radiographic and radioisotopic features of metastases from 3 common genitourinary neoplasms—adenocarcinoma of the kidney, transitional cell carcinoma of the bladder, and carcinoma of the prostate—was demonstrated.

Special attention was given to angiographic and radioisotopic features of these lesions when applicable.

Acute Rectal Hemorrhage: New Concepts in Etiology Based on Arteriography. William J. Casarella, M.D., Shelby J. Galloway, M.D., Edward M. Pollock, M.D., Richard N. Taxin, M.D., and David A. Follett, M.D., Department of Radiology, Columbia-Presbyterian Medical Center, New York, New York.

In a large series of clinical cases of acute rectal hemorrhage, emergency arteriography has been shown to be the only diagnostic technique capable of locating the bleeding point preoperatively.

Certain conclusions can be drawn from these studies:

1. Arteriography is a safe, reliable procedure.
2. Most bleeding points are located in the right colon.
3. Diverticulosis is the most common cause of acute rectal hemorrhage.

4. Arteriovenous malformations of the cecum are frequently associated with valvular heart disease.
5. Upper gastrointestinal tract lesions may present as rectal bleeding.
6. A spectrum of other lesions is recognizable on angiography.

Examples which demonstrate these points were shown along with pathologic correlation.

A scientific essay was presented on the same subject.

Xanthogranulomatous Pyelonephritis; with Angiographic Evaluation. Eung Man Cha, M.D., Fei-Shung Chang, M.D., Nader Sadoughi, M.D., George B. Greenfield, M.D., and Albert Rubenstone, M.D., The Chicago Medical School, Mount Sinai Hospital, Chicago, Illinois.

Four cases of xanthogranulomatous pyelonephritis (unilateral) studied by intravenous pyelography, nephrotomography, and renal arteriography, and proven by nephrectomy were reviewed. The intravenous pyelography and nephrotomography showed a nonfunctioning kidney or renal mass.

The renal angiographic findings were: (1) dilated renal capsular artery; (2) prominent renal perforating arteries; (3) prominent small arteries adjacent to the lesion; (4) tumor-like stain mixed with avascular areas; and (5) dilated lumbar and superior mesenteric arteries when the process extends.

Although this condition has been misdiagnosed preoperatively in the past, the Exhibitors stated that effort should be made to differentiate this benign lesion from other entities.

Radiographic Ureteral Changes with Appendicitis. Ching L. Chiu, M.D., and Ronald R. Gambach, M.D., Department of Radiology, University of Iowa, Iowa City, Iowa.

Ureteral changes on the intravenous pyelogram in patients with appendicitis include: right ureteral deviation; and ureteropyelocaliectasis and right ureteral spindle (mild fusiform dilatation of the middle one-third of the ureter above pelvic brim with ureteral opacification proximally to the ureteropelvic junction).

Patients older than 50 years with appendicitis exhibit right ureteral deviation as the most common single abnormality. Patients under 31 years exhibit right ureteral spindle as the most common single change.

Ureteral spindle with appendicitis may be due to periureteral inflammation with edema and narrowing of the ureteral lumen, ureteral atony or extrinsic compression with partial ureteral obstruction.

Roentgenograms of the Pulmonary Macrosection. Ching L. Chiu, M.D., and Earl F. Rose, M.D., Iowa University Hospital, Iowa City, Iowa.

Roentgenographic macrosections of the lung (illumination of 300–400 micra thickness of the paper mounted macrosections of the lung) are useful for demonstrating gross pathologic and functional changes and for comparing the pathology with the roentgenologic findings.

Roentgenographic macrosections provide an additional advantage of permanent fine intrinsic changes and less confusion by color in the background. These are particularly useful in postmortem evaluation of pulmonary gross pathology and are most valuable for teaching purposes.

Traumatic Diaphragmatic Hernia. J. Luther Clements, Jr., M.D., Gwynne Brunt, M.D., Turner Ball, M.D., Steven Cline, M.D., and H. Stephen Weens, M.D., Department of Radiology, Emory University School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

Traumatic diaphragmatic hernia is encountered with increasing frequency. Patients with this condition may be acutely ill or remain asymptomatic for many years following injury.

An analysis of the Exhibitors' material indicates that a surprisingly large number of cases are not properly recognized (approximately 50 per cent).

Obvious and subtle roentgen features of

traumatic diaphragmatic hernia in acute cases as well as in patients who remain symptom-free for prolonged periods of time were demonstrated and evaluated. Plain film roentgenographic findings were stressed.

Cervical Esophageal Webs—A Roentgen Anatomic Correlation. J. Luther Clements, Jr., M.D., William E. Torres, M.D., H. Stephen Weens, M.D., and George Cox, M.D., Department of Radiology, Emory University School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

With the use of cine and videotape recording, web-like structures are not infrequently detected on barium studies of the cervical esophagus. Most often these roentgenologic defects are recorded in asymptomatic individuals.

A careful roentgenologic-pathologic study has been conducted on autopsy material in an attempt to determine the true nature of these observations. The Exhibitors' studies led them to believe that there are 2 types of cervical esophageal webs: one type, most commonly seen, representing dynamic structures due to plication of normal mucosa; and a second type, less frequently observed, representing evidence of organic mucosal disease.

There was a scientific essay on the same subject.

Ultrasound "B" Scanning of Palpable Breast Masses also Examined by Mammography. Catherine M. Cole-Beuglet, M.D., The Montreal General Hospital, Montreal, P.Q., Canada.

One hundred and fifty breast masses were examined clinically, roentgenographed and studied by ultrasound "B" scanning; and pathologically confirmed. In 75 per cent of the cases, the palpable mass was identified by ultrasound "B" scanning. The ultrasound characteristics were reliable in differentiating fluid filled cysts from solid lesions and surprisingly reliable in differentiating infiltrating malignancies in younger patients, where the mammogram was not diagnostic.

In this series, pathology included fibrocystic disease (cystic type and sclerosing adenosis type), fibroadenomas, infiltrating carcinomas, and medullary carcinomas.

Criteria for ultrasonic classification were defined.

Craniofacial Anomalies: Current Surgical Approach to Management. John Marquis Converse, M.D., Donald Wood-Smith, M.D., Melvin H. Becker, M.D., Peter J. Coccaro, D.D.S., Joseph Ransohoff, M.D., Fred Epstein, M.D., Mnashe Vallo Benjamin, M.D., and Joseph G. McCarthy, M.D., New York University Medical Center, New York, New York.

Surgical treatment of craniofacial anomalies has made tremendous advances in the past few years.

This exhibit was illustrated by drawings of the surgical procedures and photographs and roentgenograms before and after the operation.

The Excretory Urogram Revisited: The Concentration/Density Misconception. Peter Dure-Smith, M.D., and Michael Simenhoff, M.D., Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

Many of the procedures used to improve the quality of the excretory urogram such as preliminary dehydration, increasing the dose of contrast medium, etc., are designed to increase the concentration of contrast medium in the urine.

This exhibit gave evidence which suggests that urinary contrast medium concentration is an unsatisfactory index for renal opacification because opacification depends on the total amount of contrast medium = concentration × depth.

During excretory urography, there are significant changes in depth in the tubules, and in the collecting system which contribute to the density of the nephrogram and pyeloureterogram, respectively.

There was a scientific essay on the same subject.

Isotopic Venography in the Evaluation of Venous Obstructive Disease. David A. Emmerson, M.D., Robert E. O'Mara, M.D., David L. Lilien, M.D., and Eric G. Mayer, M.D., Division of Nuclear Medi-

cine, University of Arizona Medical Center, Tucson, Arizona.

Radioisotopic evaluation of the superior or inferior vena cava and contiguous venous structures is of great value in the assessment of obstruction. Clinical presentations encountered may range from the mildest of symptoms to the complete syndrome as usually described.

The use of the noninvasive, simple and safe technique of isotopic evaluation of the major venous systems in these patients was described by presenting patterns of normal flow, the most usual obstructions, as well as the radioisotopic appearance of the various collateral pathways. These were correlated with the clinical and roentgenographic evaluation of the patient's condition and their use in follow-up evaluation of therapeutic modalities employed.

Mesenteric Cysts in Children. Charles Joseph Fagan, M.D., and Leonard Edward Swischuk, M.D., University of Texas Medical Branch, Galveston, Texas.

Multiple cases of surgically proven intra-abdominal mesenteric cysts, some masquerading as ascites, in children, ages 1 day to 4 years, were presented with emphasis on diagnostic roentgenographic methods.

Intramural Esophageal Lesions. Robert A. Ferris, M.D., Olcay S. Cigtay, M.D., and Homer Lee Twigg, M.D., Georgetown University Hospital, Washington, D. C.

Intramural esophageal lesions, and diseases simulating intramural esophageal lesions, present a limited but interesting variety of pathology. From the roentgenologic standpoint, because such lesions are easily accessible, familiarity with them is important.

Included in this exhibit were examples of: (a) leiomyomas; (b) duplication cyst (enteric cyst); (c) abscess; (d) bronchogenic cyst; (e) carcinoma; (f) carcinosarcoma; (g) mucocele; and (h) prominent normal mucosal folds simulating an intramural lesion, as well as other entities.

Although not pathognomonic, the Exhibitors believe that the "beak sign," as demonstrated in several of their examples, is a valuable means of localizing a true esophageal lesion.

Unusual Lesions of the Colon. Pieretta S. Ferro, M.D., Norma Fallis Harris, M.D., and Luciel B. North, M.D., V.A. Hospital, Houston, Texas.

A group of unusual conditions involving the colon that have been collected from the Baylor Affiliated Hospitals was presented. Information that might be of interest to the radiologist or pertinent from the standpoint of diagnosis was emphasized.

Fine-Detail Skeletal Radiography. Harry K. Genant, M.D., Kunio Doi, Ph.D., Jay C. Mall, M.D., Kurt Rossman, Ph.D., and James R. Williams, M.D., The University of Chicago Hospitals, Chicago, Illinois.

Fine-detail radiography is a technique which employs fine-grain film and optical magnification for detecting subtle alterations in the peripheral skeleton.

The *first part* of this exhibit demonstrated both quantitative and qualitative results from an image quality study of fine-detail radiography. Factors such as inherent film properties, beam characteristics, and geometry were shown.

The *second part* was devoted to clinical studies, comparing fine-detail and conventional radiographic techniques. The findings in patient populations having rheumatoid arthritis, primary hyperparathyroidism, pseudogout and renal osteodystrophy were summarized and examples shown. The emphasis was on the early detection of subtle disease and on the use of sequential examinations for assessing progression of disease.

The History of Scintillation Scanning. E. R. N. Grigg, M.D., Oak Park, Illinois.

The advent of imaging was instrumental in precipitating the growth of Nuclear Medicine. As a diagnostic method, nuclear imaging ranks second in importance only to roentgen imaging; even so, the scan is more informative than the respective roentgenogram.

The exhibit sketched the development to rectilinear scanning from Cassen to the present time, as a first part of what shall

become an Abbreviated Picture History of Nuclear Medicine.

The Papillary Ducts of Bellini in Hydronephrosis. N. Thorne Griscom, M.D., and Robert L. Lebowitz, M.D., Children's Hospital, Boston, Massachusetts.

Individual papillary ducts sometimes become visible during excretory urography in hydronephrosis. Reorientation of the ducts, dilatation because of stasis, and increased water reabsorption seem to be the factors allowing visualization.

A Study of Radiodispersions with the Scanning Electron Microscope. Jack N. Hall, B.S., Robert E. O'Mara, M.D., Peter B. Mee, B.S., and Richard L. Sloane, M.S., Arizona Medical Center, Tucson, Arizona.

There are approximately 30 radiodispersions suggested for use in Nuclear Medicine for imaging the lung, liver, spleen and bone marrow.

This exhibit was concerned with the use of the Scanning Electron Microscope to study the size, shape, and surface detail of these radioactive suspensions or colloids. The results of this application of the instrument in the study of radiodispersions were illustrated by means of multiple photographic comparisons of these agents as used for the various organ systems.

Clinical Trial of Bilopaque Oral Cholecystography. Sany Y. Han, M.D., and David M. Witten, M.D., Department of Diagnostic Radiology, University of Alabama School of Medicine, Birmingham, Alabama.

Eighty-eight consecutive patients underwent cholecystography at 4, 6, 8, 10 and 12 hour intervals after ingestion of Bilopaque.

This exhibit was an evaluation of the results on 78 patients with opacification, the earliest time of optimal visualization and residual contrast material in the bowel. In 61 of 78 patients (78 per cent), the gallbladder was opacified at 4 hours after ingestion. In 50 patients (64 per cent) the degree of opacification was diagnostic on the 4 hour roentgenogram. The earliest time of optimal opacification was at 4 hours in 18 patients, 6 hours in 16, and 8 hours in 26 patients. The residual contrast material was noted in only 6 patients on the 12 hour roentgenogram.

Statistical Considerations in Lung Scanning. Larry L. Heck, M.D., Medical Corps, USN, and John W. Duley, Medical Service Corps, USN, Naval Hospital, NNMC, Bethesda, Maryland.

Recent work in the Exhibitors' clinic has shown that false positive lung scans may result if too few particles are injected for lung scanning. Normal subjects may appear to have multiple pulmonary emboli or asthma if less than 10,000–15,000 microspheres are injected. Illustrative scans and scintiphotos demonstrated this phenomenon.

This observation becomes a distinct clinical problem now that most radiopharmaceutical companies are supplying Tc^{99m} generators that yield very high specific activity eluates. The exhibit emphasized the problems that may arise and discussed the practical solutions applied in their Nuclear Medicine Department.

Radiographic Manifestations of Tuberous Sclerosis. Carl W. Hoffman, M.D., Charles J. Fagan, M.D., and Leonard E. Swischuk, M.D., University of Texas Medical Branch, Galveston, Texas.

The classic clinical triad of (1) mental retardation, (2) adenoma sebaceum, and (3) convulsions or epilepsy, diagnostic of tuberous sclerosis, may not always be present. The radiologist, provided he is acquainted with the characteristic roentgen findings of this disorder, may be the first to make the diagnosis.

This exhibit illustrated the intracranial, osseous, renal and pulmonary findings diagnostic of tuberous sclerosis.

Operative Coronary Angiography, Gas Endarterectomy and Venous Bypass Grafts — A Total Approach to Coronary Artery Surgery. Martin J. Kaplitt, M.D., Stephen L. Frantz, M.D., Arthur R. Beil, Jr., M.D., Stephen J. Gulotta, M.D., and Harry L.

Stein, M.D., North Shore Hospital, Manhasset, New York.

The purpose of this exhibit was to demonstrate a new modality—operative coronary angiography—and its contribution to direct coronary artery surgery.

The exhibit illustrated with roentgenograms, drawings, and angiograms a total approach to coronary artery surgery for both segmental and diffuse disease. With the addition of high quality intraoperative angiography, a new dimension has been added to vein bypass and gas endarterectomy.

Cases were reviewed and all techniques were discussed.

The Technique and Diagnostic Excellence of Selective and Superselective Arteriography in Children. Mansour Khademi, M.D., and James R. Marquis, M.D., New Jersey College of Medicine and Dentistry and United Hospitals, Medical Center, Newark, New Jersey.

Using the Seldinger technique of percutaneous femoral puncture, 100 selective and superselective angiographies have been performed in children.

The Exhibitors' experience indicates that selective catheterizations in the young need not be more difficult than those performed in adults. The obvious diagnostic superiority of selective procedures over simple flush studies causes them to encourage the performance of such procedures and reassure arteriographers of their simplicity.

The technical aspects as well as the precautionary measures employed to prevent complications were discussed. Examples of the outstanding diagnostic value of selective arteriography in infants were given.

Angiographic Measurement of Renal Compartments (Corticomedullary Ratio in Normal, Diseased States and Sickle Cell Anemia). Mansour Khademia, M.D., New Jersey College of Medicine and Dentistry, Newark, New Jersey.

Renal cortical thickness cannot be estimated by intravenous urography because cortical material only partially occupies the space between the calyces and the renal border. This space was investigated by renal arteriography and venography. The corticomedullary junction was demarcated by arcuate arteries and veins, and in the nephrogram phase, the cortical thickness was directly measured.

These studies showed that cortex occupies only 45–55 per cent of the distance beyond the calyces, the remainder being medullary substance. In most diseases, changes in renal size do not affect corticomedullary ratio, indicating parallel changes in both components. Some cases of sickle cell anemia showed paradoxical renal enlargement with cortical thinning and reduced corticomedullary ratio.

Nonparasitic Inflammatory Diseases of the Soft Tissues. William C. Lang, Jr., M.D., J. Luther Clements, Jr., M.D., and H. Stephen Weens, M.D., Emory University School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

A series of roentgenograms was presented to illustrate the commonly seen, but not infrequently overlooked, findings in inflammatory processes involving the soft tissues. The included cases ranged from cellulitis to diabetic gangrene. Particularly emphasized were the diagnosis of osteomyelitis before any bone change, and the diagnosis and progression of emphysematous infections.

The presentation was intended to rekindle awareness of the information available in the soft tissue areas of the roentgenogram.

Ureteral and Pelvic Striations. Robert L. Lebowitz, M.D., and N. Thorne Griscom, M.D., Children's Hospital, Boston, Masschusetts.

Pelvo-ureteral striations usually represent redundant folds caused by reflux. Their location in a duplicated system helps to distinguish vesico-ureteral from ureteroureteral reflux. Striations also occur in infection and relieved obstruction.

Transfemoral Approach to Epidural Venography. James Robert LePage, M.D., Freddie Patrick Gargano, M.D., and Al-

fred Philip Coccaro, M.D., Department of Radiology, University of Miami, School of Medicine, Miami, Florida.

This is an original technique which, since its inception in 1967, has replaced intra-osseous venography in the investigation of thoracolumbar disease at the University of Miami School of Medicine.

The diagnostic accuracy of myelography in a compiled series of 1,472 cases was 84 per cent correct. The diagnostic accuracy of venography by any technique reported in the literature (surgically confirmed cases only) is 91 per cent correct. The confirmed accuracy of the Exhibitors' series of epidural venography was 91 per cent correct.

The transfemoral ascending lumbar catheter approach to epidural venography was presented as an adjunctive procedure in the diagnosis of thoracolumbar disease with more consistent results than intra-osseous venography and with results equally as good, if not better, than the diagnostic accuracy of myelography.

Angiographic Changes in Aortocoronary Vein Grafts: Lack of Progression after the First Year. J. Lespérance, M.D., M. G. Bourassa, M.D., R. Petitclerc, M.D., and J. Saltiel, M.D., Institute of Cardiology of Montreal, Montreal, P.Q., Canada.

All vein grafts undergo morphologic changes with time. These changes, characterized histologically by intimal fibrous hyperplasia, are noted in all cases after the first month.

On the angiogram at 1 year, intimal fibrosis is accompanied by a diffuse or segmental reduction in caliber of the vein grafts; this usually leads to slight or moderate narrowing, but may at times cause complete occlusion of vein grafts.

Serial angiographic studies carried out by the Exhibitors 2 weeks, 1 year and 3 years after operation showed that the angiographic changes do not progress after the first year. This suggests that intimal fibrosis hyperplasia is a self-limited process.

Pneumogynography in Childhood and Adolescence. Patrick D. Lester, M.D., and William H. McAlister, M.D., University of Utah College of Medicine, Salt Lake City, Utah, and Mallinckrodt Institute of Radiology, St. Louis, Missouri.

Several examples of pelvic pneumogynography were illustrated, focusing on the indications for this procedure in the pediatric patient and its value in evaluating endocrine disorders and pelvic masses. A relatively safe procedure, it can assist in the diagnosis of these occasional problems, often avoiding exploratory surgery or pelvic examination under anesthesia.

The techniques for performing pelvic pneumogynography in children were described.

The Radiology of Hypertension in Infancy and Childhood. William J. McSweeney, M.D., Frances M. Unger, M.D., and Jennifer M. H. Loggie, M.B., B.Ch., Cincinnati Children's Hospital, University of Cincinnati, Cincinnati, Ohio.

Forty-seven infants and children underwent extensive clinical and roentgenologic evaluation for sustained diastolic hypertension. The roentgenologic evaluation included urography and aortography in all cases, and selective renal angiography when the aortograms were nondiagnostic. Selective renal vein renin sampling was successful in 37 of the 39 patients in whom it was attempted. All but a few of the arteriotomies and venotomies were accomplished with percutaneous techniques, even in the infants less than 1 year of age.

The Exhibitors' results showed that sustained hypertension of greater than 90 mm. Hg in all pre-teenage children and white females less that 20 years of age should have a complete evaluation including angiography and selective renal vein renin sampling, since idiopathic (essential) hypertension was rarely found in these groups. Urography including hypertensive intravenous pyelography cannot be considered an adequate roentgenologic evaluation. Although urography defined associated renal diseases, it failed to define most of the renovascular conditions seen in this population. Renal vein renin ratios of

greater than 2 to 1 had good predictive value in the 4 children who underwent nephrectomy. Since significant hypertension was an incidental finding in over half of the study population, it would seem appropriate to consider a blood pressure determination to be part of the routine physical examination of any child over the age of 2 years. Blood pressure determinations on infants with congestive heart failure of unknown etiology should also be routine, as 4 of the Exhibitors' cases presented in this manner.

Arterial Insufficiency of the Leg Secondary to Acute Muscular Swelling. Rogelio Moncada, M.D., Robert J. Freeark, M.D., Neil Yeston, M.D., and Arthur Howell, M.D., Loyola University of Chicago, Maywood, Illinois.

Acute muscle swelling occurs as a result of strenuous exercise, arterial revascularization and trauma. When the increased muscle mass exceeds the capacity of the involved rigid osseo-fascial compartment, vascular insufficiency may result.

Confirmation by arteriography and prompt surgical decompression averts permanent limb damage.

The Partial Pneumoencephalogram for following Treated Pituitary Tumors. William W. Olmsted, M.D., Robert W. Rand, M.D., Gabriel H. Wilson, M.D., and John P. Gartland, M.D., UCLA Hospital, Los Angeles, California.

Recurrence of nonsecretory pituitary tumors is difficult to evaluate because biochemical parameters are not available and eye signs are often unreliable.

Recently at UCLA Hospital, "limited" pneumoencephalographies have been performed on post-therapy pituitary tumor patients. The "limited" approach allows evaluation of the parasellar and suprasellar areas within weeks of therapy, thus establishing a baseline. The patient is then restudied as an outpatient at specific intervals, *e.g.*, every 12 to 18 months, to determine if there has been recurrence of tumor.

Rationale for the procedure, technique and illustrative cases were presented.

Chronic Bronchitis: A Combined Bronchographic Pathologic Study. John Olsen, M.D., Martha Warnoc, M.D., and Axel Kunzmann, M.D., The University of Chicago Hospitals, Chicago, Illinois.

The *in vivo* bronchographic findings of chronic bronchitis were demonstrated. Postmortem bronchograms with detailed pathologic studies illustrated the specific anatomic pathologic correlations in mild and severe disease.

The Anterior Meningeal Branch of the Vertebral Artery in Extra-Axial Posterior Fossa Lesions. Harold Pachtman, M.D., Robert L. Waldron, II, M.D., Sadek Hilal, M.D., and Ernest H. Wood, M.D., University of Kentucky Medical Center and The Neurological Institute, Lexington, Kentucky; and New York, New York.

The subject matter consisted of a number of roentgenograms, descriptions and diagrams showing the anterior meningeal branch of the vertebral artery in various extra-axial tumors. Although this vessel is usually not seen on angiograms, its significance when present or enlarged was discussed.

Prominence of the anterior meningeal branch of the vertebral artery was demonstrated in a number of posterior fossa lesions. Among these, involvement in chordoma, arteriovenous malformation, traumatic arteriovenous fistula and schwannoma has not been previously reported.

Oral Examination of the Colon. Rubem Pochaczevsky, M.D., City Hospital Center at Elmhurst and the Mount Sinai Medical School, New York, New York.

In this exhibit, an alternate method of examination of the colon was described, whereby the colon was studied by means of the oral administration of a barium and sorbitol mixture.

Most of the well known shortcomings of the follow-through study have been eliminated with the new technique described and a reliably good quality visualization of the entire colon could be obtained in the large majority of cases.

The "Self-Reporting Radiograph." A New Pictorial Dimension to the Radiologist's

Report. Rubem Pochaczevsky, M.D., City Hospital Center at Elmhurst and the Mount Sinai Medical School, New York, New York.

The "Self-Reporting Radiograph" is a new system of re-enforcing formal reports by supplementing them with 100 mm. ×100 mm. direct positive copies of salient radiographs annotated by the radiologist. The 100 mm.×100 mm. copy is secured to a report form, sent to the chart, and becomes a permanent part of the patient's record.

The merits and the impact of this new system were discussed.

Spontaneous Renal Hemorrhage: Roentgenographic Manifestations. Howard M. Pollack, M.D., and George Popky, M.D., Radiology Department, Episcopal Hospital and Hospital of the Medical College of Pennsylvania, Philadelphia, Pennsylvania.

Nontraumatic renal bleeding may result from a variety of causes, the most important of which is tumor.

This exhibit illustrated several of these causes, and their varied roentgenographic presentations, including intrarenal, subcapsular and perirenal hematomas, renal cell carcinoma, polyarteritis and vasculitis.

Morphologic Analysis of Foregut Cysts of the Thorax. James C. Reed, M.D., and Richard E. Sobonya, M.D., Armed Forces Institute of Pathology, Washington, D.C.

Foregut cysts of the thorax are histologically subdivided into bronchogenic, enteric, and mixed types.

Over 100 cases were statistically compared along the following parameters: density; border; architecture; texture; shape; locus; size; number; and associated bone abnormalities.

The analysis emphasized the importance of using the roentgenograms of the gross specimen both to assist the preoperative differential diagnostic approach and to help reveal the context for the histologic diagnosis.

There was a scientific essay on the same subject.

The Value of Bronchography in Bronchogenic Carcinoma. Louis Michael Rogow, M.D., Department of Radiology, New York Medical College, New York, New York.

The 6 bronchographic signs of bronchogenic carcinoma were described. Various examples of the described diagnostic criteria were illustrated. The accuracy of bronchography (89–94 per cent) was compared to the accuracy of other available diagnostic tests.

Coronary Arteriography and Bypass Graft Surgery—the Appraisal of Distal Vessel Operability. Josef Rösch, M.D., Charles T. Dotter, M.D., Ruza Antonovic, M.D., Lawrence Bonchek, M.D., and Albert Starr, M.D., University of Oregon Medical School, Portland, Oregon.

The accuracy of preoperative coronary arteriography in determining distal vessel operability was evaluated in 166 patients studied arteriographically before and after bypass surgery. Appraisal accuracy correlated closely with the type of distal vessel filling, being highest (97 per cent) in poststenotic vessels with antegrade filling; lower (76 per cent) beyond occlusions with generous retrograde collateral flow; and lowest (65 per cent) in postocclusion vessels filled by insufficient collaterals.

Individual points of angiographic appraisal, mainly vessel size, disease beyond obstruction, peripheral runoff, and myocardial contractility were discussed and suggestions made for improving the preoperative evaluation of vessel suitability for coronary bypass grafting.

Electronic Subtraction and Color Transformation of Visceral Angiograms Based on 325 Studies. F. J. Roth, M.D., K. Ranniger, M.D., and W. Wenz, M.D., Medical College of Virginia, Richmond, Virginia.

The value of subtraction for cerebral angiography is evident. Subtraction used for visceral angiography, however, is technically more difficult because of respiratory motion as well as peristaltic movements of the bowel.

Based on the evaluation of 325 visceral angiograms, it was shown that by electronic subtraction small vessels can be seen

better, the venous system is more prominently outlined and frequently the parenchyma of the pancreas is well demonstrated. Transformation of subtracted pictures into color films represents a further aid in the interpretation of visceral angiograms.

Although subtraction and color radiography do not increase the information contained on a film, they do improve the visibility of certain structures difficult to extract from black and white nonsubtracted roentgenograms. Therefore, the evaluation is easier and might be more accurate.

Congenital Heart Disease in Adult Patients. Jacques Saltiel, M.D., Robert Petitclerc, M.D., and Jacques Lespérance, M.D., Montreal Heart Institute, Montreal, P.Q., Canada.

Congenital heart diseases are sometimes encountered in adult patients. When a murmur is discovered in an adult, the referring physician may send the patient to the radiology department with a diagnosis of acquired disease. Using chest roentgenograms and heart fluoroscopy, the radiologist can make the proper diagnosis, if he is aware of these conditions.

As a reminder, some cases from the radiologic teaching file of the Montreal Heart Institute were presented. Emphasis was made on the plain roentgenograms and chest fluoroscopic findings in this group of patients. These findings were correlated with angiography, hemodynamic studies, and surgery.

Extravasation of Urine in Obstructive Uropathy. Lewis C. Santini, M.D., Arthur F. Kriner, M.D., and John L. Williams, M.D., Geisinger Medical Center, Danville, Pennsylvania.

Extravasation of urine from small forniceal tears can be seen in up to 5 per cent of patients with acute renal colic caused by an obstructing ureteral calculus. Many cases are probably not detected because the extravasation is often subtle and may be intermittent. The urine dissects along the renal pelvis and ureter, loculates near the kidney or enters the vascular system. There is often little or no hydronephrosis in patients with extravasation, suggesting that the tears act as release valves.

The course is usually benign. Treatment is directed toward the offending calculus. The greatest hazard of extravasation is an infected urine. Catheterization and retrograde pyelography should thus be avoided since they invite infection and the development of abscesses.

The Radiology of Diffuse Airway Disease. Robert F. Scheible, M.D., John V. Forrest, M.D., and Stuart S. Sagel, M.D., Mallinckrodt Institute of Radiology, St. Louis, Missouri.

Plain film roentgenographic, tomographic, and bronchographic manifestations of diffuse airway disease were shown with an emphasis on less common disease entities.

Processes affecting the trachea and main stem bronchi include tracheobronchomegaly, chronic mediastinitis, and amyloid disease.

The intermediate sized airways are involved in sarcoid, asthma, bronchitis, and bronchiectasis including that due to cystic fibrosis, after inhalation or aspiration of noxious agents, and mucoid impaction.

Smaller airway diseases include bronchiolitis obliterans, the Swyer-James syndrome, and spasm due to pulmonary embolism.

The High K.V. Chest—Some Practical Points in Setting Up the Unit. D. James Sieniewicz, M.D., and Earl Paquette, R.T., St. Michael's Hospital, Toronto, Ontario, Canada.

The advantage of high K.V. (140–160 kev.) chest roentgenograms as compared with the standard chest technique was demonstrated.

Despite the advantages of the "Chest Units" now manufactured, there are still many technical variables which can be controlled by the radiologist. The Exhibitors found that there was a paucity of information available regarding some practical points which were demonstrated in

this exhibit. How they handled the problem of scatter and how they improved definition by trying different combinations of added tube filtration, grids, air gap distance, high contrast screens, and films were demonstrated.

Bowed Limbs at Birth: A Differential Diagnosis. Richard R. Six, M.D., William M. Thompson, M.D., and Herman L. Grossman, M.D., Duke University Medical Center, Durham, North Carolina.

Bowed limbs at birth have been described as isolated findings, as well as in association with other anomalies. Several different forms of bone dysplasias manifested by bowed limbs are recognizable at birth, and may be differentiated clinically and roentgenographically.

Camptomelia, hypophosphatasia, osteogenesis imperfecta, prenatal bowing of long bones and thanatophoric dwarfism comprise this group. The first 3 entities are heritable, prenatal bowing is developmental, and thanatophoric dwarfism is a lethal mutation. Although these entities are rare, definitive diagnosis is necessary for proper management and genetic counseling.

The important manifestations aiding in this differential diagnosis were illustrated.

Perinephric Masses—a Cause for the Page Kidney Phenomenon. Thomas A. Sos, M.D., and Harold A. Baltaxe, M.D., Cornell University Medical Center, New York, New York.

Renovascular hypertension produced by compression of the renal parenchyma, the "Page Kidney" phenomenon, is not widely recognized.

This compression produces an effective increase in renovascular resistance decreasing the renal blood flow, thus reproducing the physiologic equivalent of the "Goldblatt Kidney."

Extrarenal, perinephric, and subcapsular masses may be responsible for the compression, and their removal or nephrectomy relieves the hypertension.

The case material included perinephric hematoma and subcapsular hematoma. The roentgen findings were demonstrated.

Patterns of Lobar Collapse in the Dog Studied by Whole-Body Freezing. Lawrence A. Stein, M.D., Theresa C. McLoud, M.D., James C. Hogg, M.D., and Robert G. Fraser, M.D., Royal Victoria Hospital, McGill University, Montreal, P.Q., Canada.

There is indirect evidence of the precise shape and orientation of collapsed pulmonary lobes.

The Exhibitors collapsed O_2 filled lobes of the 2 lungs by endobronchial obstruction. When gasless, the animal was sacrificed, frozen whole, and its thorax sectioned into 2 cm. slices.

Interrelationships between the collapsed lobe, the remainder of the lung, and chest wall were observed.

The precise form of a collapsed lobe is very similar to that of lobar atelectasis described. Volume and wet-to-dry weight ratios lead to the conclusion that the "collapsing" lobe becomes rapidly gasless not only by absorption of gas, but also from partial replacement by edema fluid.

Gallium 67—A Nuclide with Multiple Uses. Samuel Saunders Steinberg, M.D., Gerald D. Wood, M.D., and Chris V. Payton, R.T., Department of Nuclear Medicine, Memorial Hospital of Southern California, Culver City, California.

Originally, gallium 67 was successfully utilized in the staging of lymphoma. In 64 consecutive cases, in addition to the lymphoma, the Exhibitors found localization of the nuclide in the following (A) neoplastic and (B) benign conditions: (A) 1. adenocarcinoma; 2. squamous cell carcinoma; 3. transitional cell carcinoma; 4. hemangiosarcoma; 5. fibrosarcoma; 6. malignant pleural fluid; and (B) 1. inflammatory processes; 2. recent operative sites; 3. abscesses.

For meaningful conclusions, each gallium 67 examination must be evaluated in conjunction with the total clinical, laboratory and other findings.

The full potential of this interesting nuclide has not yet been realized.

Pathophysiology of Congenital Megacalyces. Lee B. Talner, M.D., and Ruben F. Gittes, M.D., University of California, San Diego, California.

Megacalyces is a congenital nonobstructive enlargement of the calyces due to a malformation of the renal papillae. The renal cortex is normal but the medulla is hypoplastic, resulting in a variable, mild reduction in maximal renal concentrating ability. Because of stasis there is a predisposition to stone formation and infection. The dilated calyces and reduced parenchymal thickness may resemble obstructive hydronephrosis or postobstructive atrophy, but the urographic and angiographic features of megacalyces are sufficiently distinctive for the correct diagnosis to be made in most cases.

Renal function tests and radioisotope studies aid in confirming the diagnosis.

There was a scientific essay on the same subject.

A New Simplified Method for Cannulating Lymphatic Ducts. Charles John Tegtmeyer, M.D., University of Virginia Medical Center, Charlottesville, Virginia.

A simplified technique for cannulating lymphatic vessels utilizing a new lymph duct cannulator was illustrated. Once a suitable duct has been isolated, the cannulator automatically directs the needle into the lumen of the duct while holding the vessel firmly in place.

The lymph duct cannulator method is easier to teach and is mastered more quickly than existing free-hand procedures of lymph duct cannulation. It has been tested successfully in 119 out of 122 attempts on individual extremities.

The cannulator has been manufactured and is readily available.

Alveolar Hydatid Disease, a Comparison with Cystic Hydatid Disease. William M. Thompson, M.D., Dan P. Chisholm, M.D., and Arvin E. Robinson, M.D., Department of Radiology, Duke University Medical Center, Durham, North Carolina.

Three forms of hydatid disease due to the larval stage of Echinococcal tapeworms have been reported: alveolar hydatid disease (*E. multilocularis*); and sylvatic and pastoral cystic hydatid disease (*E. granulosus*).

Patients with both forms of cystic hydatid disease have cysts in the lungs and liver as commonly described. In alveolar hydatid disease, which is gaining recognition in North America, the larvae grow exogenously, invading and destroying the affected organ (usually liver). Roentgenographically, heptomegaly, multicystic hepatic calcification, and filling defects on liver scan are demonstrated.

The exhibit illustrated these roentgenographic findings and the differences in life cycles, endemic location, morphology, host (clinical) response, and site of involvement.

The Michelangelo Models. Gordon E. Trueman, M.D., Elwood C. Wylie, R.T., and James F. Walker, R.T., University of British Columbia, Vancouver General Hospital, Vancouver, British Columbia, Canada.

Michelangelo made small models in sculpture as conceptual guides for definitive larger works. He destroyed most of the models, presumably to prevent others from using them. Of the few models which survive, most are in the Vancouver collection and are valued at several million dollars.

The exhibit displayed color transparencies for their beauty alone, and showed corresponding roentgenograms for analysis of the artist's technique, for proof of identity, and for study of damage and repair.

Radiologic Diagnosis of Carcinoma of the Biliary Tree. Robert L. Waldron, II, M.D., and Gerard Kenny, M.D., Mount Auburn Hospital, Harvard Medical School, Cambridge, Massachusetts, and College of Physicians and Surgeons of Columbia University, New York, New York.

The radiographic findings of carcinoma of the biliary tree were demonstrated, emphasizing percutaneous transhepatic cholangiography (PTC). The method was described in detail and the complications and

success rate in the over-all series of 31 patients were listed. It has been valuable in the diagnosis as well as the surgical management in the jaundiced patient, especially those with carcinoma of the biliary tree.

Selective celiac arteriograms in 3 patients demonstrated a tumor stain in the capillary phase. The Exhibitors' experience indicates that it is possible to differentiate tumors of bile duct origin from primary tumors in the head of the pancreas.

The 2 techniques complement each other.

Renal Dialysis Shunts: Improved Angiographic Evaluation. Alan Furness White, M.D., Louis D. Napoli, M.D., and Homer L. Twigg, M.D., Department of Radiology, Georgetown University Hospital, Washington, D.C.

Previous methods required arterial and venous injections in several projections. A cassette was placed under the patient's arm for each exposure.

The present method uses a blood pressure cuff, a rapid film changer and a single injection of contrast material into the arterial side, and then the venous side of the shunt. Multiple roentgenograms in 3 projections are obtained. The entire examination is completed in 5 minutes.

The advantages of this method include:

1. *Safety.* The use of a blood pressure cuff protects against retrograde embolization of a clot into the vertebral artery.
2. *Less pain for the patient.* Only a single arterial injection of contrast material is required.
3. *Excellent quality roentgenograms.* Multiple views are obtained with the film changer, allowing dynamic evaluation of any abnormality without superimposed vessels.

There was a scientific essay on the same subject.

Differential Diagnosis of Skull Fracture. J. Powell Williams, M.D., Charles I. Weiner, M.D., Marvin P. Rozear, M.D., and G. Thomas Gilmore, M.D., Department of Radiology, University of Maryland, Baltimore, Maryland.

Despite studies demonstrating the low yield and high cost of routine skull roentgenograms in patients with minor head trauma, medicolegal pressures have caused continual increase in the number of examinations ordered. Even though uncomplicated skull fractures in the absence of brain damage are not treated, the courts have decided that good medical care requires their roentgenographic demonstration.

This exhibit illustrated the optimum plain film roentgenographic examination of the skull following head trauma and the various entities which may mimic skull fracture. These were classified as: I. Artifacts (a) roentgenographic, (b) nonroentgenographic; II. Sutures; III. Vascular grooves.

Suspected Acute Extensive Bowel Ischemia —the Role of the Radiologist. Jack Wittenberg, M.D., Christos A. Athanasoulis, M.D., Lester F. Williams, Jr., M.D., and Jerome H. Shapiro, M.D., Boston City Hospital, Boston, Massachusetts; and Boston University School of Medicine, Boston, Massachusetts.

Twenty-eight patients clinically suspected of having extensive acute bowel ischemia were studied and a comprehensive roentgenologic approach developed. Its purpose was to differentiate between the multiple etiologies of bowel ischemia and to diagnose other abdominal diseases with a similar clinical presentation.

Angiography, performed on all patients, distinguished between an occluded and nonoccluded mesenteric circulation. In the latter group, immediate gastrointestinal barium examinations were performed attempting to separate patients with nonocclusive bowel ischemia from those *without* ischemic disease *at all.*

The scheme allows rapid and specific localization of occlusive mesenteric vascular disease, replaces an exploratory laparotomy for the diagnosis of the nonocclusive ischemic state, and suggests a new

modality for treating patients with the low mesenteric flow state.

Analysis of the Radiographic and Related Features of Breast Carcinoma. John N. Wolfe, M.D., Hutzel Hospital, Detroit, Michigan.

Breast carcinomas were analyzed and the features tabulated to demonstrate the changes that appear on the radiographic image inherent in the cancer itself and also those in the breast itself as generalized changes.

Some correlation with the histologic features was also presented.

A scientific essay was presented on the same subject.

The Value of Arteriography in Benign and Malignant Lesions. Issa Yaghmai, M.D., Associate Professor of Radiology, Tehran Medical School, Tehran, Iran; Visiting Professor of Radiology, Medical College of Virginia, Richmond, Virginia.

Arteriography was carried out without mortality or morbidity in 156 selected bone lesions in the Radiology Department of Tadj Pahlavi Hospital in Tehran, Iran.

Visualizing the vascular patterns made it possible, in most instances, to distinguish accurately between benign and malignant bone lesions. It was used to show the intramedullary extent as well as the extent of the tumor outside of the bone—an exact delimitation helpful in determining treatment. Knowledge of the vascular patterns was used in locating viable tumor tissue from which a biopsy could be taken.

Arteriography also demonstrated the effect of radiation therapy on a given malignant bone tumor. Used in conjunction with conventional radiographic procedures, arteriography can improve both the accuracy of diagnosis and the effectiveness of treatment in bone neoplasms.

Review and "Self Evaluation" in Radiology. Peter Zanca, M.D., Michael Howard, M.D., Antonio C. Gonzalez, M.D., Robert Waggener, Ph.D., Ben Logan, M.D., Alvin Woldman, M.D., Robert Young, D.V.M., Roy Keenan, Ph.D., and Thomas J. Cody, M.D., The University of Texas Medical School at San Antonio, San Antonio, Texas.

This scientific exhibit consisted of a series of 2×2 inch slides representing related teaching radiologic subjects that were shown with built-in carrousel projectors.

Diagnostic radiology, radiation therapy, nuclear medicine, specialized techniques, radiation physics, and animal laboratory experimentation were covered. For each of the subject areas, 15 to 20 questions and answers were given on different, interesting and educational radiologic cases, stressing the method of diagnosis and the therapy for each case, as indicated.

Research slides presented animal experimentation on intravenous pancreatography with radioisotope scanning.

James F. Martin, M.D.
Chairman of the Committee on Scientific Exhibits

THE TECHNICAL EXHIBITS

The Technical Exhibits of the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, held at the Queen Elizabeth Hotel with its ultramodern facilities, September 25–28, 1973, were located, conjoint with the Scientific Exhibits, on the spacious Convention Floor, in the immediate vicinity of Le Grand Salon, where the Scientific Sessions of the Society were held. This fortunate arrangement afforded the members and many guests of the Society a unique opportunity to view the magnificent and most artistically displayed latest progress in the technical aspect of the science of Radiology—a factor which enhanced immeasurably the over-all success of the Annual Meeting.

The Society is greatly indebted to Dr. James C. Cook, Manager and Dr. George A. Kling, Associate Manager of the Annual Meeting, who assembled and organized these splendid exhibits and expresses its sincerest thanks, and deep appreciation to the following participating firms:

AGA Corporation, Secaucus, New Jersey; Association of International Medical Sciences, Princeton, New Jersey; Atomic Energy of Canada Limited, Ottawa, Ontario; Barber-Colman Company, Rockford, Illinois; Barnes-Hind Pharmaceuticals, Inc., Sunnyvale, California; Bar-Ray Products, Inc., Brooklyn, New York; Becton-Dickinson, Rutherford, New Jersey; CGR Medical Corporation, Baltimore, Maryland; Cook Incorporated, Bloomington, Indiana; Cordis Corporation, Miami, Florida; Dunlee Corporation, Bellwood, Illinois; Du Pont Company, Wilmington, Delaware; Eagle Enterprises, Metuchun, New Jersey; Eastman Kodak Company, Rochester, New York; Eimac Division of Varian, Salt Lake City, Utah; Electro-Catheter Corporation, Rahway, New Jersey; Elema-Schonander, Inc., Elk Grove Village, Illinois; E-Z-Em Company, Westbury, New York; Hewlett-Packard, McMinnville, Oregon; GAF Corporation, New York, New York; General Electric Company, Medical Systems Division, Milwaukee, Wisconsin; Warren H. Green, Inc., St. Louis, Missouri; Ilford Inc., Paramus, New Jersey; Katum Corporation, Longmont, Colorado; Keuffel & Esser X-Ray, Inc., Teterboro, New Jersey; Lear Siegler, Inc., Elyria, Ohio; Liebel-Flarsheim Company, Cincinnati, Ohio; LogEtronics Inc., Springfield, Virginia; Low X-Ray, Valhalla, New York; The Machlett Laboratories, Inc., Stamford, Connecticut; Mallinckrodt, St. Louis, Missouri; Medi-Tech, Watertown, Massachusetts; Medrad Incorporated, Allison Park, Pennsylvania; Memco, Bristol, Pennsylvania; The C. V. Mosby Company, Ltd., Toronto, Ontario; N & H Instruments, Inc., Carrollton, Texas; Neutron Products, Inc., Dickerson, Maryland; Pako Corporation, Minneapolis, Minnesota; Picker Corporation, Cleveland, Ohio; Radx Corporation, Houston, Texas; S & S X-Ray Products, Inc., Brooklyn, New York; W. B. Saunders Company Canada Limited, Toronto, Ontario; Frank Scholz X-Ray Corporation, Needham Heights, Massachusetts; Siemens Corporation, Iselin, New Jersey; Spectrum X-Ray Corporation, Westville, New Jersey; Springer-Verlag New York, Inc., New York, New York; Squibb Hospital Division, E. R. Squibb & Sons, Inc., Princeton, New Jersey; Texas Instruments Incorporated, Houston, Texas; Charles C Thomas, Publisher, Springfield, Illinois; Vanguard Instruments Corporation, Melville, New York; Williams & Wilkins Company, Baltimore, Maryland; Wolf X-Ray, Jamaica, New York; Xerox Corporation, Pasadena, California; Year Book Medical Publishers, Chicago, Illinois.

NEWS ITEMS

NEW OFFICERS OF THE AMERICAN ROENTGEN RAY SOCIETY

At the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, held at the Queen Elizabeth Hotel, Montreal, P.Q., Canada, September 25–28, 1973 the following new officers were elected: *President:* William B. Seaman, M.D., New York, New York; *President-Elect:* Colin B. Holman, M.D., Mayo Clinic, Rochester, Minnesota 55901; *1st Vice-President:* Walter M. Whitehouse, M.D., Ann Arbor, Michigan; *2nd Vice-President:* Roderick L. Tondreau, M.D., Meadowbrook, Pennsylvania; *Secretary:* Ted F. Leigh, M.D., Emory University Clinic, Atlanta, Georgia 30322; *Treasurer:* Joseph D. Calhoun, M.D., 500 South University Avenue, Little Rock, Arkansas 72205.

The Chairman of the Executive Council is Harry Z. Mellins, M.D., 721 Huntington Avenue, Boston, Massachusetts 02115.

The Seventy-fifth Annual Meeting of the Society will be held at the Hilton Hotel, San Francisco, California, September 24–27, 1974.

SOCIETY FOR PEDIATRIC RADIOLOGY

At the Sixteenth Annual Meeting of the Society for Pediatric Radiology held at the Queen Elizabeth Hotel, Montreal, P.Q., Canada, September 20–21, 1973 the following officers were elected: *President:* Hooshang Taybi, M.D., Oakland, California; *President-Elect:* John L. Gwinn, M.D., 4650 Sunset Boulevard, Los Angeles, California 90027; *Secretary-Treasurer:* John P. Dorst, M.D., 601 North Broadway, Baltimore, Maryland 21205.

The Seventeenth Annual Meeting of the Society will be held at the Hilton Hotel, San Francisco, California, September 22–23, 1974.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

At the last annual meeting of the Rocky Mountain Radiological Society, new officers for 1974 were elected as follows: *President:* W. Kemp Absher, M.D., Pueblo, Colorado; *President-Elect:* Edward E. Tennant, M.D., Sterling, Colorado; *1st Vice-President:* Lowell H. Hansen, M.D., Denver, Colorado; *2nd Vice-President:* George C. Babcock, M.D., Denver, Colorado; *Secretary-Treasurer:* Lorenz R. Wurtzebach, M.D., Denver, Colorado; and *Historian:* H. Milton Berg, M.D., Bismarck, North Dakota.

The next Annual Meeting of the Society will be held in Denver at the Brown Palace Hotel, August 22–24, 1974.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS

The Association of University Radiologists wishes again to announce an annual Memorial Medal Award that will be given to the radiology resident writing the best original paper. The paper can represent the work of one or more investigators, but the resident submitting the paper must qualify as the senior author.

Manuscripts to be considered for the 1974 competition must be received by March 1, 1974. Inquiries should be addressed and papers submitted to: Richard H. Greenspan, M.D., Department of Radiology, Yale University School of Medicine, New Haven, Connecticut 06510.

The winner of the competition will be invited to present his work at the Annual Meeting of the A.U.R. and will receive a monetary award in addition to the medal.

The next A.U.R. annual meeting will be in New York, New York, May 8–11, 1974.

RADIOLOGY/ASPEN

The Fourth Annual Aspen Radiology Conference will be held March 4–8, 1974, under the auspices of the Aspen Institute for Humanistic Studies, Aspen, Colorado.

For further information and conference registration please write to: Emanuel Salz-

man, M.D., Chairman, Division of Radiology, Beth Israel Hospital, West 17th Avenue and Lowell Boulevard, Denver, Colorado 80204.

TUTORIAL POSTGRADUATE COURSE: RADIOLOGY OF THE CHEST

The Department of Radiology, Duke University Medical Center, sponsors a Tutorial Postgraduate Course "Radiology of the Chest," to be held at the Durham Hotel-Motel, Durham, North Carolina, March 25–27, 1974.

The first half day is devoted to basic lectures to the entire group oriented to subsequent tutorial sessions. A detailed abstract booklet with selected references will be provided.

One hour tutorial sessions will then be held, in which 12 registrants will join one faculty member in a separate quiet room with a bank of viewboxes for organized film reading-discussions with 10–12 case presentations on a basic subject or two. There will be 12 of these one hour tutorial sessions over the 3 day period such that each registrant will have one session on Pediatric Radiology, one session on Nuclear Medicine and ten other sessions with each of the Guest and Duke General Diagnosis Faculty covering one or two of the basic subjects.

The maximum registrants accepted are 144 (12 registrants/faculty tutorial ×12).

The Program Chairman is Richard G. Lester, M.D., Professor and Chairman of Radiology, Duke University Medical Center.

The Guest Faculty includes: Robert G. Fraser, M.D., Montreal, Canada; E. Robert Heitzman, Jr., M.D., Syracuse, New York; Theodore E. Keats, M.D., Charlottesville, Virginia; Ted F. Leigh, M.D., Atlanta, Georgia; George Simon, M.D., University of London, England; and William J. Tuddenham, M.D., Philadelphia, Pennsylvania, in addition to the Duke University Medical Center Faculty.

For further information please write Program Director: Robert McLelland, M.D., Department of Radiology, Box 3808, Duke University Medical Center, Durham, North Carolina 27710.

NEW ORLEANS RADIOLOGY SOCIETY

The Spring Roentgen Conference of the New Orleans Radiology Society will be held at the Marriott Hotel in New Orleans on April 25–27, 1974.

The meeting will have several well known guest speakers and the subjects will be general radiology, nuclear medicine, and ultrasound.

For information, please write to: J. L. Mains, M.D., Secretary-Treasurer, New Orleans Radiology Society, P.O. Box 51706, New Orleans, Louisiana 70151.

THE JOHNS HOPKINS MEDICAL INSTITUTIONS

The Second Annual Symposium: "Recent Practical Advances in Diagnostic Radiology and Nuclear Medicine" sponsored by the Department of Radiology and Radiological Science, The Johns Hopkins Medical Institutions, Baltimore, Maryland, will be held at the Turner Auditorium, Johns Hopkins Medical Institutions, May 1 through 4, 1974.

Further information can be obtained from Frederick P. Stitik, M.D., Box 23, Department of Radiology and Radiological Science, The Johns Hopkins Medical Institutions, Baltimore, Maryland 21205.

RADIONUCLIDES IN NEPHROLOGY

The Third International Symposium "Radionuclides in Nephrology" will be held at Hotel Kempinski, West-Berlin, Germany, Federal Republic, April 1–3, 1974.

The main topics will be: renal blood flow; radionuclides in functional and morphologic studies of the kidney (clearance, imaging procedures, renography, etc., including data processing systems); radioimmunoassay in renal diseases (angiotensin, parathormone, aldosterone, prostaglandin, etc.); investigation with radionuclides in follow-up studies after renal transplantation; renal handling of radioactive com-

pounds including autoradiography; radioisotopic methods for studying metabolic disorders due to renal disease.

For more information, please contact Dr. zum Winkel, Klinikum Westend, Strahleninstitut, 1 Berlin 19, Spandauer Damm 130.

THIRD SOUTH AFRICAN NATIONAL AND INTERNATIONAL RADIOLOGICAL CONGRESS

The South African Radiological Society is holding its Third South African National and International Radiological Congress August 29 to September 4, 1974 at the Carlton Hotel, Johannesburg.

The Congress comprises a large eminent international faculty and will discuss radiodiagnosis, nuclear medicine, radiation physics, radiotherapy and other forms of diagnostic imaging.

For further information please contact Doctor Paul Sneider, Congress Director, P.O. Box 4878, Johannesburg, South Africa.

FIRST ASIAN REGIONAL CONGRESS ON RADIATION PROTECTION

The First Asian Regional Congress on Radiation Protection will be held in Bombay, during December 1974, under the auspices of the Indian Association for Radiation Protection. The Congress is sponsored by the International Radiation Protection Association.

The theme of the Congress will be "Regional Cooperation in the Field of Radiation Protection." The Congress will have a number of plenary sessions in which subjects of general interest will be discussed. These subjects will include, among others, the following: organization of radiation protection services on a countrywide scale; significant experiences and problems faced in radiation protection with reference to applications of ionising radiations and nuclear technology in various fields; education and training programs and facilities for radiation protection; general problems of supply and maintenance of radiation protection equipment and instruments; mutual assistance in radiation emergencies; radiation protection legislation.

In addition, it is proposed to have technical sessions in which topics of special interest will be discussed.

For further information please write to: S. Somasundaram, Secretary, IARP, c.o. Body Burden Measurement Section, Health Physics Division, Bhabha Atomic Research Centre, Old Yacht Club, C.S.M.Marg, Bombay 400 001.

BOOK REVIEW

Disproportionate Short Stature: Diagnosis and Management. By Joseph A. Bailey, II, M.D., F.A.C.S., Consultant in Orthopaedic Genetics, Medical Center of El Monte, Hospital and Clinic, El Monte, Calif.; Consultant in Orthopaedic Genetics, Hospital for Joint Diseases and Medical Center, New York, N. Y. Cloth. Pp. 589, with 301 illustrations. Price, $22.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

Accurate diagnosis for patients of short stature is desirable not only to further knowledge, but also to permit reliable genetic counseling and to provide an accurate prognosis for the patient and his family. Some disorders of growth can be definitively diagnosed by biochemical tests or by chromosomal analysis; however, for most of these diseases we have reached only the stage of describing their morphology. As the bones are often malformed, sometimes in a fairly specific way, the radiologist is often called upon to offer a diagnosis based on roentgenograms of a patient with short stature.

The difficulty of making a diagnosis stems from the large and ever-increasing number of syndromes which have to be considered, and the horrendous confusion in their classification and nomenclature. Dr. Bailey offers a book which is helpful in that it deals with a great many disorders associated with short stature. One hundred different conditions are discussed, albeit some quite briefly, as causes of disproportionate short stature, that is dwarfing due to abnormal shortness of the trunk or of the limbs. Some of these diseases have been reported as affecting only a few persons; these particular reports are unlikely to come to the attention of any but specialists in this field. Moreover, Dr. Bailey is up to date in his references.

The clinical descriptions of the commoner syndromes are divided into sections pertaining to the findings at different stages in development, from infancy to adulthood, which is an effective way of presenting this information. Prominent roentgenographic findings are described, but often with less detail than the standard textbooks of x-ray diagnosis. Reproduction of the roentgenograms is adequate, but no better. Treatment of such conditions for which any is available is briefly discussed as is the mode of inheritance. The sections on some syndromes, particularly the metaphyseal dysostoses, the multiple epiphyseal dysplasias and the spondylo-epiphyseal dysplasias are expansive and detailed. These sections alone make the book worth having, but in his effort to be all-inclusive, Dr. Bailey has added short chapters on fragile bone diseases, scoliotic syndromes and mineral metabolic disturbances which are so inadequate that they detract from the remainder.

The considerations of disproportionate short stature make up the bulk of the book. At the end are tabulated 200 disorders associated with proportionate short stature each with a dozen or so words of clinical description and a reference.

In all, Dr. Bailey provides an introduction to short stature syndromes which is exceptionally comprehensive in its coverage, although only a few conditions are described in sufficient depth that further reading would not be necessary. For this purpose an extensive bibliography is provided.

A. H. Woodward, M.D.

BOOKS RECEIVED

Chest Roentgenology. By Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati College of Medicine and Medical Center; Consultant to Cincinnati and Dayton Veterans Administration Hospitals; National Consultant to the Medical Corps of the United States Air Force, Army, Navy and Veterans Administration, United States Public Health Service, Armed Forces Institute of Pathology, and Walter Reed General Hospital. Cloth. Pp. 574, with 518 illustrations. Price, $17.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

Major Problems in Childhood Cancer. Edited by Carl Pochedly, M.D., Director, Pediatric Hematology, Nassau County Medical Center, East Meadow, Long Island, N. Y.; and Thomas F. Necheles, M.D., Ph.D., Chief, Pediatric Hematology, New England Medical Center Hospitals; Associate Professor of Pediatrics, Tufts University School of Medicine, Boston, Mass. With a foreword by Sydney S. Gellis, M.D. Cloth.

Pp. 379, with many illustrations. Price, $14.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

CONFERENCE AND WORKSHOP ON CELLULAR IMMUNE REACTIONS TO HUMAN TUMOR-ASSOCIATED ANTIGENS. Held at National Cancer Institute, Bethesda, Md., June 8 and 9, 1972. National Cancer Institute Monograph 37, June, 1973. DHEW Publication No. (NIH) 73-399. Edited by Ronald B. Heberman and Clarice E. Gaylord. Cloth. Pp. 221, with some figures. Price, $4.85. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, 1973.

RADIOLOGY SPECIAL PROCEDURE ROOM. By Ray A. Brinker, M.D., Department of Radiology, St. Vincent's Hospital and Medical Center, New York, N. Y.; and Jovitas Skucas, M.D., Department of Radiology, Indiana University School of Medicine, Indianapolis, Ind. Cloth. Pp. 188, with some figures. Price, $9.50. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1973.

ENVIRONMENT AND CANCER. A Collection of Papers Presented at the 24th Annual Symposium on Fundamental Cancer Research, 1971. Published for The University of Texas at Houston, M. D. Anderson Hospital and Tumor Institute, Houston, Texas. Cloth. Pp. 475, with some figures. Price, $19.00. Williams & Wilkins Company, 428 East Preston Street, Baltimore, Md., 1972.

HEALTH PHYSICS PROBLEMS OF INTERNAL CONTAMINATION. Proceedings of the IRPA Second European Congress on Radiation Protection, Budapest, May 3–5, 1972. Edited by E. Bujdosó. Cloth. Pp. 655, with many figures. Price, $24.00. Akadémiai Kiadó, Budapest V. Alkotmány U.21, Hungary, 1973.

X-RAY PHYSICS FOR RADIOLOGIC TECHNOLOGISTS. By Richard H. Schmidt, M.D., D.A.B.R., Associate Director, Department of Radiology, Illinois Masonic Medical Center; Associate, Department of Radiology, Medical School, Northwestern University, Chicago, Ill. Cloth. Pp. 143, with some figures. Price, $12.50. Warren H. Green, Inc., 10 South Brentwood Boulevard, St. Louis, Mo. 63105, 1973.

RADIOLOGY FOR THE RADIOLOGIST. By Eric J. Hall, M.A., D.Phil., Professor of Radiology, Columbia University, New York, N. Y. Cloth. Pp. 308, with 115 illustrations. Price, $14.95. Harper & Row Medical Department, 2350 Virginia Avenue, Hagerstown, Md., 1973.

TEXTBOOK OF DENTAL RADIOLOGY. By Olaf E. Langland, D.D.S., M.S., F.A.C.D., Professor and Head, Department of Oral Diagnosis/Medicine/Radiology, School of Dentistry, Louisiana State University, New Orleans, La.; and Francis H. Sippy, B.S., M.Ed., Instructor, Division of Dental Radiology, College of Dentistry, University of Iowa, Iowa City, Iowa. Cloth. Pp. 380, with many illustrations. Price, $17.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

COMPUTERS IN RADIATION THERAPY. Proceedings of the Fourth International Conference of the Use of Computers in Radiation Therapy, Uppsala, Sweden, Aug. 7–11, 1972. Edited by Arvin Glicksman, New York; Jan Cederlund, Uppsala; Montague Cohen, London; Jack Cunningham, Toronto; Bo Jung, Uppsala; Bengt Olsen, Uppsala; and Stewart Orr, Glasgow. Paper. Pp. 300, with 30 illustrations. Price, Sw.Cr.95. Fourth International Conference on the Use of Computers in Radiation Therapy, Radiofysikavdelningen, Akademiska Sjukhuset, S-750 14 Uppsala, Sweden, 1973.

FORMATION OF THE TOMOGRAPHIC IMAGE: WITH SPECIAL REFERENCE TO BLURRING. By Ove Mattsson, Stockholm, Sweden. Paper. Pp. 58, with some figures. Price, Sw.Kr. 35:-. Acta Radiologica Supplementum 318. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1972.

PROGRESS IN VETERINARY RADIOLOGY. Proceedings of the 2nd International Conference of Veterinary Radiologists held in Stockholm, 1970. Edited by Sten-Erik Olsson. Paper. Pp. 303, with many illustrations. Price, Sw.Kr.45:-. Acta Radiologica Supplementum 319. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1972.

CARCINOMA OF THE UTERINE CERVIX: ASPECTS OF CLINICAL ONCOLOGY IN PATIENTS REFERRED FOR RADIATION THERAPY. By Tjakko Kuipers, Rotterdam, The Netherlands. Paper. Pp. 252, with some figures. Price, Sw.Kr. 50:-. Acta Radiologica Supplementum 320. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1972.

ANGIOGRAPHIC ABNORMALITIES FOLLOWING PERCUTANEOUS NEEDLE BIOPSY OF THE KIDNEY. By Bo Lundström, Umeå, Sweden. Paper. Pp. 67, some figures. Price, Sw.Kr. 40:-. Acta Radiologica Supplementum 321. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1972.

MODE OF ACCUMULATION OF IODOPHENYLALANINES IN THE EXOCRINE PANCREAS AND CERTAIN TUMOURS. By Lars Blomquist, Uppsala, Sweden. Paper. Pp. 140, with some figures. Price, Sw.Kr. 40:-. Acta Radiologica Supplementum 322. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1972.

RADIOLOGIC REPORTING. I. Problems of Structuring and Coding Information from Diagnostic Radiology. II. Medela Reporting System. By Inger Brolin, Gothenburg, Sweden. Paper. Pp. 150, with some figures. Price, Sw.Kr. 40:-. Acta Radiologica Supplementum 323. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

THE ROLE OF HOST TISSUE IN SKIN CARCINOGENESIS: AN INVESTIGATION WITH SKIN-TUMOR-RESISTANT AND SKIN-TUMOR-SUSCEPTIBLE MICE. By Timo Telaranta, Helsinki, Finland. Paper. Pp.

74, with some figures. Price, Sw.Kr. 35:-. Acta Radiologica Supplementum 324. Acta Radiologica, Stockholm 2, Sweden, 1973.

Accumulation of Drugs on Melanin. By Nils Gunnar Lindquist, Uppsala, Sweden. Paper. Pp. 92, with many figures. Price, Sw.Kr. 40:-. Acta Radiologica Supplementum 325. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

Hysterography and Diagnostic Currettage in Carcinoma of the Uterine Body: An Evaluation of Diagnostic Value and Therapeutic Implications in Stages I and II. By John-Erik Johnsson, Lund, Sweden. Paper. Pp. 79, with some figures. Price, Sw.Kr. 40:-. Acta Radiologica Supplementum 326. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

Tentorial Notch and Adjacent Major Vessels in Carotid Angiography. By Eric Bergquist, Uppsala, Sweden. Paper. Pp. 129, with some figures. Price, Sw.Kr. 45:-. Acta Radiologica Supplementum 327. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

Angiographic Abnormalities in the Urinary Wall After Irradiation. Part I: Animal Experiments, by O. Hassler and S.-O. Hietala. Part II: Clinical Investigation, by S.-O. Hietala, Umeå, Sweden. Paper. Pp. 117, with some figures. Price, Sw.Kr. 45:-. Acta Radiologica Supplementum 328. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

Tomography of the Temporomandibular Joint: Correlation between Tomographic Image and Histologic Sections in a Three-dimensional System. By Olof Eckerdal, Umeå, Sweden. Paper. Pp. 107, with many figures. Price, Sw.Kr. 40:-. Acta Radiologica Supplementum 329. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

Radiation Injury of Connective Tissue: A Biochemical Investigation with Experimental Granuloma. By Jorma Rantanen, Turku, Finland. Paper. Pp. 92, with some figures. Price, Sw.Kr. 40:-. Acta Radiologica Supplementum 330. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Surfer Hotel, Maui, Hawaii, April 21–25, 1974.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 25–30, 1973.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Roosevelt Hotel, New Orleans, La., April 1–6, 1974.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Chicago, Ill., June 15–19, 1974.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.
Oral examinations will be held in the following cities during the next 2 years: Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel; New York, N. Y., June 10–14, 1974, New York Hilton Hotel; and Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.
Written examinations are scheduled in 13 large centers in June 1974.
Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.
Deadline for filing applications for any examination in 1974 was September 30, 1973.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Chairman, Dr. Merrill A. Bender, Suite 2402, 211 E. 43rd St., New York, N. Y. 10017.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D. C. 20010. Annual Meeting: Jointly with RSNA, Palmer House, Chicago, Ill., Nov. 25–30, 1973.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting: Royal Sonesta, New Orleans, La., Oct. 24–28, 1973.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting: Ann Arbor, Mich., Oct. 14–18, 1973.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907. Meeting: Feb. 16–21, 1975.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Meeting in June, 1975 in Edinburgh, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Richard H. Greenspan, Dept. of Radiol., Yale University School of Medicine, 333 Cedar St., New Haven, Conn. 06510. Twenty-second Annual Meeting, New York, N. Y., May 8–11, 1974. Dr. Norman E. Chase is in charge of arrangements, New York Medical Center, New York, N. Y. 10016.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Charles J. Masters, Maj. MC, Department of the Air Force, United States Air Force Hospital, Wiesbaden (USAFE) APO New York 09220.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Giacomo Pantaleo, 69-13 Forest Ave., Brooklyn, N. Y. 11227. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Ru-Kan Lin, 191 North St., Buffalo, N. Y. 14201. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jasumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month Holiday Inn, Morgantown, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer, Dr. William H. Roush, Radiology Service, Veterans Administration Hosp., Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Lee Radford, 6011 Harry Hines, Suite 900, Dallas, Tex. 75235. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309. Fall meeting at Cloister, Sea Island, Ga., Sept. 15–16, 1973.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Menzie H. Brown, 155 S. 18th St., Kansas City, Kans. 66102.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Richard E. Litt, 6125 S. W. 31st St., Miami, Fla. 33155. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Raymond W. Brust, Jr., St. Francis Hosp., 2260 Liliha St., Honolulu, Hawaii 96817. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Shirley D. Vickers, Catholic Medical Center of Brooklyn and Queens, 88-25 153rd St., Jamaica, N. Y. 11432. Annual Meeting: Houston, Texas, June 16–20, 1974.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John R. Dehner, Reid Memorial Hosp., Richmond, Ind. 47374.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference.

LOUISIANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

MAINE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

MASSACHUSETTS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Eli A. Etscovitz, 10 Stonewood Dr., Newton Centre, Mass. 02159.

MEMPHIS ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MICHIGAN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MICHIGAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary-Treasurer, Dr. A. G. Fleurquin, Pontiac General Hosp., Pontiac, Mich. 48053. Meets bi-monthly during the academic year.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. Howard L. Copas, 827 South 131st St., Omaha, Neb. 68154. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting: May 10, 1974.

NEW ENGLAND SOCIETY FOR RADIATION ONCOLOGY
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS, CHAPTER OF ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW ORLEANS RADIOLOGICAL SOCIETY, INC.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 25–27, 1974. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

NORTH CAROLINA CHAPTER OF ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Marshall Landa, 1702 S. University Dr., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Mark D. Reiss, 2100 Rockwood Dr.,

Sacramento, Calif. 95825. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 22–24, 1974.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. Robert M. Maurer, Santa Rosa Medical Center, San Antonio, Tex. 78207. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President, Dr. Leland E. Kellerhouse, 2466 First Ave., San Diego, Calif. 92101. Meets the first Monday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, Mary's Help Hospital, 1900 Sullivan Ave., Daly City, Calif. 94015.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Chairman, Dr. Henry W. Wiggins, Jr., Mercy Hosp., 2537 South Prairie Ave., Chicago, Ill. 60616. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting.

South Bay Radiological Society
Secretary, Dr. Virgil H. Voss, 900 Kiely Blvd., Santa Clara, Calif. 95051. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St.,

Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane I. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 311 Medical Arts Bldg., Chattanooga, Tenn. 37402. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neil, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205. Annual Meeting: La Mansion Hotel, San Antonio, Tex., March 21–23, 1974.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada. Annual meeting.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Don H. Nichols, 5770 S. 300 East, Murray, Utah 84107. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 8952 Bellfonte Rd., Richmond, Va. 23229.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 260 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Isaiah J. Seligman, 16 Guion Pl., New Rochelle, N. Y. 10801. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, Gundersen Clinic, LaCrosse, Wis. 54601. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Francisco Miranbell S., Apartado 3352, San José, Costa Rica. Thirteenth Central American Congress of Radiology, San José, Costa Rica, Dec. 4–8, 1973.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. T. Kerr, 32 Welbeck St., London, W1M/7PG, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, 1555 Summerhill Ave., Montreal 109, Que., Canada. Thirty-seventh Annual Meeting, Regency Hyatt House, Vancouver, B. C., April 21–26, 1974.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln Inn Fields, London WC2A 3PN, England.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

Sociedad Argentina de Radiología
Secretary-General, Dr. Tomás J. Gutiérrez E, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. J. Jääskeläinen, The Children's Hospital, Stenbäckinkatu 11, SF-00290 Helsinki 29, Finland. 11th Annual Meeting: May 29–June 1, 1974, Helsinki.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting: Salpetrière, Paris, Dec. 1, 1973.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK-8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland. Annual Meeting in Uppsala, Sweden in June 1974.

Socied ad pañola de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa 1-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Japanese American Radiological Society
Secretary, Thomas J. Imray, Maj. MC, Chief, Radiol. Dep., US Army Hosp., Camp Zama, Japan, APO San Francisco, Calif. 96343. Meets 3rd Thursday, monthly at various locations in Tokyo.

Philippine College of Radiology
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ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Neck and Chest

Spindola-Franco, Hugo, Adams, Douglass F., Herman, Michael V., and Abrams, Herbert L. Reciprocating flow in the coronary circulation. *Radiology*, June, 1973, *107*, 497–504. (From: Departments of Radiology and Medicine [Division of Cardiology], Harvard Medical School and Peter Bent Brigham Hospital, Boston, Mass.)

The application of coronary arteriography to the study of coronary disease in man has received a major impetus from the development of surgical procedures to augment myocardial blood flow. Dynamic recording, utilizing cine techniques, has also shed new light on coronary blood flow patterns. Flow in the coronary arteries is normally phasic, although the normal flow pattern is almost certainly altered by rapid selective injection of contrast material into the coronary artery. A type of reversal of flow has been observed in some patients with coronary artery disease. This flow pattern, designated reciprocating flow, may be defined as the phasic alternation of the direction of blood movement in a coronary artery, so that in some phase of the cardiac cycle it is antegrade, and in others, retrograde. Critical analysis of this pattern, and its significance, are the subject of the present report.

One-hundred-twenty-five patients with a clinical diagnosis of coronary artery disease were studied with selective coronary injections and cine recording. The direction of flow was determined and related to the events of the cardiac cycle by means of frame-by-frame analysis. Flow was considered antegrade when the contrast medium column moved away from the coronary orifice into which it had been injected, and retrograde when the contrast medium column moved toward the coronary orifice. Reciprocating flow was considered to be present only when demonstrated in sequential beats on multiple injections.

Twenty-five of the 125 patients had normal arteriograms or stenosis with less than 50 per cent reduction of the arterial lumen and no collaterals. One hundred coronary arteriograms showed occlusion or stenosis greater than 50 per cent and 75 of these had demonstrable collaterals. In 22 patients with collaterals, reciprocating flow was detected in 1 branch and in 3 patients it was detected in 2 branches. Reciprocating flow was associated with either severe stenosis or segmental occlusion of a coronary artery. Conversely, reciprocating flow was not detected in any coronary artery with multiple stenoses of greater than 80 per cent. Beyond the stenosis, the vessel in which the reciprocating flow was detected usually had a normal or near normal caliber.

Three distinct patterns of reciprocating flow evolved from the correlation of the direction of flow to the events of the cardiac cycle: primary epicardial; secondary epicardial; and intramyocardial.

The primary epicardial reciprocating flow pattern is identical to the normal coronary flow pattern in that there is antegrade flow in the rapid ejection period which decreased at the end of systole and increased at the beginning of diastole. It then progressively diminished during diastole. Antegrade flow ceased with the inception of atrial systole and there was retrograde flow during isovolumetric contraction.

In contrast, secondary epicardial reciprocating flow represented the reverse of the primary epicardial flow pattern, with respect to the cardiac cycle. Both truly reflect the same flow pattern, but with different ends of the blood column opacified by the selective injection.

The intramyocardial flow pattern is characterized by retrograde flow during the ejection period. It is usually observed in the septal arteries and appears to represent a squeezing of septal arteries by the septal ventricular musculature. This pattern has been observed in subjects with normal coronary arteries, but is much more obvious in patients with coronary disease because the septal arteries are frequently enlarged and form a part of the collateral network.

The outflow of blood from a vessel exhibiting reciprocating flow is into the capillary bed, which represents a compressible reservoir in the blood column. Therefore, when reciprocating flow is visualized, a capillary bed—and hence perfusable myocardium—are assumed to exist beyond the branch displaying reciprocating flow. Because reciprocating flow is usually associated with a hemodynamically significant lesion and because the recipient artery beyond the stenosis is generally ample in size, its presence should be considered a favorable sign, if coronary bypass surgery is contemplated.—*E. B. Best, M.D.*

Strauss, H. William, Zaret, Barry L., Martin, Neil D., Wells, Harry P., Jr., and Flamm, M. D., Jr. Noninvasive evaluation of regional myocardial perfusion with potassium 43: technique in patients with exercise-induced transient myocardial ischemia. *Radiology*, July, 1973, *108*, 85–90. (From: Department of Medicine, Sections of Nuclear Medicine and Cardiology, David Grant USAF Medical Center, Travis Air Force Base, Calif.)

Cardiac imaging was performed following intravenous administration of potassium 43 in 6 patients with angina pectoris and 3 normal individuals, both at rest and following exercise stress tests. Three patients with documented myocardial infarction were studied at rest.

The resting studies were performed following the intravenous injection of 400–800 uc potassium 43 in supine position. For the exercise study, the patients underwent a graded treadmill exercise test continued to the endpoint of angina pectoris or fatigue. The potassium 43 was injected 30 to 60 seconds prior to halting exercise. The time from injection to the beginning of the scan was less than 5 minutes in all cases.

In the 3 individuals with no evidence of heart disease, the myocardial scans had a uniform pattern of potassium 43 accumulation with maximum concentration in the left ventricular wall. No change was seen when the potassium 43 was administered during the stress of maximal treadmill exercise.

In the 3 patients who had sustained a myocardial infarction, the scan obtained at rest revealed a clearly demarcated area of decreased uptake of radioactivity which corresponded to the electrocardiographic site of infarction.

In the 6 patients with angina but without previous infarction, the myocardial scans at rest were essentially normal. The scans, obtained following the administration of the potassium 43 during the exercise stress test demonstrated clearly demarcated zones of relative decrease of tracer. Coronary angiography performed in 4 of these patients revealed significant obstruction of vessels perfusing the region of decreased uptake seen on the scan.

The images obtained represent the combined effect of blood flow and the uptake of the potassium 43 by the tissue. During exercise stress, the difference in blood flow between normal and abnormal coronary arteries is greatest and the absolute quantity of potassium 43 presented to the ischemic myocardium is less than that available to normal myocardium. The increased loss of potassium from an ischemic region may also play a role.

The authors believe that these initial data appear promising in providing a simple, safe, noninvasive technique to assess abnormalities in regional myocardial perfusion.—*W. R. Carpentier, M.D.*

GENITOURINARY SYSTEM

CATTELL, W. R., MCINTOSH, C. S., MOSELEY, I. F., and FRY, I. KELSEY. Excretion urography in acute renal failure. *Brit. M. J.*, June, 1973, *2*, 575–578. (From: St. Bartholomew's/St. Leonard's Regional Renal Unit, and the Departments of Radiology, St. Bartholomew's Hospital, and St. Leonard's Hospital, London, England.)

High dose excretion urography (1 ml./lb. body weight of hypaque 45 per cent) with tomography was carried out in 32 patients presenting with acute nonobstructive renal failure. Most (22) were oliguric (less than 500 ml. of urine per day) and all were uremic. Histologic diagnosis was obtained by biopsy or at autopsy in 19 patients.

After the intravenous injection (3–5 minutes) immediate tomograms were obtained (1 minute) and usually repeated with most of the other conventional films exposed at 5, 10, 30 and 60 minutes and at intervals up to 24 hours.

The films were examined for renal size, time of first appearance of the nephrogram, maximum nephrogram density, change in and total duration of nephrogram density as well as the presence or absence of a pyelogram.

Twelve patients with uncomplicated acute tubular necrosis and 3 patients with acute suppurative pyelonephritis showed an early (1 minute), dense, persisting (1–24 hours) nephrogram bilaterally. A pyelogram was observed in all patients with daily urine outputs in excess of 1 liter. In general, but not invariably, oliguria is associated with no pyelogram or only a faint one. These findings are modified in complicated (severe glomerular disease) acute tubular necrosis (2 patients), and acute pyelonephritis (2 patients), with a delayed (30 minutes), increasingly dense nephrogram or very delayed (24 hours) faint nephrogram with variable pyelogram.

Acute renal failure due primarily to glomerular disease showed a different distinctive nephrographic pattern. Three patients with acute glomerulitis showed delayed (1–60 minutes), increasingly (1–24 hours) dense nephrograms and no pyelograms. Patients with chronic glomerulonephritis (3) and malignant essential hypertension (2) had early (1–30 minutes), faint, persisting (24 hours) nephrograms accompanied by a pyelogram.

In 5 of the 32 patients studied, no nephrogram or pyelogram was observed. Two had total renal infarction, 1 had severe interstitial (drug) nephritis with severe tubular compression by edema with normal glomeruli, 1 had combined tubular blockage (myeloma protein) and glomerulonephritis, and 1 extremely severe anuric acute glomerulonephritis.

Careful analysis of the evolution (changes in density observed with time) of the nephrogram in patients with acute nonobstructive renal failure will provide valuable information about the nature of the underlying parenchymal disease.

No adverse effects attributable to excretion urography were encountered.—*Richard Pfister, M.D.*

ATHANASOULIS, CHRISTOS A., BROWN, BORDEN, and BAUM, STANLEY. Selective renal venography in differentiation between congenitally absent and small contracted kidney. *Radiology*, Aug., 1973, *108*, 301–305. (From: Departments of Radiology, Harvard Medical School and the Massachusetts General Hospital, and the Departments of Radiology, Boston University School of Medicine and Boston City Hospital, Boston, Mass.)

Presented is a study of 10 patients by aortography and selective renal venography after an excretory

urography revealed only 1 kidney. In 9 patients the left kidney did not visualize and in 1 the right. Cystoscopy was performed in 5 patients.

The aortogram showed no vessels to the side of the nonvisualized kidney. Delayed opacification was not seen. In 7 patients venography confirmed the aortographic findings indicating unilateral renal agenesis. In 2 patients venography demonstrated a small kidney and in 1 predicted the presence of a dysplastic kidney, later proved by surgery. In 1 patient cystoscopy confirmed unilateral renal agenesis with absence of the left ureteric orifice and the ipsilateral hemitrigone. Cystoscopy was inconclusive in the remaining 4 patients.

In unilateral agenesis the renal vein does not develop. However, on the left side the renal collar will go on to form, ventral to the aorta, a vein into which the suprarenal and sex veins drain. On the right, since the renal vein enters directly into the inferior vena cava, there will be no renal vein.

When absence of a kidney is indicated by angiography, a renal venogram can show the plexiform pattern of the intrarenal system. In 6 of the 7 cases of left renal agenesis, reflux into the left adrenal vein demonstrated the adrenal gland. In the 7th the adrenal vein was opacified, but did not reflux into the intra-adrenal venous plexus.

Revision of the term renal hypoplasia is proposed in the case of the small contracted kidney.

It is suggested by the authors that renal venography, a relatively easy procedure, may offer additional useful information in the evaluation of unilateral renal agenesis and the small contracted kidney.

Three selected case histories are presented.—*M. J. Sedlmeir, M.D.*

BLOOM, H. J. G. Adjuvant therapy for adenocarcinoma of the kidney: present position and prospects. *Brit. J. Urol.*, June, 1973, *45*, 237–257. (From: Royal Marsden Hospital and Institute of Cancer Research, and the St. Peter's Group of Hospitals and Institute of Urology, London, England.)

The present 5 year survival figure for all surgically treated (nephrectomy) cases of adenocarcinoma of the kidney is about 50 per cent. This communication directs itself to exploring 4 major ancillary methods of treatment to augment or assist the main surgical attack on the primary tumor, and prevent or delay the development of local recurrence and distant metastases.

(1) *Postoperative radiotherapy* has long been used in conjunction with surgery for renal cell carcinoma. In recent years its value has become a subject of wide controversy. The reputation of radioresistance and the frequency of tumor deaths from distant metastases are responsible for the reluctance of many urologists to consider its adjuvant use. Until large comparative studies are carried out, postoperative radiotherapy is presently suggested for residual disease, capsular invasion, high-grade histologic malignancy and lymph node positive cases. *Preoperative radiotherapy* (4,000 rads in 4 weeks) is presently undergoing cooperative study trials in the United States, London, and Rotterdam. Its aims are to shrink the tumor, reduce cellular reproductivity, destroy direct extensions, increase the mobility of a tethered organ and control limited metastases in regional lymph nodes. Until the results of the clinical trials are available, its use is recommended for large or tethered tumors.

(2) *Cytotoxic drugs* have occasionally produced objective responses of renal carcinoma, but any improvement has invariably been limited and of brief duration. Presently no useful compound against renal adenocarcinoma has been found, and its use appears limited to perhaps the advanced tumors including metastases.

(3) *Hormone therapy* (progestins, testosterone) for renal cell neoplasia is based on observations in laboratory animals on the production of renal cortical tumors in male hamsters by estrogen, together with their inhibition by estrogen-antagonists and castration. The present limited clinical experience has yet to be established, but suggests a 17 per cent objective response, chiefly to progestins.

(4) *Immunotherapy* has so far met with little success in terms of tumor regression or increased patient survival. Vaccines prepared from the patient's own tumor or specific adoptive immunotherapy with sensitized lymphocytes are hoped to prove effective. The immune capabilities of patients with low grade malignancy (90 per cent 5 year survival rate with nephrectomy alone) should be studied so that an effective host resistance against renal cell carcinoma may be found.—*Richard Pfister, M.D.*

ANDERSON, MAVIS F., and ATKINSON, DAVID W. Renal carcinoma in pregnancy. *Brit. J. Urol.*, June, 1973, *45*, 270–272. (From: Departments of Obstetrics and Gynaecology and Surgery, University Hospital of the West Indies, Mona, Kingston, Jamaica.)

This report adds 3 additional cases of renal cell carcinoma seen in pregnant women to the 16 previously reported cases.

Adenocarcinoma of the kidney is seldom encountered in pregnant women since renal malignancy is most common in the 50 to 70 year age group; however, its occurrence below the age of 30 years is not rare.

Their patients were 27, 38, and 42 years old at the time of diagnosis and in their 15th, 16th, and 14th week of pregnancy respectively when initial intravenous urographic studies were done.

Nephrectomy was performed at the 16th week, 6

months post partum (mistakenly carried as renal cyst) and 15th week respectively (provisional diagnosis of ovarian tumor). All women were delivered of normal infants. Two patients have survived 5 years, the third 4 years after nephrectomy, and are alive and well, and free of metastatic disease.

The rarity of renal carcinoma in pregnant women and the reluctance to undertake full radiologic investigations during pregnancy, is likely to make the diagnosis even more difficult. Adequate shielding of the uterus and fetus is mandatory in all radiologic studies including limited selective renal arteriography.

Although not mentioned by the authors, ultrasonography is perhaps the method of choice for further evaluation after limited intravenous urography has delineated the presence of a renal mass.—*Richard Pfister, M.D.*

Hutchison, Robert J., and Nogrady, M. Bernadette. Late sequelae of renal trauma in the pediatric age group. *J. Canad. A. Radiologists*, March, 1973, *24*, 3–11. (Address: M. Bernadette Nogrady, M.D., Department of Diagnostic Radiology, The Montreal Children's Hospital and McGill University, Montreal, Quebec, Canada.)

The authors reviewed the clinical records and roentgenologic examinations of 123 children, ages 3 to 16 years, investigated at the Montreal Children's Hospital in the last 10 years for abdominal or multiple trauma. All patients presented with hematuria and sometimes flank pain or tenderness. Initial radiographic investigation consisted of plain film roentgenography followed by excretory urography as soon as clinically feasible. Renal angiography was performed on 5 patients.

The patients were divided into 4 groups based on the radiologic findings.

In Group I, 46 children showed no urographic abnormality and were not investigated further.

Fifty-seven Group II patients had decreased renal function on the injured side, sometimes associated with poor visualization or distortion of the calyceal pattern. All patients were treated conservatively. Eleven demonstrated post-traumatic scarring of the kidney; 10 were lost to follow-up; 36 demonstrated complete recovery.

Group III included 14 patients in whom extravasation of contrast medium occurred in addition to the changes described above. In 7, the extravasation was intrarenal and follow-up urography demonstrated normal kidneys in 2; diminished growth or parenchymal scarring in 3; 2 were lost to follow-up. Seven patients had extrarenal extravasation, indicating more serious injury. One patient had a gradual return of the kidney to normal and it showed normal renal growth 3 years later. Of the other 6 patients, 2 underwent surgical exploration with drainage of pararenal fluid collections. All 6 patients subsequently demonstrated renal scarring and calyceal distortion. The greatest reduction in kidney size was shown to occur within the first year after injury. The atrophied kidney subsequently demonstrated slower growth than the normal kidney. This is believed in part due to compensatory hypertrophy of the normal contralateral kidney. Focal hypertrophy may be confusing and simulate development of neoplasm. Two of these patients were lost to follow-up. The others demonstrated no development of hypertension and had no clinical signs or symptoms referrable to the kidney during the follow-up period. The authors recommend that patients be informed of their urologic status and undergo periodic determinations of blood pressure into adulthood.

Group IV included 6 patients with evidence of renal rupture and/or nonfunctioning kidneys. All these underwent nephrectomy or heminephrectomy. —*John T. Underberg, M.D.*

Gavelli, Giampaolo, De Florio, Luigi, Moretti, Saverio, and Babini, Lucio. L'effetto organografico a livello degli organi genitali femminili interni in corso di urografia perfusionale. (The organographic effect at the level of the female internal genital organs during perfusion urography.) *Radiol. med.*, July, 1972, *58*, 547–553. (From: Istituto de Radiologia e del Radio "L. Galvani" dell'Università, di presso il Policlinico S. Orsola, Bologna, Italy.)

The authors have observed that during drip infusion urography performed in female patients, very often the faint outline of the uterus and, at times, of the adnexa, noted in the preliminary roentgenogram of the abdomen, was definitely more evident because of an organographic effect. In order to confirm their findings, 3,000 drip infusion urograms obtained in the past 4 years were reviewed.

A critical evaluation of the authors' findings can be summarized as follows: (1) an organographic effect was demonstrated in 85 to 95 per cent of the cases, and in a third of the cases the demonstration of the uterus and adnexa was excellent; (2) this organographic effect was greater and more homogeneous in patients of childbearing age; (3) this effect was less evident in patients in the older age groups, even though it was noted that the shadow produced by the uterus, and demonstrable in the preliminary roentgenogram, was denser than in patients of the younger age groups; (4) this "gynecographic" effect appeared later than the nephrographic effect, and lasted longer; (5) this "gynecographic" effect allowed the drawing of definite and quite accurate conclusions as regards the size, shape and position of the uterus and, to a lesser extent, of the adnexa; and (6) in a large number of cases a suspected ab-

normality in the organs was confirmed by pelvic pneumotomography, lymphangiography, and pelvic angiography.

Ten cases are briefly reported.—*A. F. Govoni, M.D.*

Gynecology and Obstetrics

Strachan, C. J. L. Intrapelvic dermoid cysts in adolescent girls presenting with urinary retention: a report of 2 cases. *Brit. J. Surg.*, April, 1973, *60*, 287–288. (From: Western Infirmary, Glasgow, Scotland.)

Two cases of acute urinary retention due to intrapelvic dermoid cysts are reported. The author could find only 2 other cases in the literature: 1 case of a 40 year old male with a presacral dermoid cyst; and a single case of a female with an infected pelvic dermoid cyst.

Both of the author's cases were in girls who were 13 years old when acute urinary retention first developed. In 1 case a huge presacral dermoid cyst was removed surgically 2 weeks after acute urinary retention developed. In the other case a large dermoid cyst was removed from the left ovary after the third episode of acute urinary retention which recurred at 2 year intervals. On the first occasion the urinary retention was relieved by a single catheterization, while on the second and third occurrence an intramuscular injection of 0.25 mg. of carbachal was effective.

The acute urinary retention was due to a chronic process. At surgery both cases had considerable extrinsic obstruction at the bladder neck.

Roentgenograms showed a huge soft tissue mass in the first case and a pelvic mass with teeth-like structures in the second case.

Diagnostic ultrasound was very useful in establishing the preoperative diagnosis in the first case and corroborated the preoperative diagnosis in the second case.—*Paul M. Dassel, M.D.*

Blood and Lymph System

Melcore, Giuseppe, and Chiariotti, Franco. Quadro radiologico delle gambe nell'insufficienza cronica venosa. (Radiologic appearance of the legs in chronic venous insufficiency.) *Radiol. med.*, Nov., 1972, *58*, 867–885. (From: Istituto di Radiologia dell'Ospedale Civile di Gallarate, Varese, Italy.)

The authors have reviewed the roentgenograms of the lower limbs of 150 patients examined during a period of 3 years and presenting chronic venous insufficiency. The age of the patients varied between 19 and 85 years, and the pathologic changes in the venous system of the legs had been present from 2 months to 45 years prior to the examination.

The female sex was more often affected than the male: 58 per cent and 42 per cent, respectively. In 84 per cent of the cases, the phlebopathy was complicated, in the leg involved, by cutaneous ulcerations.

Bone changes were demonstrated in 88 patients, and in about half of the cases there was associated pathologic involvement of the soft tissue.

As previously observed, the authors could not establish a direct relationship between the site of the ulcer formations and the associated bone changes, although they noted a greater incidence of periosteal reaction in those patients who presented cutaneous ulcerative processes, in particular in the distal half of the lower limbs.

The roentgen findings, secondary to a periosteal reaction, were characterized by: (1) a thick bone reaction involving a long segment of the cortex; (2) an undulated appearance of the thickened cortex, and involving either the diaphysis of the tibia and/or of the fibula; (3) a saw-tooth or spicular bone reaction; and (4) a bony layering in which there was new bone formation separated from the cortex by a thin layer of radiolucency, as observed in chronic pneumopathies of the Pierre Marie type.

The pathologic changes involving the bone structure were characterized by: (1) eburnization of the cortex; and (2) osteoporosis.

The chronic venous insufficiency produced also cutaneous and deeper changes in the soft tissues.

Of the 150 patients observed by Melcore and Chiariotti, 56 presented changes in the soft tissues, and in 27 (19 per cent) the roentgen findings were of calcific deposits. The age varied between 28 and 85 years, and the process had been present between 3 and 45 years.

The other roentgen findings in the soft tissues were due to: (1) chronic edema; (2) pressure of varices; and (3) endovascular calcifications.

The illustrations are clear and numerous and the references are up-to-date.—*A. F. Govoni, M.D.*

Miyame, Tatsuya. Interpretation of ^{99m}Tc superior vena cavograms and results of studies in 92 patients. *Radiology*, Aug., 1973, *108*, 339–352. (From: Department of Radiology, Faculty of Medicine, University of Tokyo, Tokyo, Japan.)

The usefulness of vena cavography employing opaque contrast media, and its relative ease of performance, are well established. In obstructions of the superior vena cava, the secondary edema may necessitate a "cut-down," and the injection of large boli of contrast media may result in unfavorable side-effects. Since 1966 radionuclide angiography has been proposed as a suitable substitute.

This paper is presented to substantiate its use in a large series of patients, and to analyze the patterns to clarify the interpretations. Variant patterns, especially collateral pathways associated with superior vena caval obstructions are discussed. The

pulmonary circulation phase and the transit time from the antecubital vein to the right heart are established by inspection of time-lapse 35 mm. sequences.

From April 1967 until September 1971, 260 patients had 411 angiographies. Of the 260 patients, 150 had radionuclide angiography of either the superior, or inferior, vena cava. Only 92 are taken for study here: 45, or 126 examinations, with superior vena caval obstruction; and 47, or 53 examinations, of normals. Technetium 99m pertechnetate obtained from a molybdenum 99 generator was used in 5–10 mc amounts. The scintillation camera with the 4,000 hole collimator placed as close to the chest wall as possible was used. Both Polaroid and 35 mm. time-lapse recordings of the images were obtained.

Several normal variants were recognized: the pericardial reflection about the superior vena cava; the accumulation at valves in the larger veins; and defects secondary to marked supination or abduction of the arm during the antecubital injection.

The internal mammary route should be recognized and not mistaken for the superior vena cava when it has developed as a large collateral on the right. The visualization of the musculophrenic vein followed by the delayed visualization of the right heart and pulmonary artery should obviate this error. The development of the left internal mammary artery as a collateral is less confusing, for in addition to the delayed visualization of the right heart, the distended collateral lies to its left. The "shepherd's crook" configuration of the internal mammary artery may occasionally provide a basis for identification also.

The vertebral and azygos routes are best demonstrated when the images are obtained posteriorly. Attenuation of the gamma ray of Tc^{99m} by tissue and distance constitute a problem in demonstrating this route on anterior images, and distended superior vena cavae or internal mammary channels may overlie and obscure these more posterior channels. Lateral images may serve to clarify the latter situation.

A collateral route through the lateral thoracic vein may be seen with retrograde flow into the inferior vena cava, and a very delayed appearance in the right heart. An example of an extension on this is presented with retrograde flow down the superior epigastric vein into the femoral vein. More characteristically, the pattern of superior vena caval obstruction results in bilateral backflow into the internal jugular veins.

A means for relating numbers of frames of 35 mm. time-lapse films to transit time is presented and correlated with severity of the superior vena caval syndrome in 17 cases of complete obstruction. As expected, transit time was extended as the severity of the syndrome increased. This was not correlated with chronicity, which seemed to have a reciprocal effect.

Although several variables such as the effects of aging, respiration, and the valvular system of the veins themselves are indeterminate; the procedure stands as a rapid, innocuous, accurate means of establishing the diagnosis, its severity, and its secondary effects.

This article should be read in the original, for the accompanying reproductions of roentgenograms, scintiphotos, and labelled diagrams are worth 10,000 words.—*F. C. Petty, M.D.*

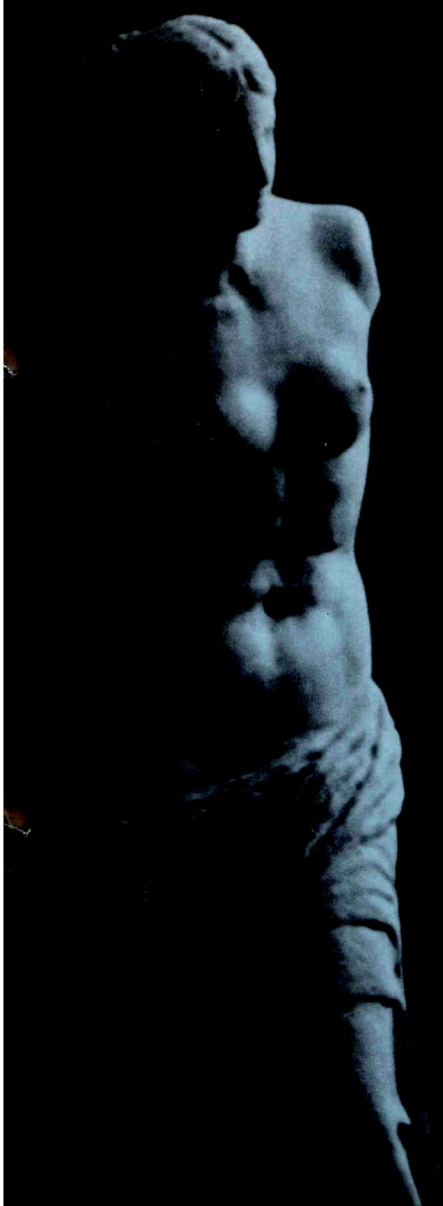
Wirtanen, George W. A new angiographic technique in the diagnosis of liver tumor. *Radiology*, July, 1973, *108*, 51–54. (From: Radiotherapy Center, Department of Radiology, University of Wisconsin School of Medicine, Madison, Wis.)

The hepatic angiograms of 332 patients with metastatic (96 per cent) or primary (4 per cent) liver cancer were evaluated for the presence of neovascularity and tumor staining. The majority of the metastatic cancer was from colorectal (39.5 per cent), breast (16.6 per cent), pancreatic (7.8 per cent), and biliary (7.2 per cent) malignant lesions.

The preferred technique used is to inject 50 ml. of meglumine diatrizoate-76 at 5 ml. per second selectively into the right and left hepatic arteries. Films are exposed every 2 seconds for 20 seconds and every 5 seconds for 10 seconds.

Since contrast material preferentially flows to the majority of tumors and circulates in tumors for prolonged periods of time, the slow injection rate allows the unopacified portal vein blood to clear the contrast material from the normal parenchyma. This enhances the tumor staining.

Only 8 patients (2.4 per cent) failed to reveal some degree of tumor stain or neovascularity. Well-defined tumor nodules or well-developed neovascularity was seen in 271 patients (81.6 per cent). Nodules as small as 1–2 mm. were demonstrated.—*A. K. Teaford, M.D.*



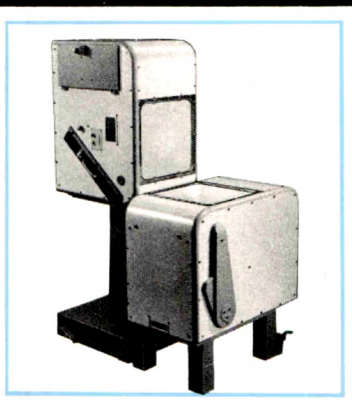
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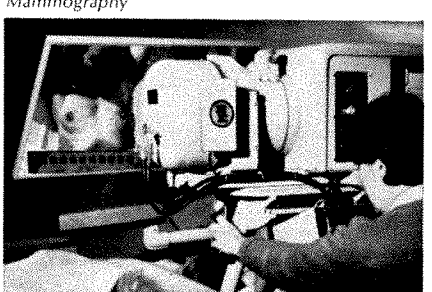
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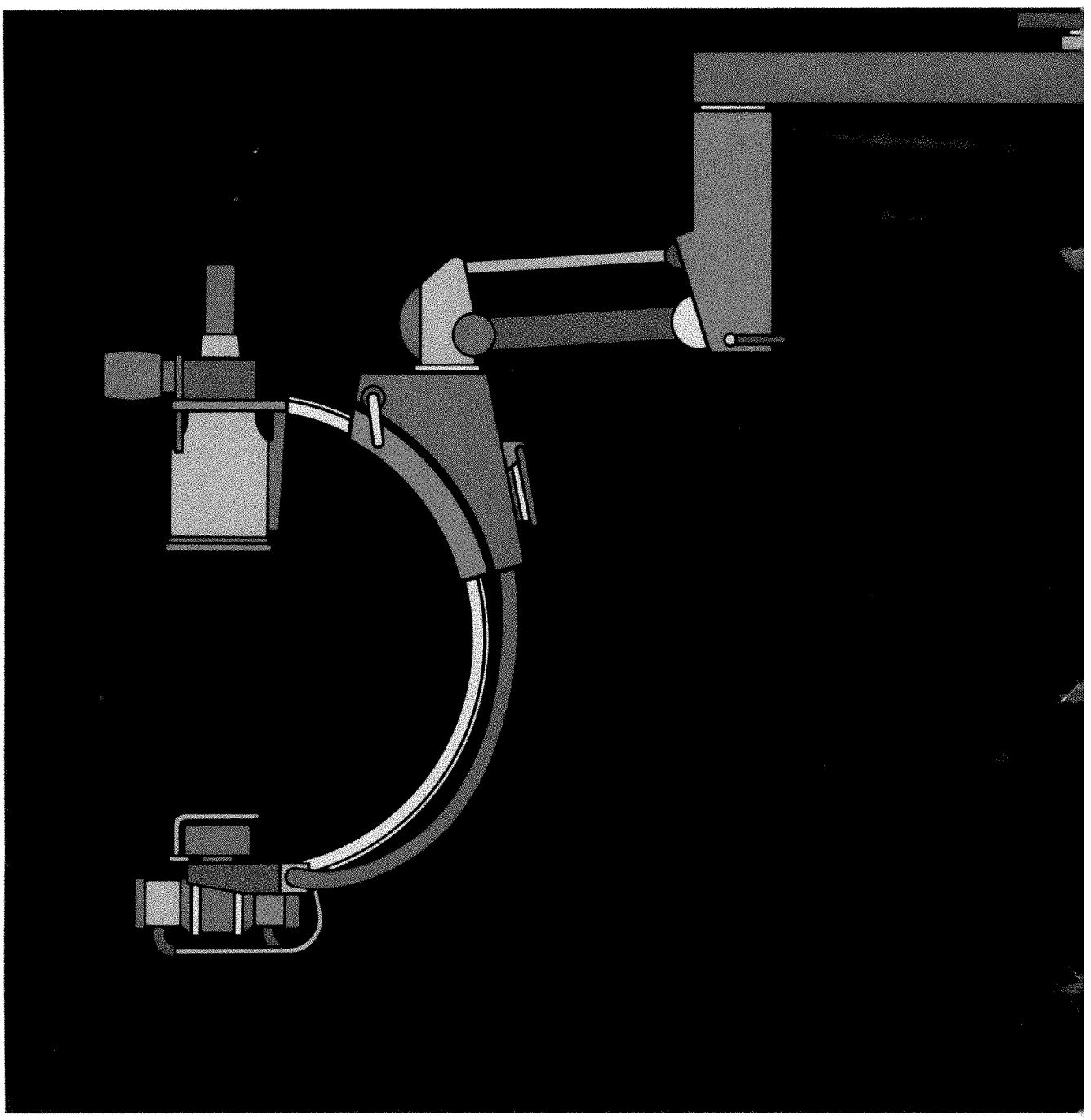
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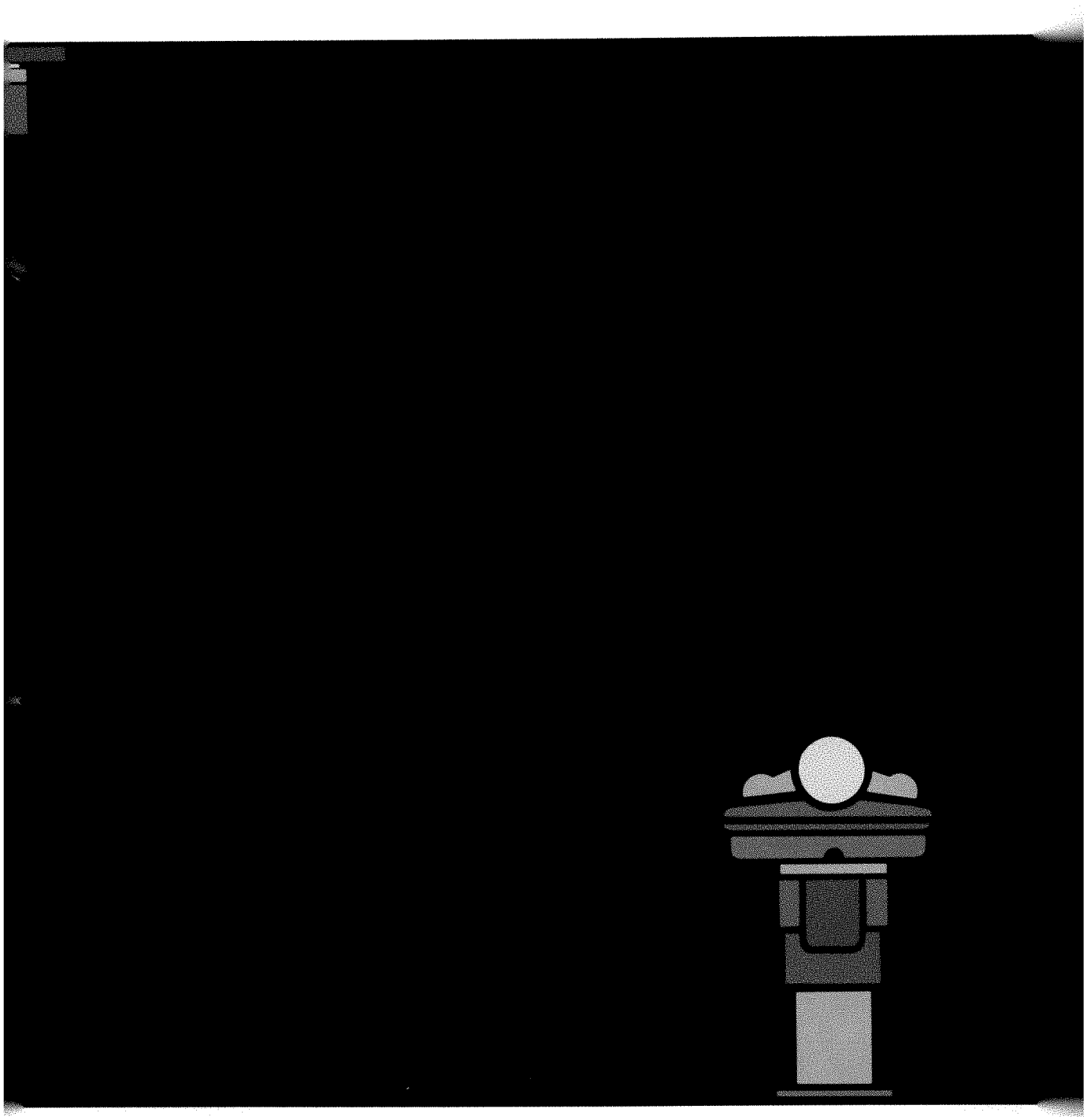
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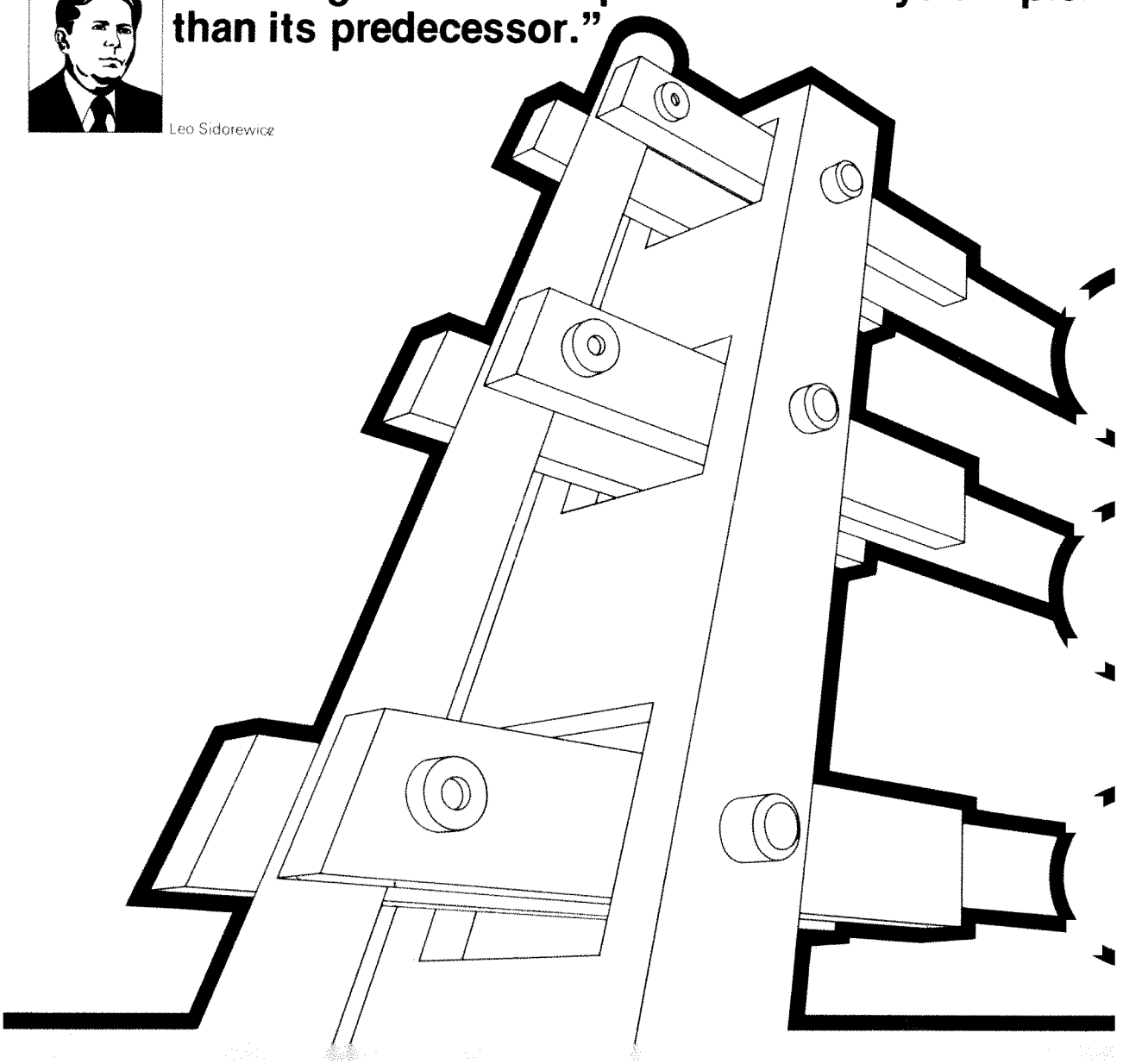
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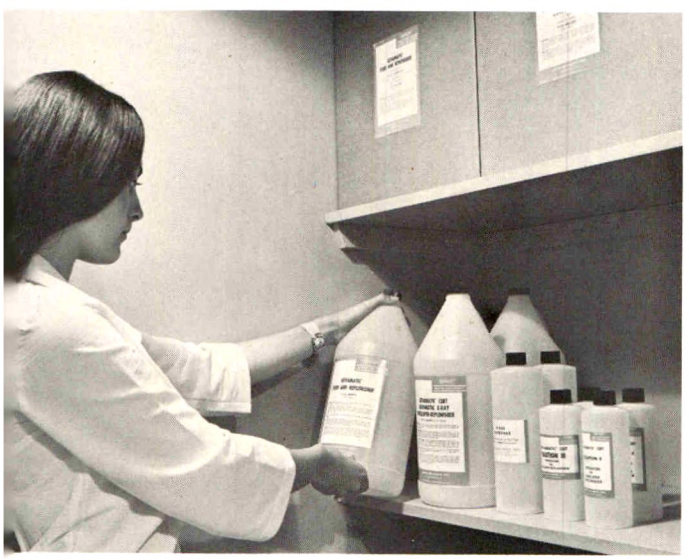
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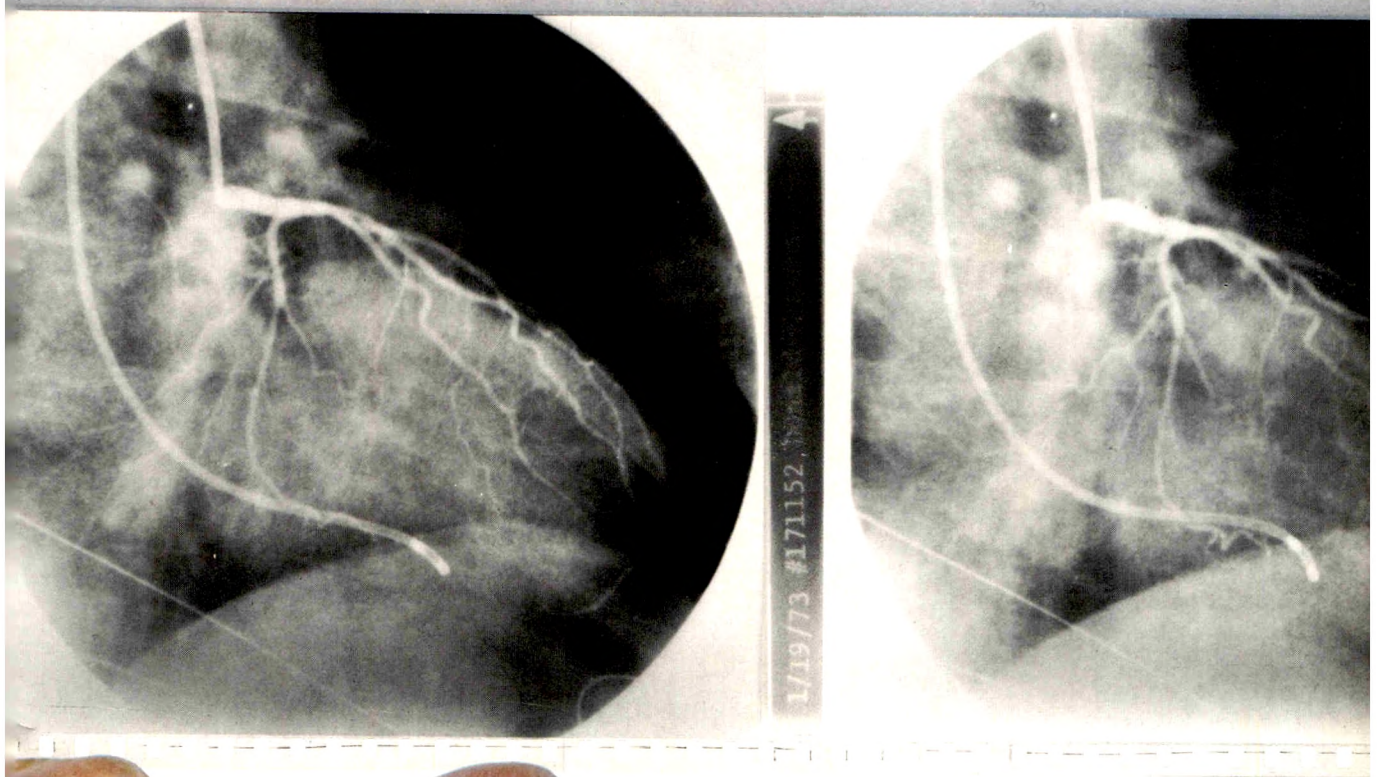


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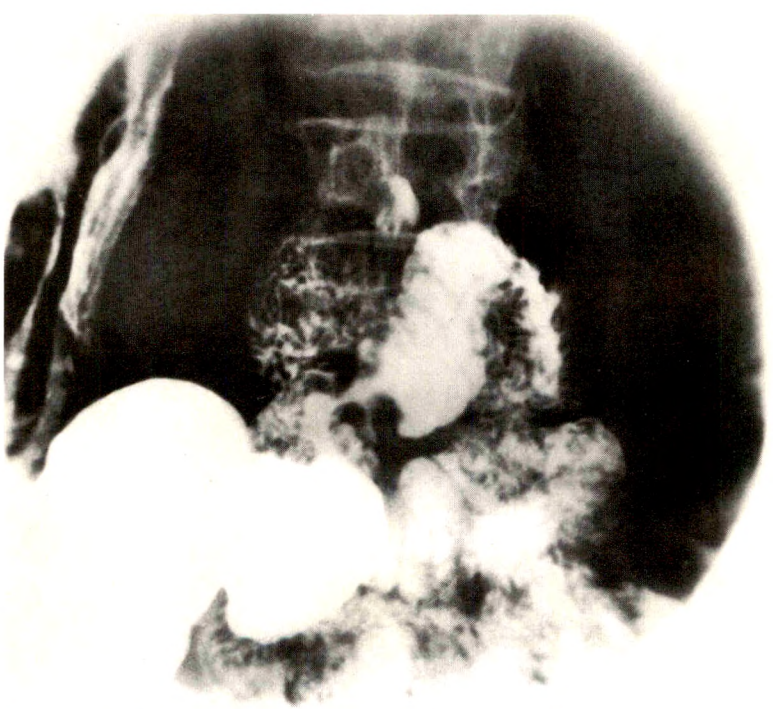
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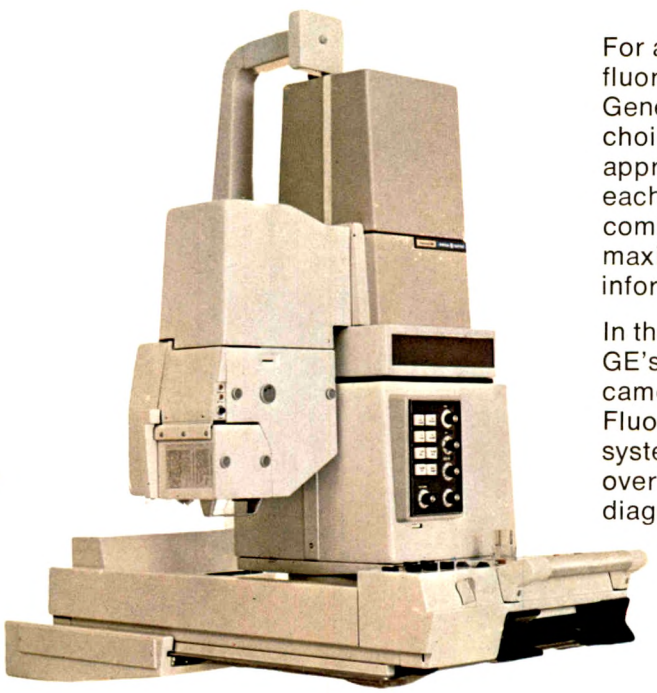
Here, the 105 mm photospot camera mounts inside the 90°-90° table on the image system. From remote control, record desired images, such as those during anatomical variance fluoroscopy. For this technic, the system's multiple, oblique tube unit angulations let you look around either side and to the head or foot. In effect, it rotates the patient by video instead of by hand. The 105 camera's recording range also extends to specialized radiographic studies such as voiding cystourethrograms.



Reduce or eliminate cassette changing during GI examinations with the 105 mm camera. Image shown here at actual size.

R&F systems

the tableside approach

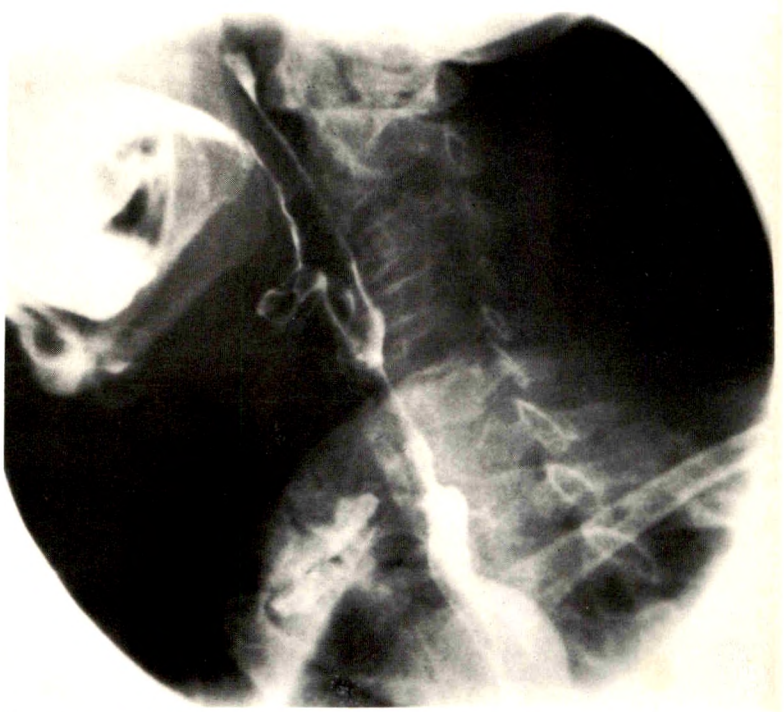


For all radiographic and fluoroscopic procedures, General Electric offers a choice of working approaches. And, within each, a coordinated combination of equipment maximizes total informational performance.

In the tableside approach, GE's 105 mm photospot camera mounts on the Fluoricon® 300 image system and is supported over a Monitrol® diagnostic x-ray table.

This system application for the 105 camera permits such technics as life size photospot images, in the intensifier's 4½ inch magnified mode; and the ability to perform rapid sequence filming, such as duodenal cap series and esophageal studies. For these and other examinations, a significant reduction in radiation can be achieved, compared with conventional spot-filming.





Recording of rapid anatomical motion of the esophagus is facilitated by frame rates up to 12 per second and exposure times as short as 2 ms. This 105 mm image shown actual size.

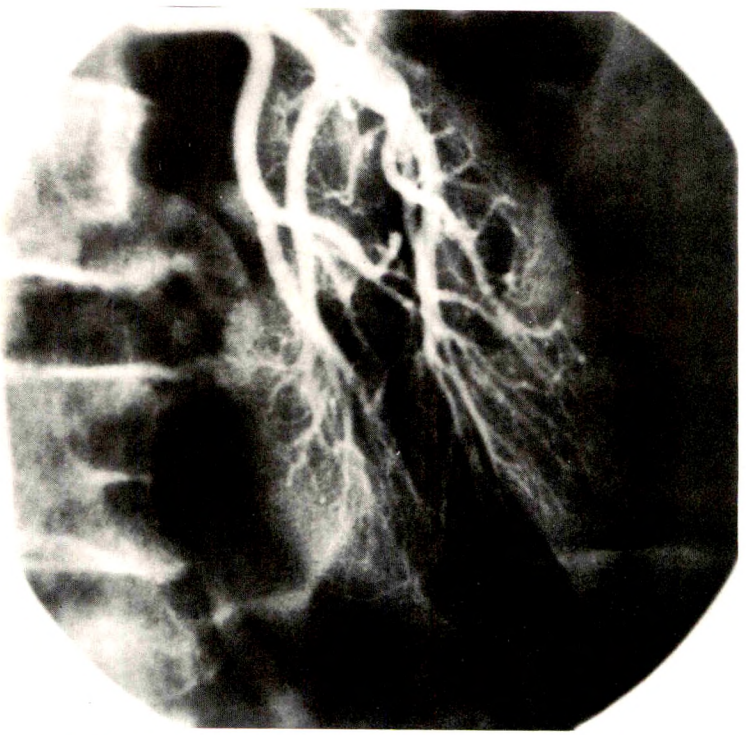
Vascular systems

the general approach

In general vascular procedures rooms, the 105 camera/image system/cath table equipment configuration permits still more photospot technic applications.

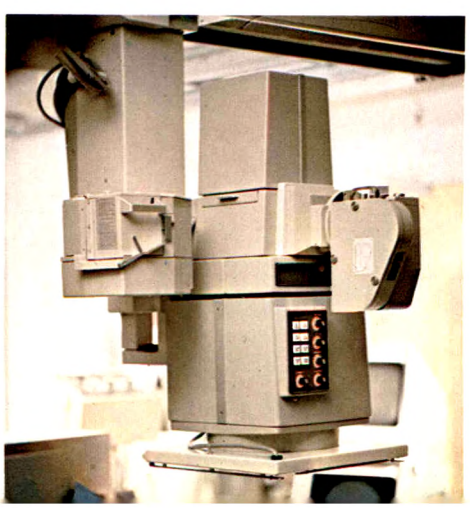
Including: fluoroscopic localization of the catheter and rapid-film recording. Lets you make sure the anatomical part and/or the catheter are precisely positioned before injection; allows minimum use of the contrast medium per examination. With all the operating benefits of a see-through film changer.

Another application of the 105 camera is quick, post-operative filming of certain surgical procedures, such as coronary bypass and pacemaker implantation. Permits a permanent record of the position observed during fluoroscopic confirmation.



Recording of general vascular procedures with the 105 mm camera makes optimum use of the system's full capability, including all generator mA stations, choice of focal spots, tube capacity and digital display of exposure time. Image shown here actual size.

the cardiovascular approach

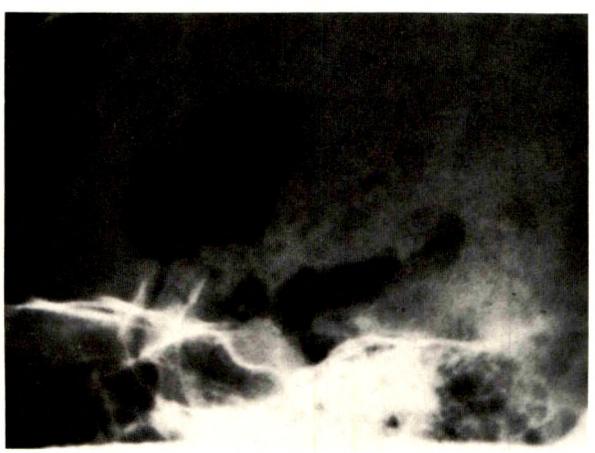


Now high frame rate photospot recording—up to 12 exposures per second—is as easy to perform as cine filming for cardiovascular procedures. Enables fluoroscopic confirmation of the catheter position prior to film recording. A press of the foot switch activates the GE 105 mm photospot camera.

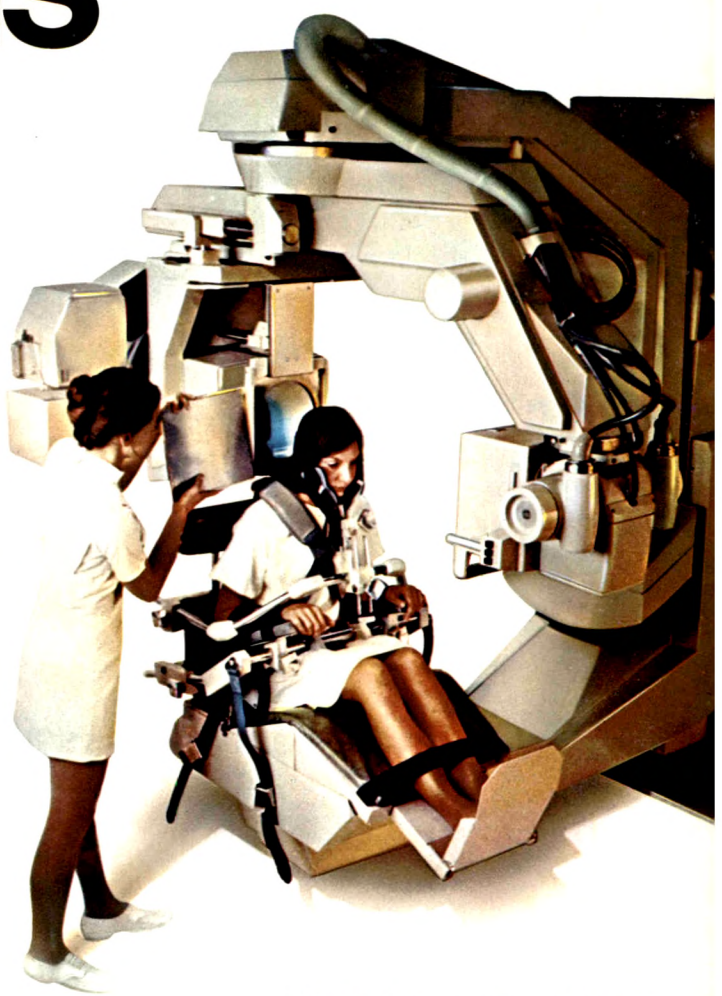
In addition, the 105 camera can reduce the number of film changer runs required for an examination. For example, make one changer run, then check other areas in question with the 105 camera. Means minimum contrast injected; less elapsed time per examination; greater technic convenience.

Neurological systems

the specialized approach



Collimated 105 mm image of ventricle filling, shown actual size.



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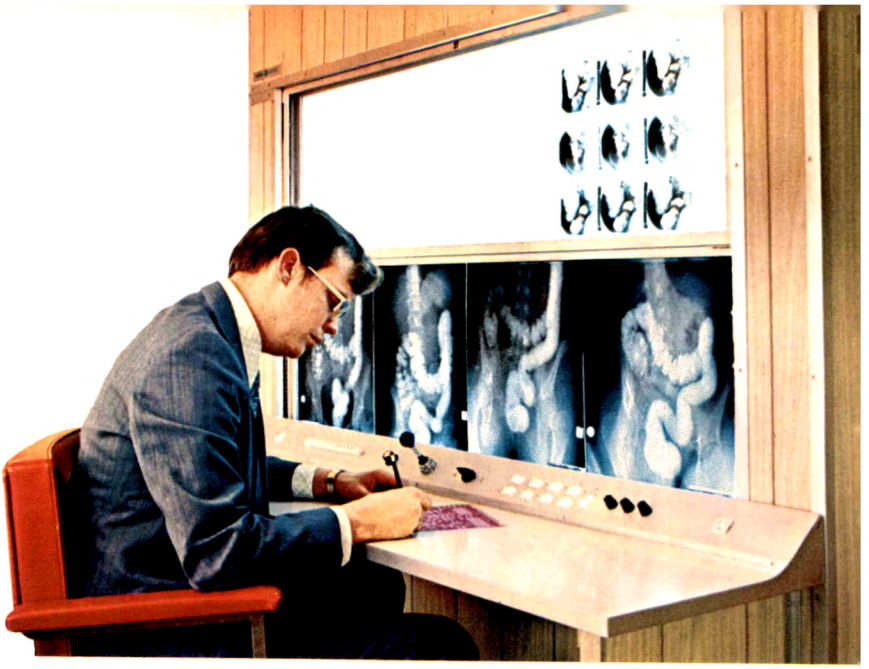
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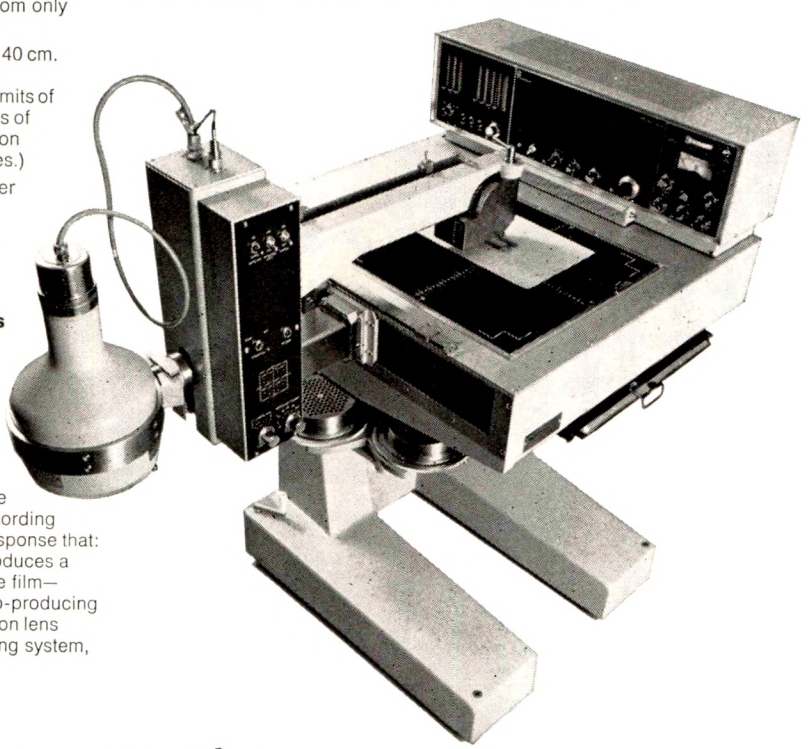
Quick-Change Collimators—Collimators are stored in a lazy susan tray below the scanning head—the 4-collimator capacity tray easily swings into position for collimator changing.

Digital Response—Both the photorecording and dot recording systems feature a digital response that: 1) with no suppression, produces a sharp-isotope image on the film—thanks to the digitized photo-producing light source and the precision lens system in the photorecording system, and that, 2) allows you to operate on a one-dot per one-count basis over a count-rate range of 0-4,000 counts per minute! Thanks to the exclusive Rapi/Dot™ tapper. (With this system you can obtain a tap scan that provides a sharp, continuous-tone reproduction of the isotope pattern!)

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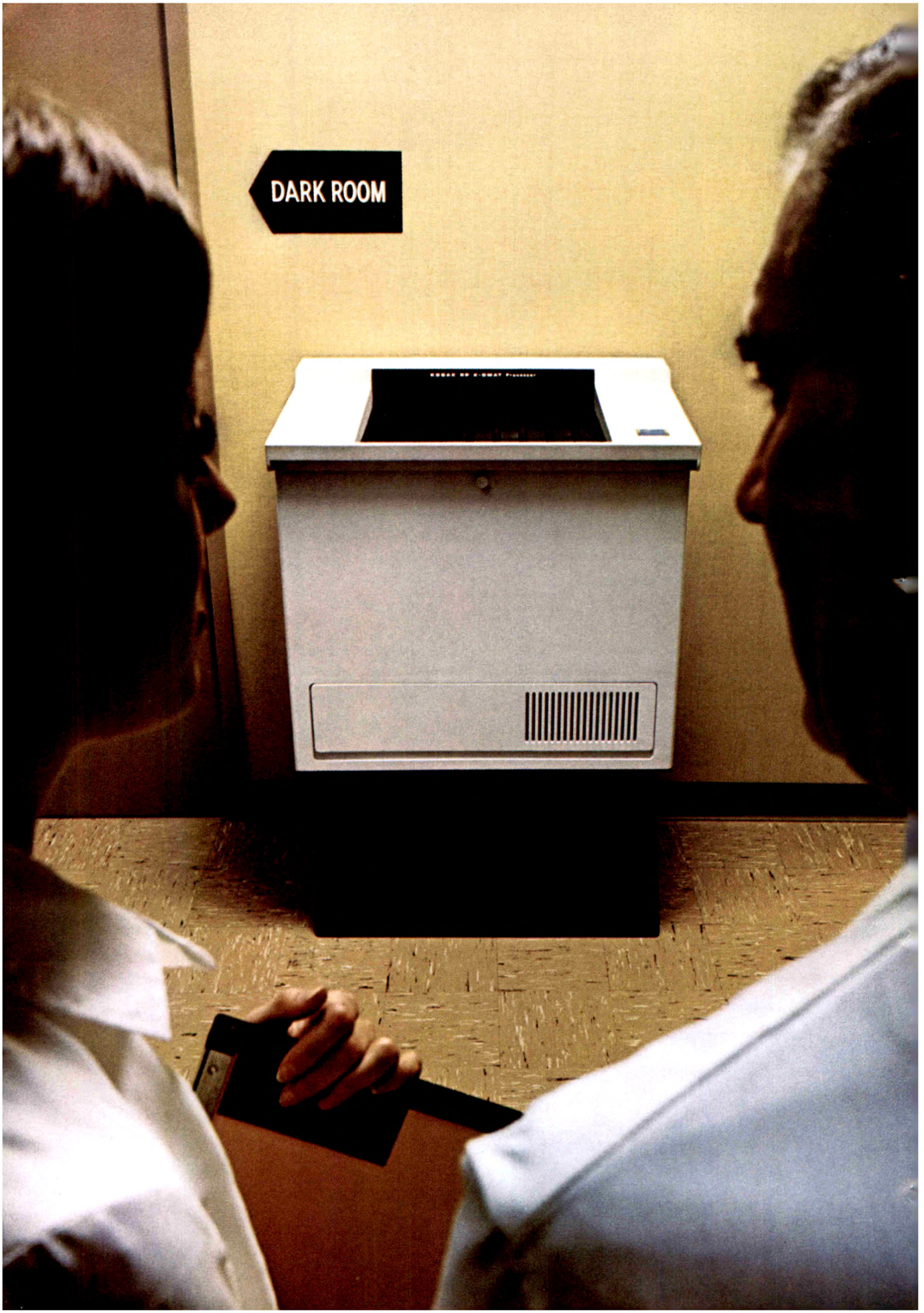
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Indication: Hypaque-Cysto is indicated for retrograde cystourethrography.

Adverse Reactions: No irritation or other undesirable effects have been reported following the administration of Hypaque-Cysto in clinical investigations.

Dosage and Administration: After the bladder is emptied, Hypaque-Cysto is gently instilled without force, often beyond the first desire to micturate, but not beyond the point of urgency or mild discomfort. The volume required to fill the bladder to slightly less than capacity may vary from patient to patient.

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In disease, bladder capacity in adults may vary from 50 ml. in neurogenic "reflux" bladder to over 1000 ml. in autonomous bladder or chronic lower urinary tract obstruction.

Repeat examination may be required to detect reflux, or in function studies.

The concentration varies with technique and equipment used. Hypaque-Cysto may be diluted with sterile water or 5 per cent dextrose solution, as indicated in the following table.

To Make		Add	Final Solution Contains
Final conc.	Final volume	Sterile water or 5% dextrose solution	Iodine
30 %	250 ml.	—	141 mg./ml.
25 %	300 ml.	50 ml.	118 mg./ml.
21.4%	350 ml.	100 ml.	101 mg./ml.
20 %	375 ml.	125 ml.	94 mg./ml.
18.8%	400 ml.	150 ml.	88 mg./ml.
16.7%	450 ml.	200 ml.	78 mg./ml.
15 %	500 ml.	250 ml.	71 mg./ml.

How Supplied: Calibrated 500 ml. dilution bottles containing 250 ml. Hypaque-Cysto; rubber stoppered, with inner removable seal and screw neck.



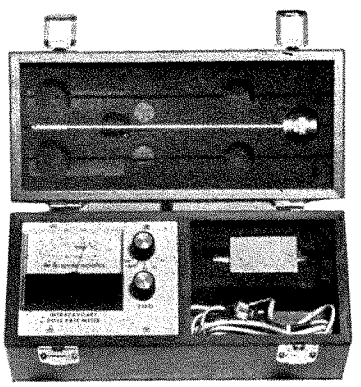
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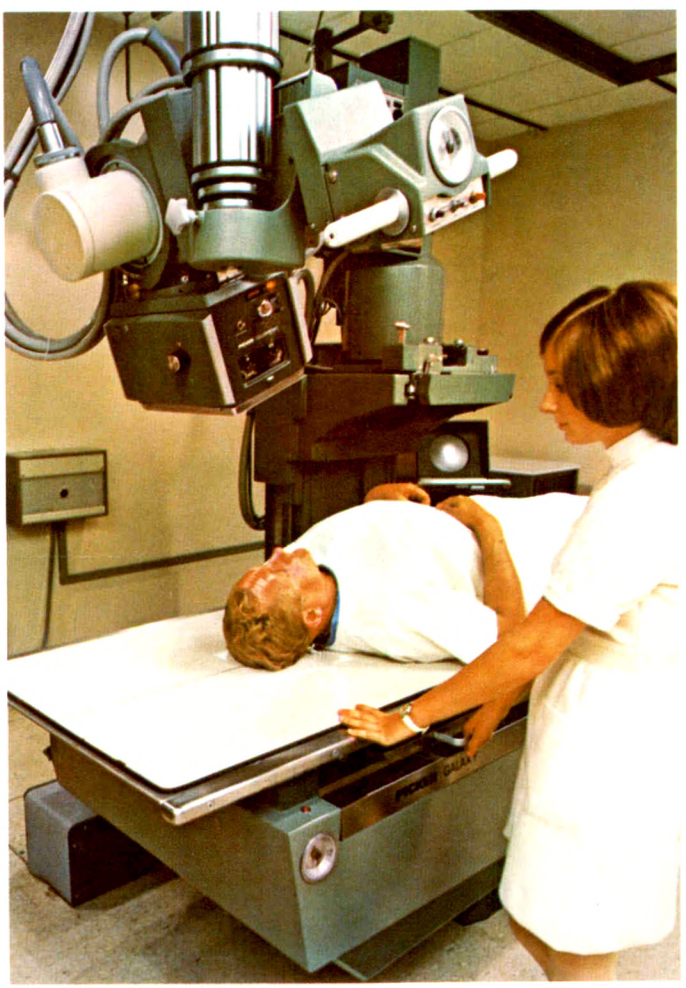
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The ultimate aim of the Act is to reduce patient exposure to only that amount of radiation necessary for accurate diagnosis. Recently proposed rules under the Act require our industry to certify that equipment manufactured for use in the USA after August 1, 1974 is capable of meeting certain standards of performance.

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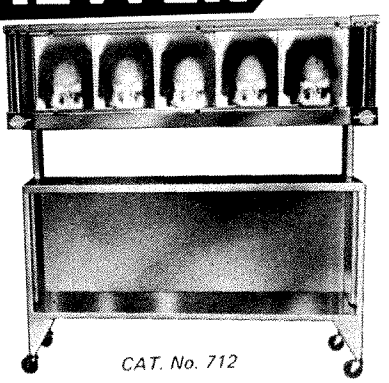
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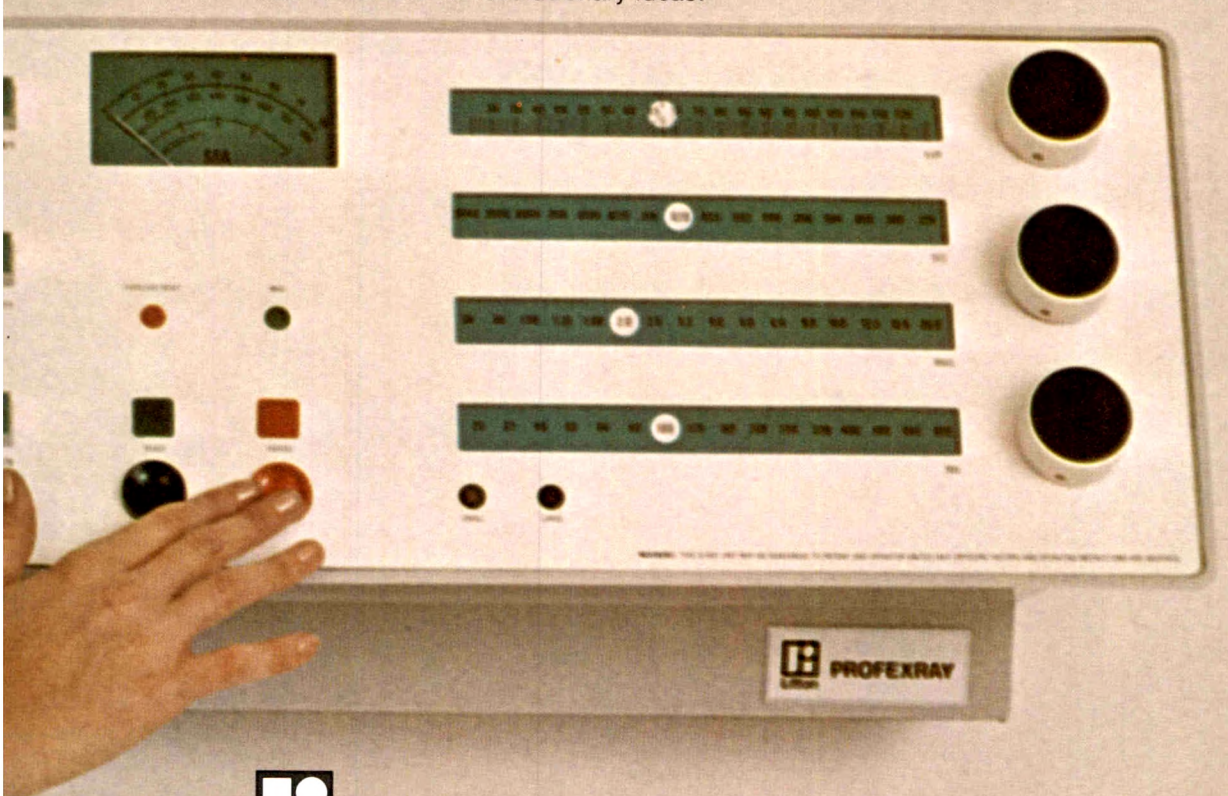
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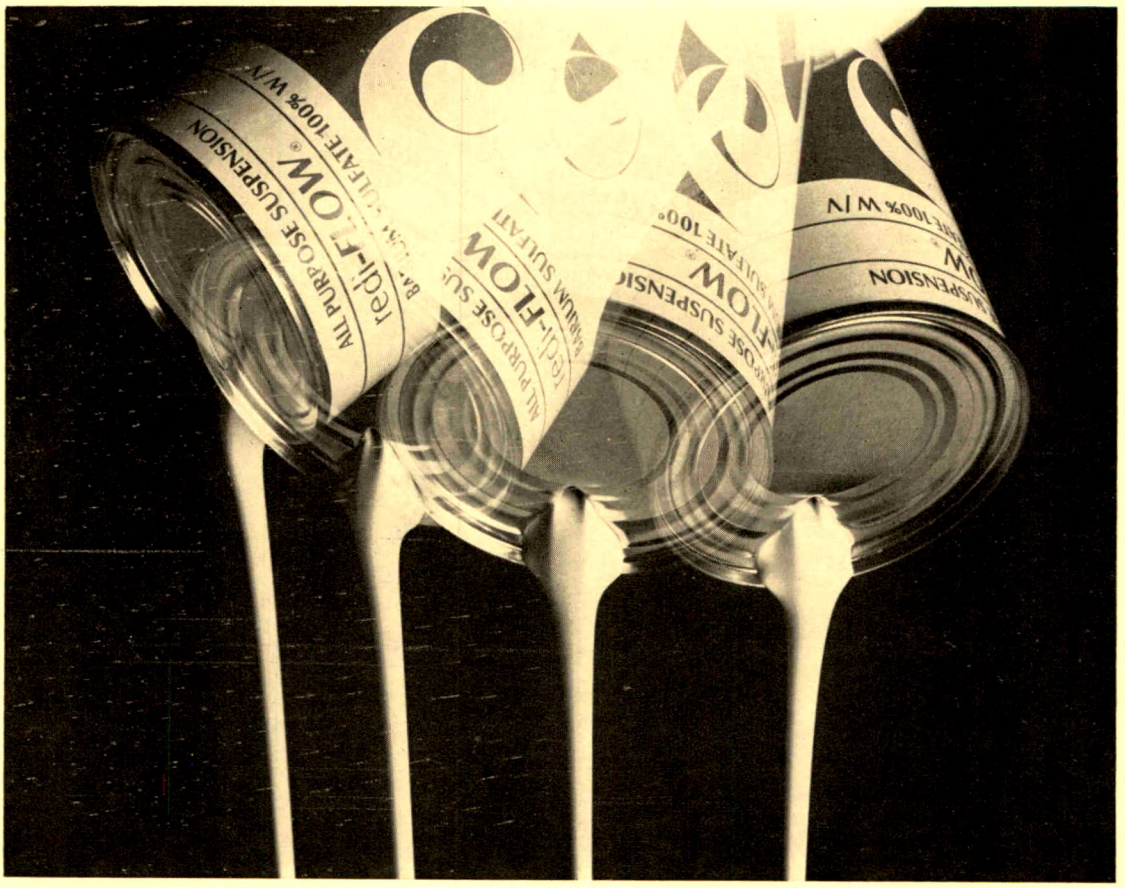
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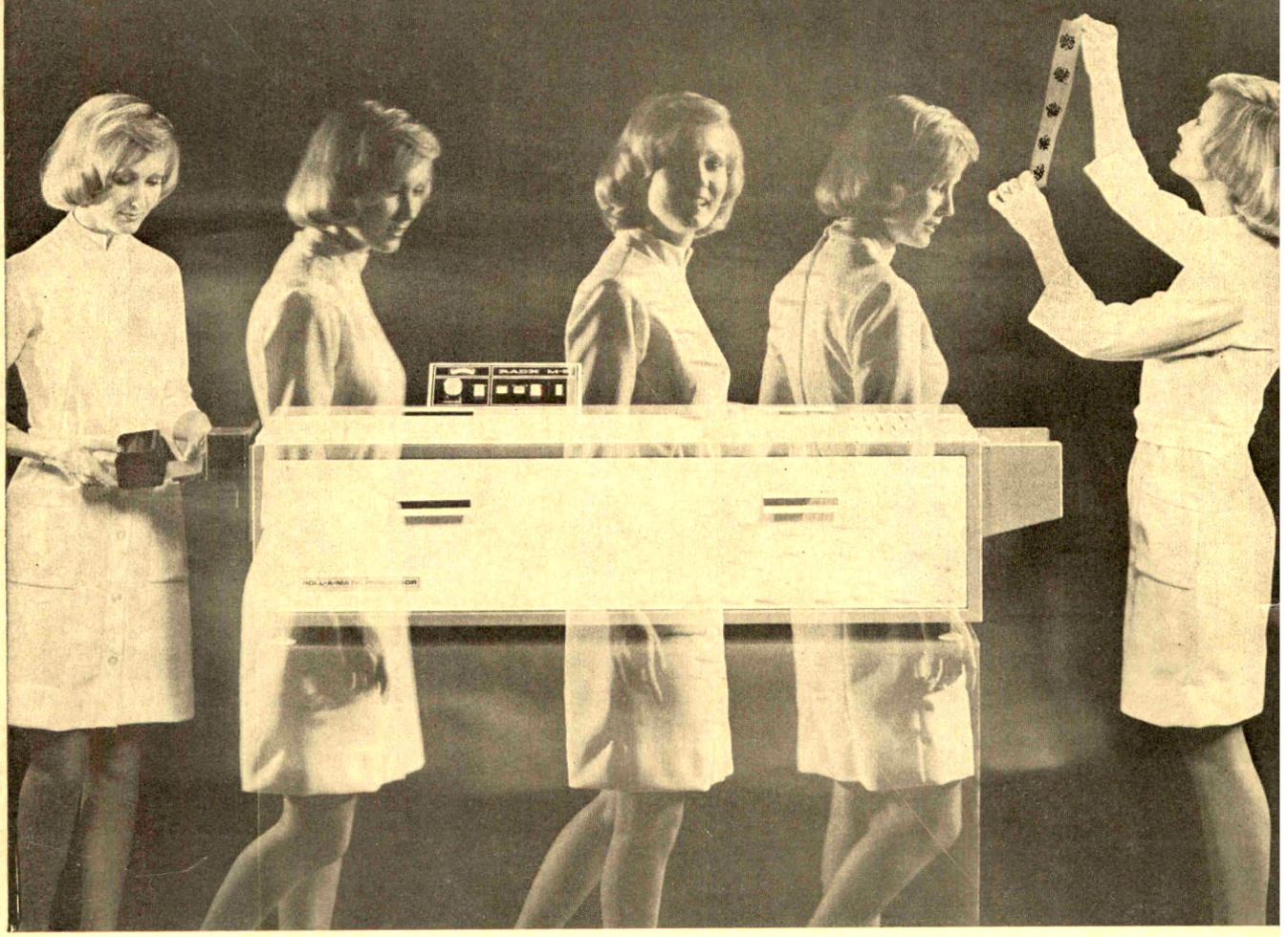
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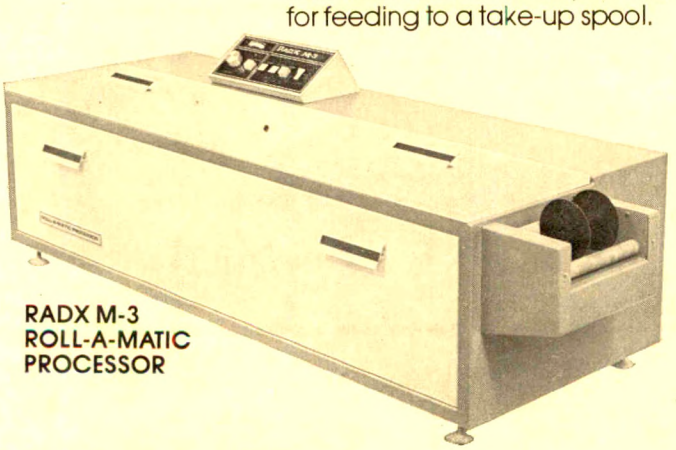


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Almost any designer of x-ray equipment could say that. Many often do — or something like it. But not so many are willing (or able) to practice what they preach.

Leo Sidorewicz is part of the design team at Profexray. He's spent many years *working* on simplicity, instead of just talking about it. And succeeded again, with another technological breakthrough.

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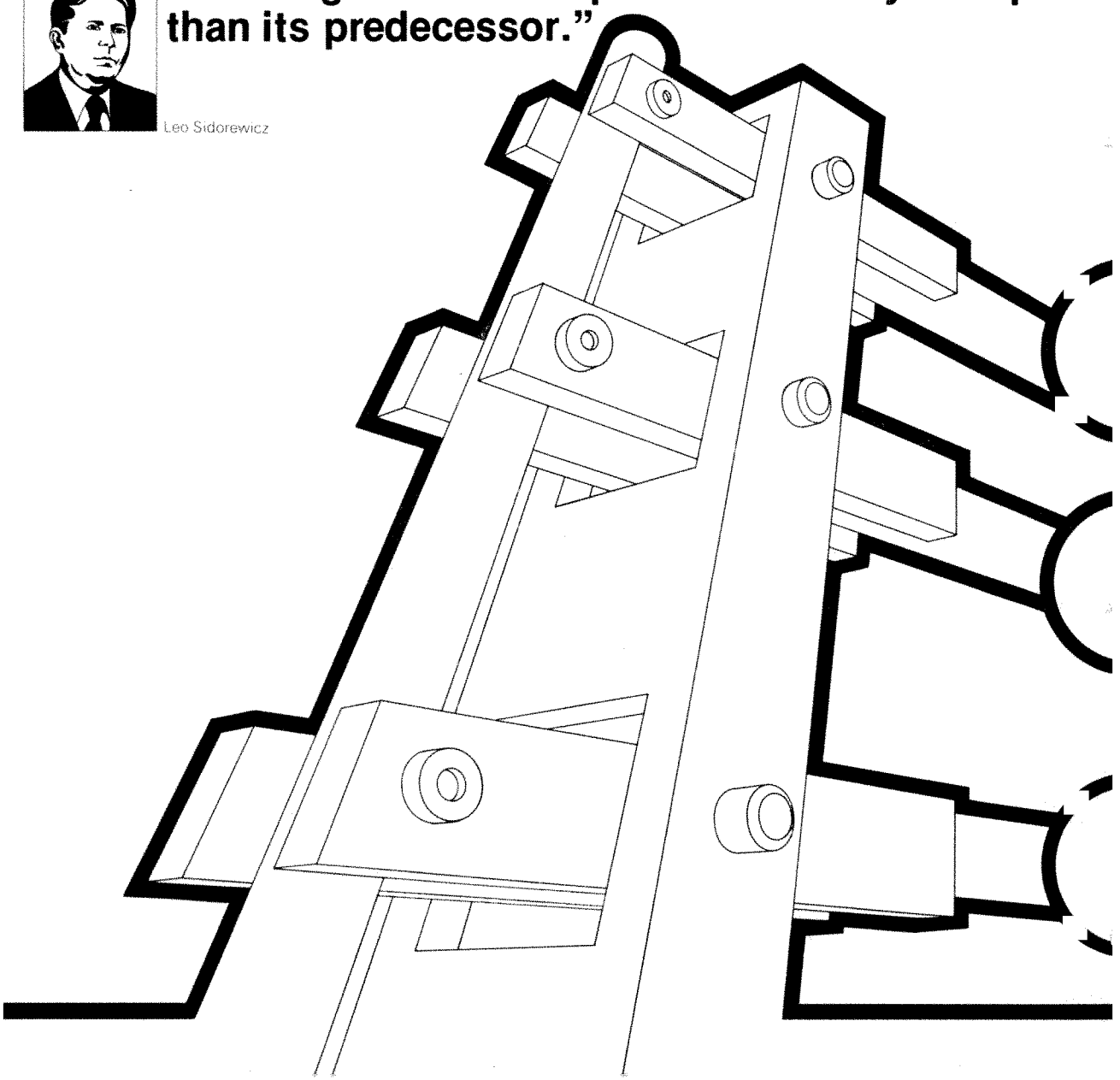
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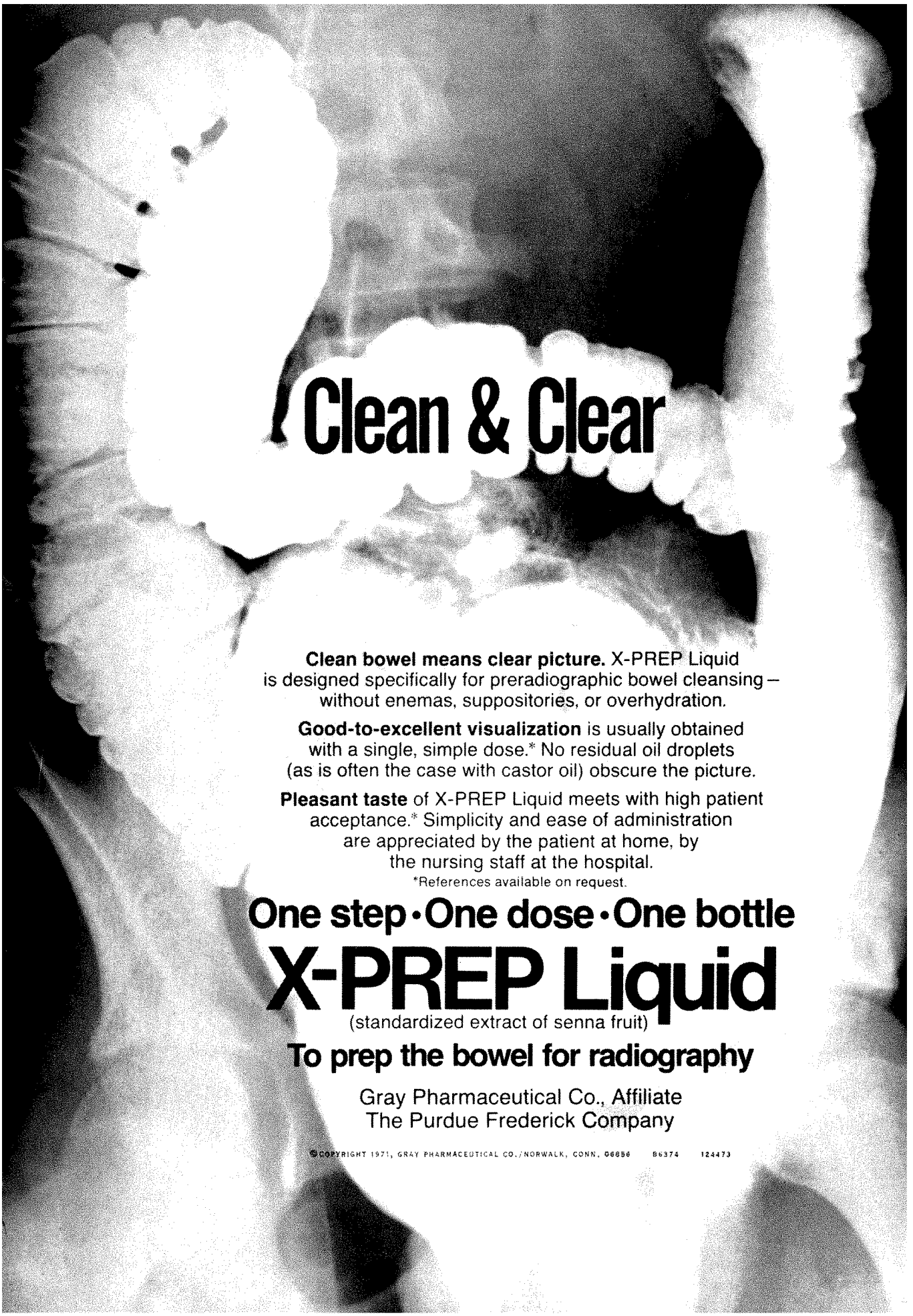
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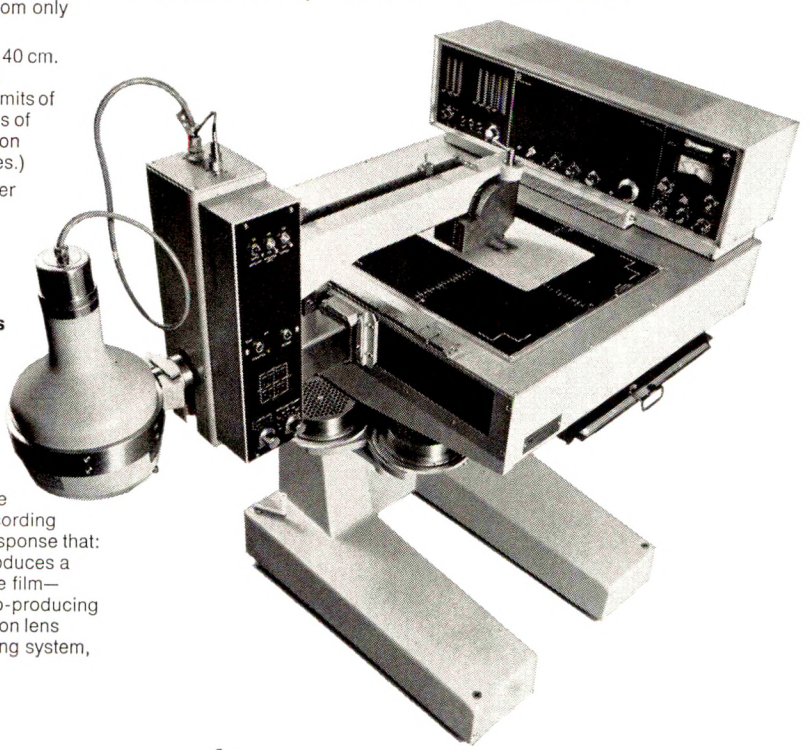
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THE PHYSICS OF RADIOLOGY (3RD ED., 3RD PTG.) *by* **HAROLD ELFORD JOHNS** *and* **JOHN ROBERT CUNNINGHAM**, *both of the Univ. of Toronto, Canada.* '74, about 812 pp. (6 3/4 x 9 3/4), 398 il.

"The new edition to this excellent book again represents an outstanding contribution to the literature on radiation physics. Physicists engaged in this field will find it to be a superbly clear exposition of their subject – especially complete in topics related to radiotherapy physics. The treatment of the subject is essentially concerned with clinical radiation physics and the book therefore is also recommended for radiotherapists and radiologists who wish to understand the physical aspects of their subject. It is well written and produced and as pleasurable to read as a first class text book should be." – CLINICAL RADIOLOGY

THE BIOLOGICAL AND CLINICAL BASIS OF RADIOSENSITIVITY *edited by* **MILTON FRIEDMAN**, *Instituto Regina Elena, Rome, Italy. (35 Contributors)* '73, about 589 pp. (7 x 10), 373 il., 67 tables

To reduce the gap between laboratory and clinical schools, authorities from the fields of radiobiology, radiation pathology, preclinical radiobiology and experimental clinical radiotherapy are asked to relate their investigations to the common denominators to radiosensitivity and chemosensitivity. The pertinent is clarified; the unproven is defined; and irrelevant concepts are eliminated. Topics discussed include: changes produced by irradiation at the molecular level; recovery from irradiation; factors like DNA strand breaks which can influence radiosensitivity; and clinical concepts of radiosensitivity such as tumor lethal dose.

RADIOASSAY IN CLINICAL MEDICINE *by* **WILLIAM T. NEWTON** *and* **ROBERT M. DONATI**, *both of Washington Univ. School of Medicine, Saint Louis.* '73, about 147 pp., 41 il., 24 tables

The advent of radionuclides that can be measured in extremely low chemical concentrations and techniques to attach them to many compounds as trace labels have opened new approaches to quantitative study. However, in spite of a well-informed public, the editors of this volume have the impression that there is lack of appreciation of both the variety of approaches and classes of compounds that can be examined. Consequently, this short volume is a set of papers chosen to illustrate not only this variety but to detail individual systems in order that the reader might gain insight into the problems inherent in each.

ROENTGEN EXAMINATIONS IN ACUTE ABDOMINAL DISEASES (3RD ED.) *by* **J. FRIMANN-DAHL**, *Ulleval Hospital, Oslo, Norway.* '73, about 580 pp., 614 il. (4 in full color)

This third edition with 133 new illustrations is mainly concerned with roentgen anatomy of the abdomen and diagnosis of the most important acute abdominal diseases. Diagnosis of lesions, emergencies requiring immediate diagnosis, and the important role of roentgen examination in correct diagnosis and treatment are among the topics discussed. Acute abdominal diseases often require instant treatment, and, since a prolonged discussion of cases is often not feasible, this book will be a valuable information source for radiologists, students in diagnostic radiology, and surgeons in emergency medicine to have close at hand.

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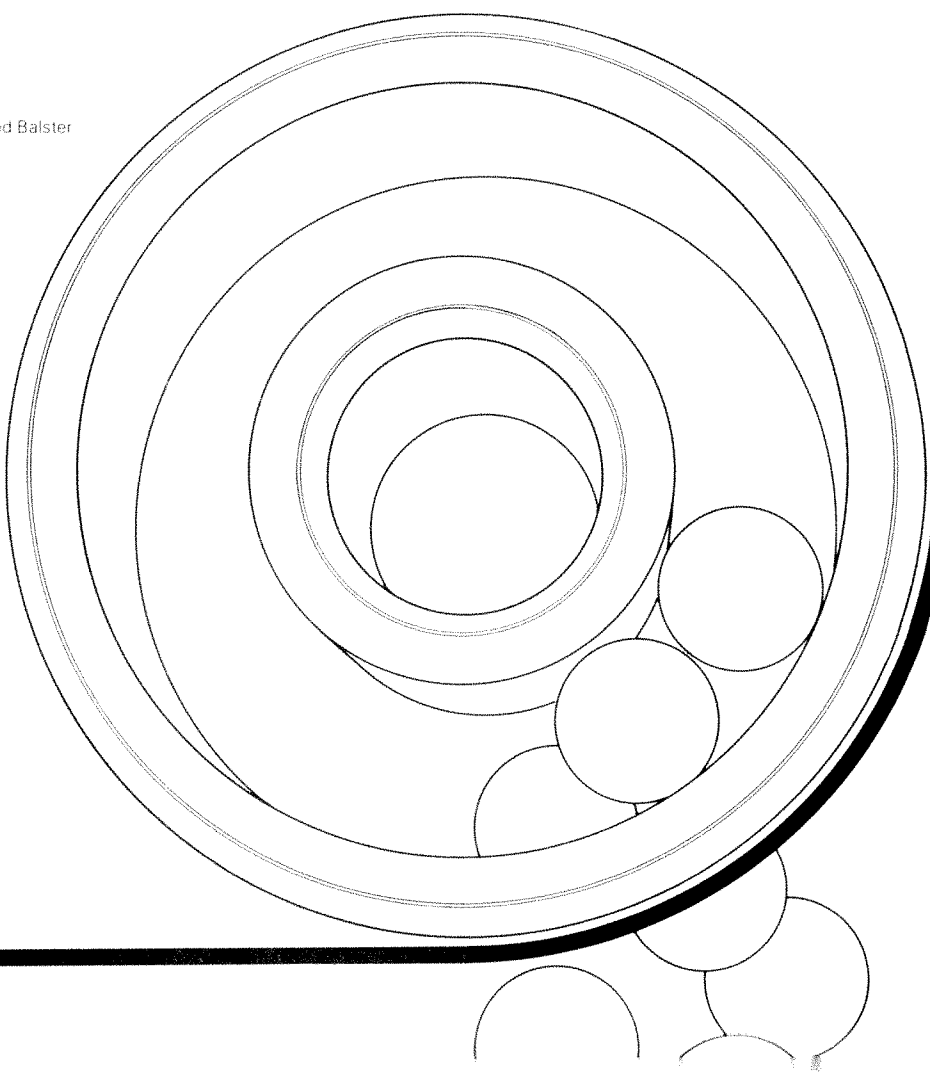
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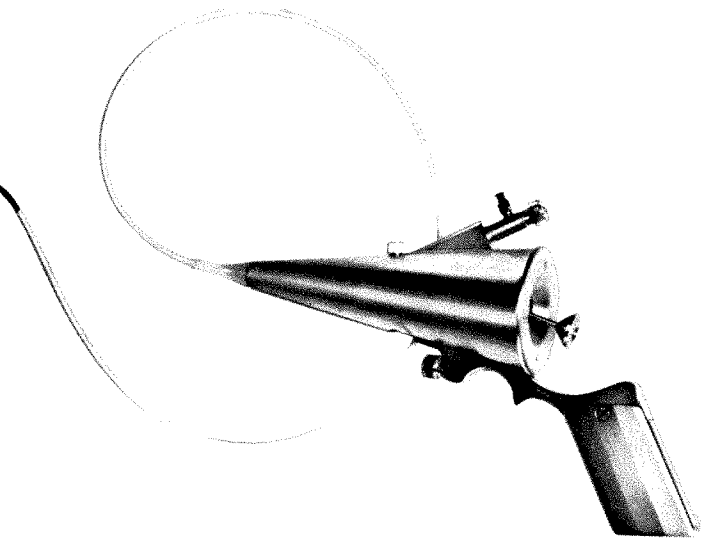
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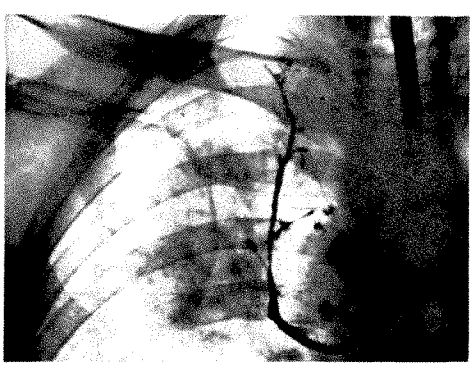
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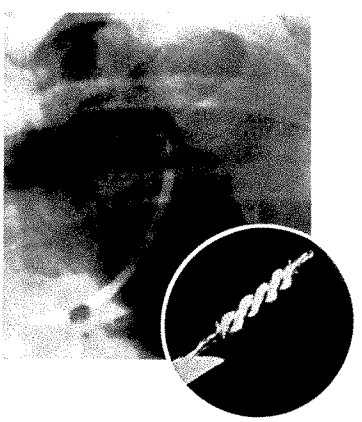
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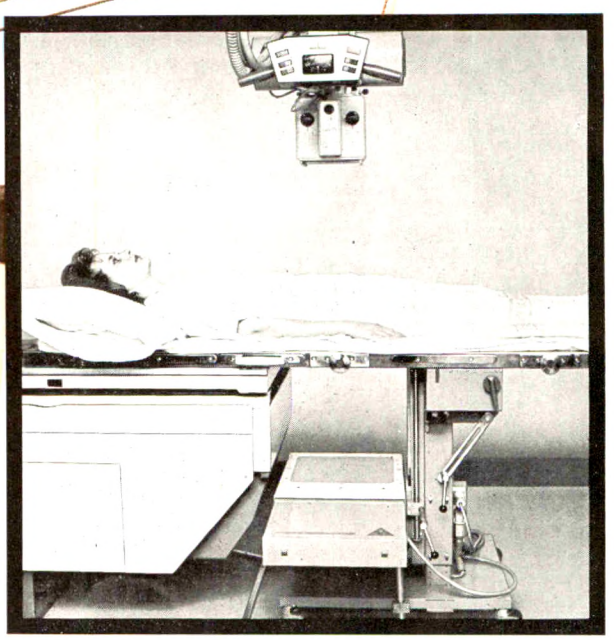
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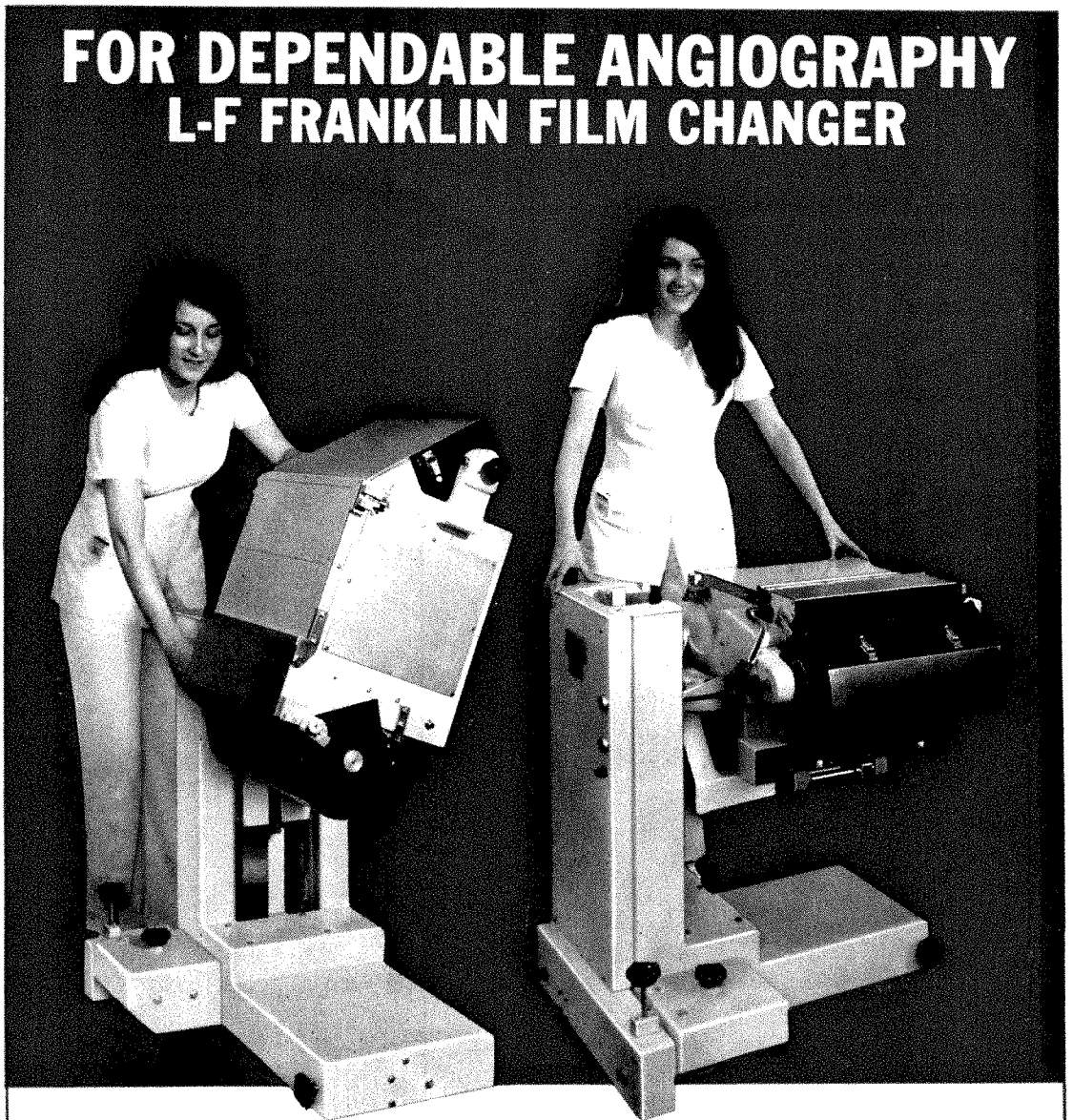
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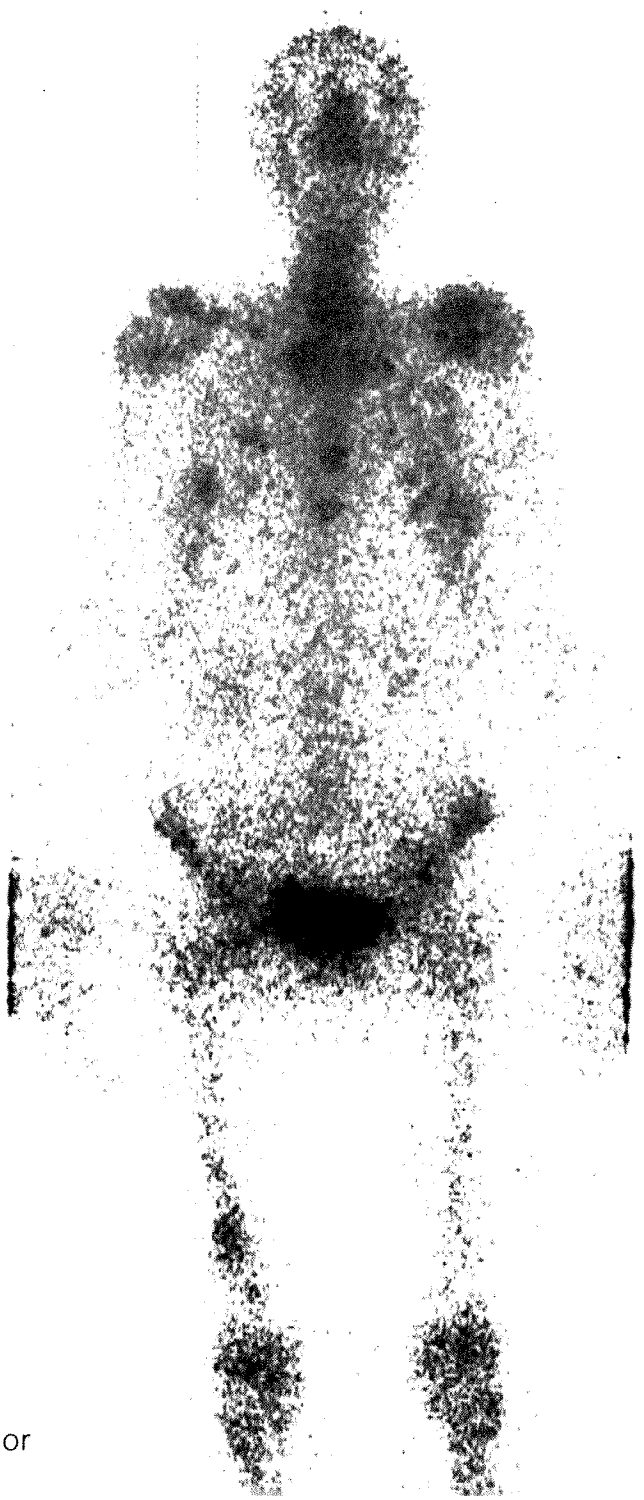
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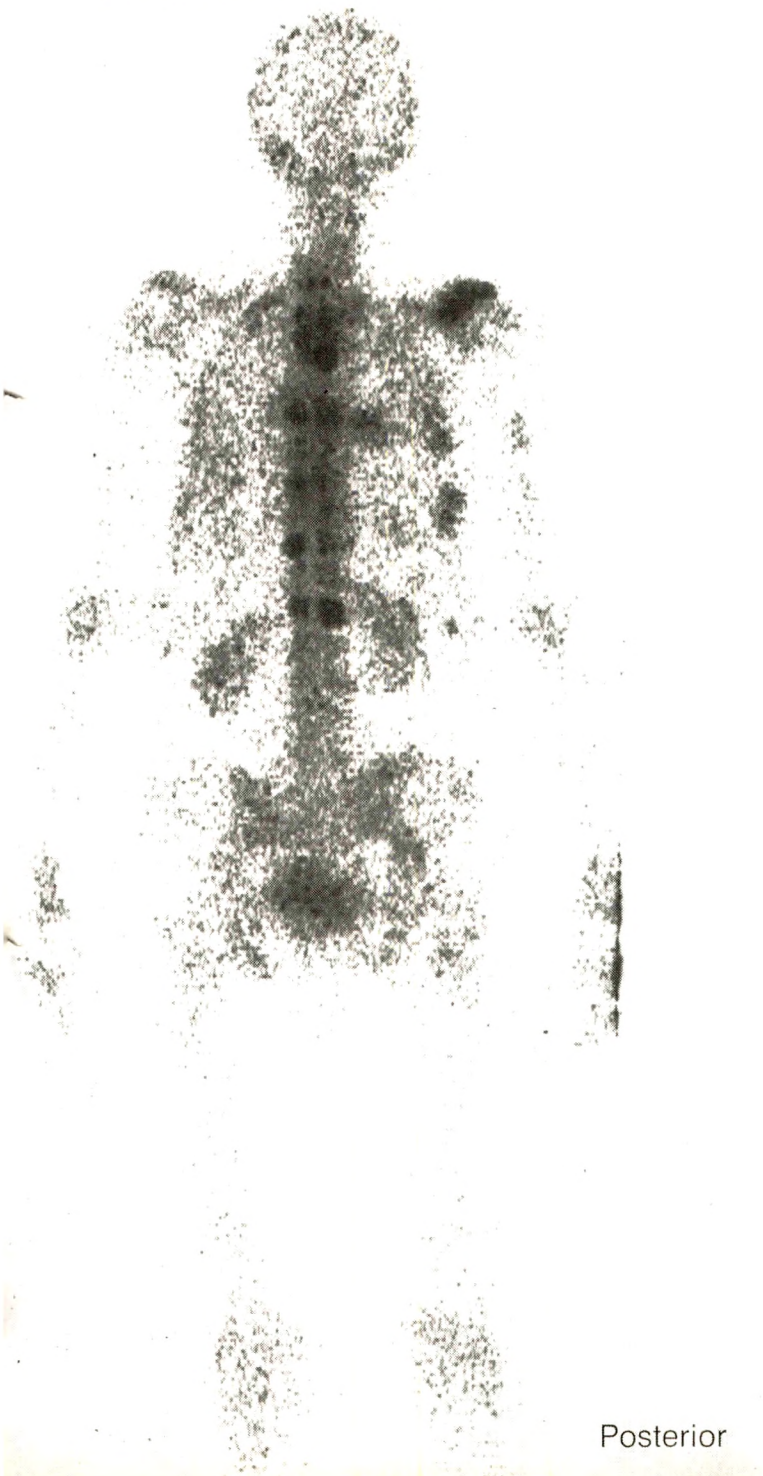
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- High bone affinity
- Low soft tissue uptake
- High labeling efficiency
- Dry mix formula
- Stable *in vivo*

SAFETY

- Sterile and pyrogen-free
- Well-tolerated; no contraindications
- Minimal patient radiation exposure

EASE OF USE

- 6-month shelf life at room temperature no refrigeration required
- Technetium-labeled
- Can be used with either scanner or camera

The use of Osteoscan, when labeled with technetium-99m, is now classified as a well-established diagnostic procedure. Osteoscan is available to properly licensed radiology and nuclear medicine departments.

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PROCTER & GAMBLE

OSTEOSCAN
(5.9 MG DISODIUM ETIDRONATE 0.16 MG STANNOUS CHLORIDE)
SKELETAL IMAGING AGENT

Each vial of OSTEOSCAN contains 5.9 mg disodium etidronate and 0.16 mg stannous chloride as active ingredients. Upon addition of ADDITIVE-FREE ^{99m}Tc-pertechnetate, these ingredients combine with ^{99m}Tc to form a stable soluble complex.

ACTIONS (CLINICAL PHARMACOLOGY)

When injected intravenously, ^{99m}Tc-labeled OSTEOSCAN has a specific affinity for areas of altered osteogenesis. Areas of bone which are undergoing neoplastic invasion often have an unusually high turnover rate which may be imaged with ^{99m}Tc-labeled OSTEOSCAN. Three hours after intravenous injection of 1 ml ^{99m}Tc-labeled OSTEOSCAN, an estimated 40-50% of the injected dose has been taken up by the skeleton. At this time approximately 50% has been excreted in the urine and 6% remains in the blood. A small amount is retained by the soft tissue. The level of ^{99m}Tc-labeled OSTEOSCAN excreted in the feces is below the level detectable by routine laboratory techniques.

INDICATIONS

OSTEOSCAN is a skeletal imaging agent used to demonstrate areas of altered osteogenesis.

CONTRAINDICATIONS

None.

WARNINGS

This radiopharmaceutical should not be administered to patients who are pregnant or lactating unless the information to be gained outweighs the potential hazards.

Ideally, examinations using radiopharmaceuticals, especially those elective in nature, of a woman of childbearing capability should be performed during the first few (approximately 10) days following the onset of menses.

Radiopharmaceuticals should be used only by physicians who are qualified by specific training in the safe use and handling of radionuclides produced by nuclear reactor or particle accelerator and whose experience and training have been approved by the appropriate government agency authorized to license the use of radionuclides.

The ^{99m}Tc-generator should be tested routinely for molybdenum breakthrough and aluminum. If either is detected, the eluate should not be used.

PRECAUTIONS

Both prior to and following ^{99m}Tc-labeled OSTEOSCAN administration, patients should be encouraged to drink fluids. Patients should void as often as possible after the ^{99m}Tc-labeled OSTEOSCAN injection to minimize background interference from accumulation in the bladder and unnecessary exposure to radiation.

As in the use of any other radioactive material, care should be taken to insure minimum radiation exposure to the patient, consistent with proper patient management, and to insure minimum radiation exposure to occupational workers.

ADVERSE REACTIONS

None.

DOSAGE AND ADMINISTRATION

The recommended adult dose of ^{99m}Tc-labeled OSTEOSCAN is 1 ml with a total activity range of 10-15 mCi. ^{99m}Tc-labeled OSTEOSCAN should be given intravenously by slow injection over a period of 30 seconds within three (3) hours after its preparation. Optimum scanning time is 3-4 hours postinjection.

The patient dose should be measured by a suitable radioactivity calibration system immediately prior to administration.

PHYSICAL CHARACTERISTICS

Technetium-99m decays by isomeric transition with a physical half-life of 6 hours[1]. Photons that are useful for imaging studies are listed in Table 1.

Table I. Principal Radiation Emission Data

Radiation	Mean % / Disintegration	Mean Energy (keV)
M int. con. electron, γ-1	98.6	1.7
Gamma-2	88.3	140.5
K int. con. electron, γ-2	8.8	119.5
L int. con. electron, γ-2	1.1	137.7
Gamma-3	0.03	142.7
K int. con. electron, γ-3	0.96	121.7
Kα X-rays	6.5	18.4

[1]Dillman, L.T., Radionuclide Decay Schemes and Nuclear Parameters for Use in Radiation-Dose Estimation, Supplement No. 2, MIRD pamphlet No. 4, *J. Nucl. Med.*, p.22, 1969.

The specific gamma ray constant for ^{99m}Tc is 0.72 R/mCi-hr at 1 cm. The half-value layer is 4 mm of Pb.

To correct for physical decay of this radionuclide, the fractions that remain at selected intervals after the time of calibration are shown in Table II.

Table II. Physical Decay Chart; ^{99m}Tc, half-life 6 hours

Hours	Fraction Remaining	Hours	Fraction Remaining
−5	1.779	5	.562
−4	1.587	6	.500
−3	1.414	7	.446
−2	1.260	8	.397
−1	1.122	9	.354
0*	1.000	10	.315
1	.891	11	.281
2	.794	12	.250
3	.707	18	.125
4	.630	24	.063

*Calibration time

RADIATION DOSIMETRY

The estimated absorbed radiation doses[1] to an average patient (70 kg) from an intravenous injection of a maximum dose of 15 millicuries of ^{99m}Tc-labeled OSTEOSCAN are shown in Table III. For comparison, the estimated radiation doses from a maximum dose of 4 millicuries of ^{18}F used as a bone imaging agent are also included.

Table III. Radiation Doses

Tissues	Absorbed Radiation Dose	
	^{99m}Tc-OSTEOSCAN (rads/15 mCi)	^{18}F (rads/4 mCi)
Skeleton*	0.59	0.64
Testes	0.32	0.83
Ovaries	0.33	0.85
Total Body	0.13	0.18
Bladder 4.8 hour void	8.4	
Bone Marrow	0.14	

*Local dose may be a factor of 10 or more greater.

[1]Method of Calculation: A Scheme for Absorbed-Dose Calculations for Biologically Distributed Radionuclides, Supplement No. 1, MIRD pamphlet No.1, *J. Nucl. Med.*, p.7, 1968.

HOW SUPPLIED

The OSTEOSCAN kit contains five (5) vials Each vial contains 5.9 mg disodium etidron and 0.16 mg stannous chloride as active ingredients. The contents of each vial are prepared by appropriate manufacturing procedures to be sterile and pyrogen-free.

PREPARATION FOR USE

The following aseptic procedure should be followed in the preparation of the ^{99m}Tc-labeled OSTEOSCAN skeletal imaging age

STEP 1.

Remove central metal disc of the OSTEOSC vial and swab the top of the vial with alcohc to sterilize the surface of the closure.

STEP 2.

Place the OSTEOSCAN vial in a radiation shield. In a sterile syringe, collect 5 ml of sterile pyrogen-free ^{99m}Tc-pertechnetate fr an additive-free ^{99m}Tc-pertechnetate sourc which has been checked for molybdenum breakthrough. Check the activity of the ^{99m} pertechnetate to avoid exceeding 50-75 mCi/5 ml. If the activity exceeds this level, dilute with ADDITIVE-FREE sterile saline o such that a 5 ml portion will contain the 50 mCi activity.

STEP 3.

Add the ^{99m}Tc-pertechnetate to the vial. Af adding the ^{99m}Tc-pertechnetate to the vial, withdraw an equivalent amount of air to equalize the pressure inside the vial to prevent spray contamination. CAUTION: DO NOT USE ^{99m}Tc-PERTECHNETATE WHICH CONTAINS AN OXIDIZING AGENT. INTRO DUCTION OF AN OXIDANT MAY RESULT II SOLUTION UNSUITABLE FOR SKELETAL IMAGING. Commercial sources of ^{99m}Tc-pertechnetate that have been used in clinic trials with OSTEOSCAN include the New England Nuclear Technetium-99m Generat the Mallinckrodt Technetium-99m Generato the Squibb Hi-Con Generator, Medi+Physi Instant Technetium, and Cambridge Nuclea Instant Technetium.

STEP 4.

Shake the vial well for three (3) minutes to assure complete dissolution of the contents Minimal exposure can be obtained by use c either an ultrasonic agitator or mechanical shaker.

STEP 5.

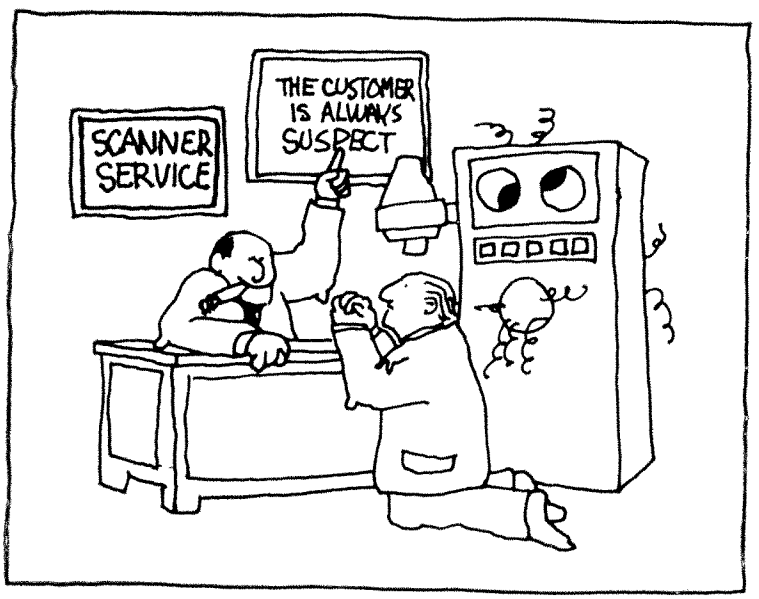
Record the time and date of preparation an the activity of the ^{99m}Tc-labeled OSTEOSCAN on the radiation shield label contained in the kit and affix this label to th shield.

STEP 6.

Use within three (3) hours of preparation. Discard excess material.



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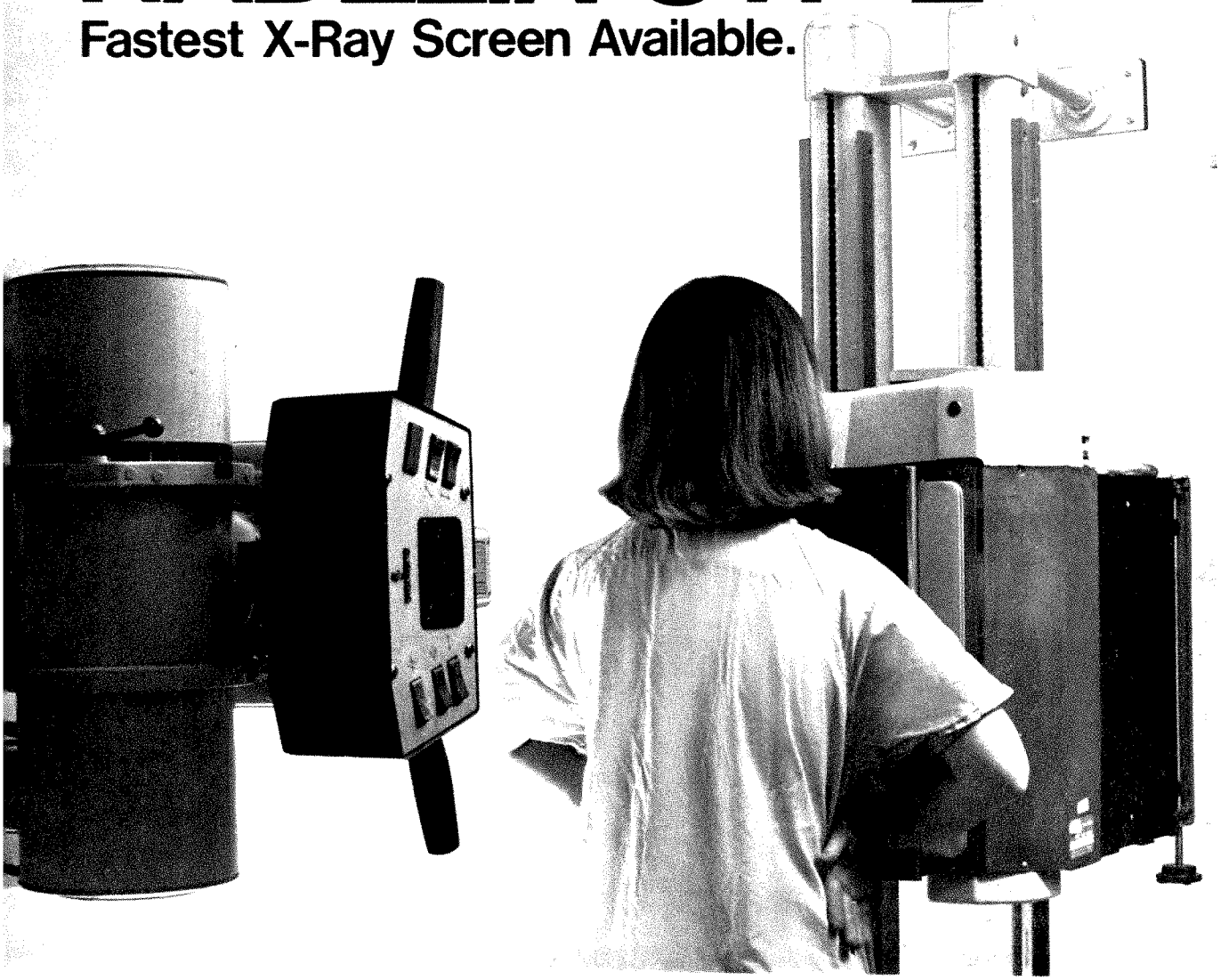
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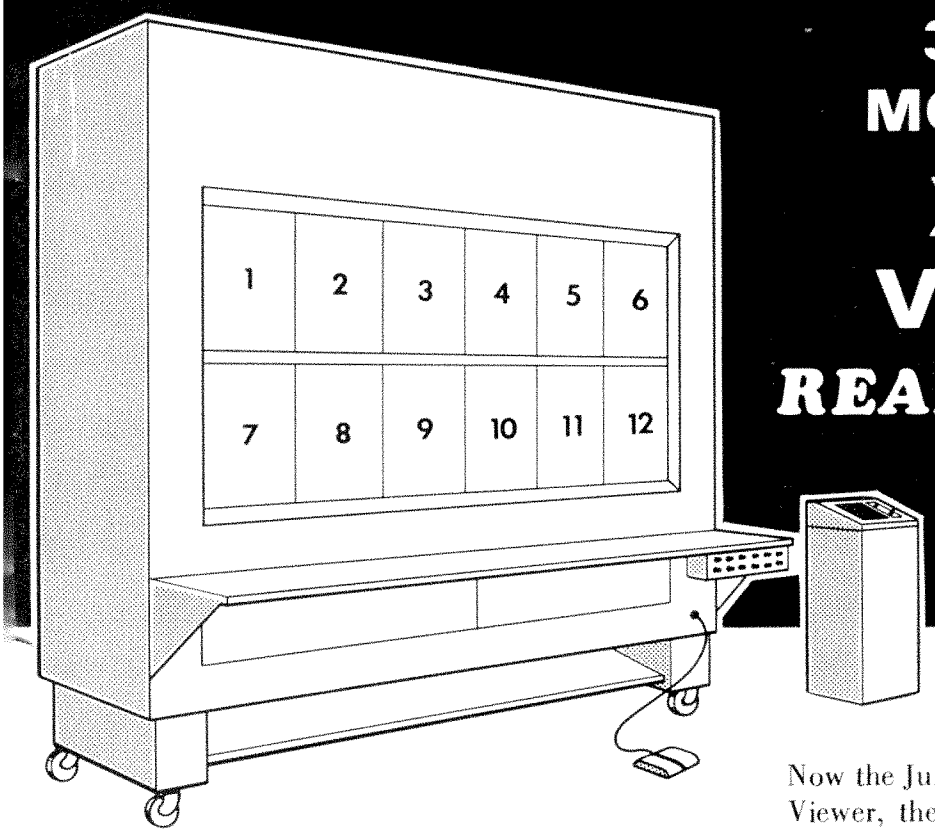
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Action: Hypaque-DIU 30 per cent is a diagnostic radiopaque medium which provides visualization of internal structures.

Indication: Hypaque-DIU 30 per cent is indicated for infusion urography.

Warnings: *Usage in Pregnancy.* No teratogenic effects attributable to a 60 per cent solution of Hypaque meglumine have been observed in reproduction studies in animals. However, before administration of Hypaque-DIU 30 per cent to women of child-bearing potential, the benefit to the patient should be carefully weighed against the possible risk to the fetus. In addition, most authorities consider elective contrast radiography of the abdomen contraindicated during pregnancy.

Serious or fatal reactions have been associated with the administration of radiopaque media. It is of utmost importance that a course of action be carefully planned in advance for the immediate treatment of serious reactions, and that adequate and appropriate facilities be readily available in case a severe reaction occurs.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravascularly.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake and direct thyroxine assays, are not affected.

Precautions: Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered. **Diphenhydramine hydrochloride (i.e., Benadryl®), however, should not be mixed with Hypaque-DIU 30 per cent since it may cause precipitation.** The intravenous injection of a test dose of 0.5 to 1 ml. of the contrast agent before infusion of the full dose has been employed in an attempt to predict severe or fatal adverse reactions. The preponderance of recent scientific literature, however, now demonstrates that this provocative test procedure is not reliably predictive of serious or fatal reactions. Severe reactions and fatalities have occurred with the test dose alone, with the full dose after a nonreactive test dose, and with or without a history of allergy. No conclusive relationship between severe or fatal reactions and antigen-antibody reactions or other manifestations of allergy has been established. A history of allergy may be useful in predicting reactions of a mild or intermediate nature.

Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations, or of sensitivity to iodine.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure, it is essential to omit preparatory dehydration, to administer fluids, and attempt to alkalinize the urine.

Immediate adverse reactions to infusion urography have not been reported to occur at a higher frequency or with greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Infusion urography imposes a sudden osmotic load. This should be considered in patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, there is a possibility of the development of congestive heart failure hours after the procedure.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating infusion urography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion urography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

Adverse Reactions: Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

The following have been reported after administration of diatrizoates and other iodinated contrast media. *Cardiovascular reactions:* vein cramp and thrombophlebitis following intravenous injection, rare cases of cardiac arrhythmias, hypertension, hypotension and shock, and cardiac arrest. *Allergic reactions:* asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria, and rarely, anaphylactic reactions. Signs and symptoms related to the *respiratory system:* pulmonary or laryngeal edema, bronchospasm; or to the *nervous system:* restlessness, convulsions. *Other reactions:* flushing, pain, warmth, nausea, vomiting, anxiety, headache, dizziness, hematoma, and ecchymosis. Occasionally, transient proteinuria, and rarely, oliguria or anuria. Rarely, immediate or delayed rigors can occur, sometimes accompanied by hyperpyrexia. Infrequently, "iodism" (salivary gland swelling) from organic iodinated compounds appears two days after exposure and subsides by the sixth day.

Dosage and Administration: The recommended dose for infusion urography is calculated on the basis of 2 ml. of Hypaque-DIU 30 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimal results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

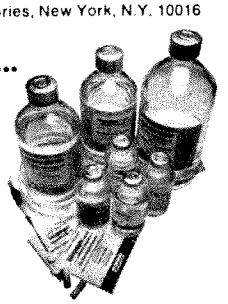
The solution is infused over a period of three to ten minutes. Radiographs are usually taken at 10, 20, and 30 minutes from the beginning of the infusion. Earlier films may be obtained for the evaluation of hypertension. Nephrograms are usually obtained at the end of the infusion, and voiding cystourethrograms are usually obtained 30 minutes after the beginning of the infusion.

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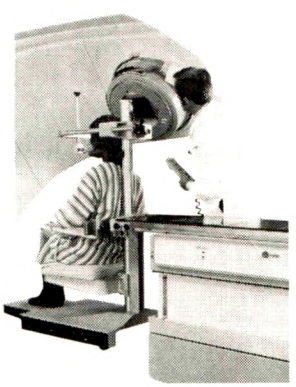


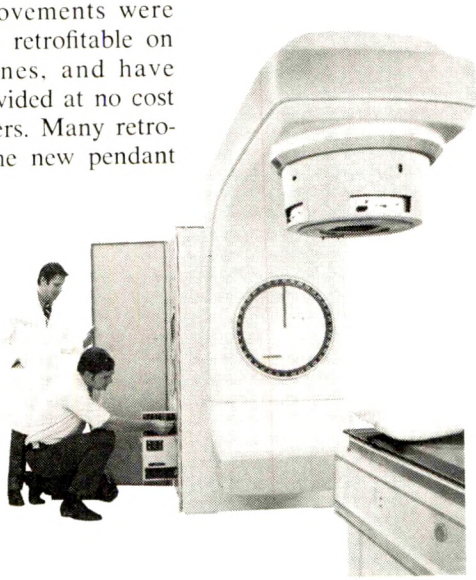
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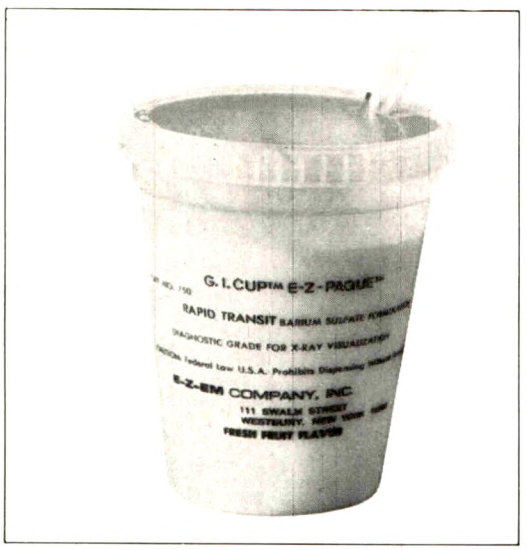
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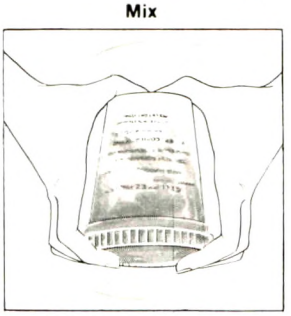
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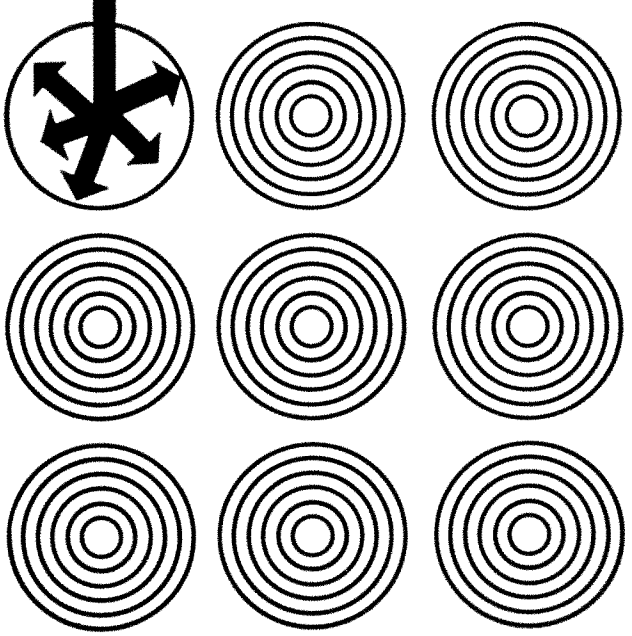
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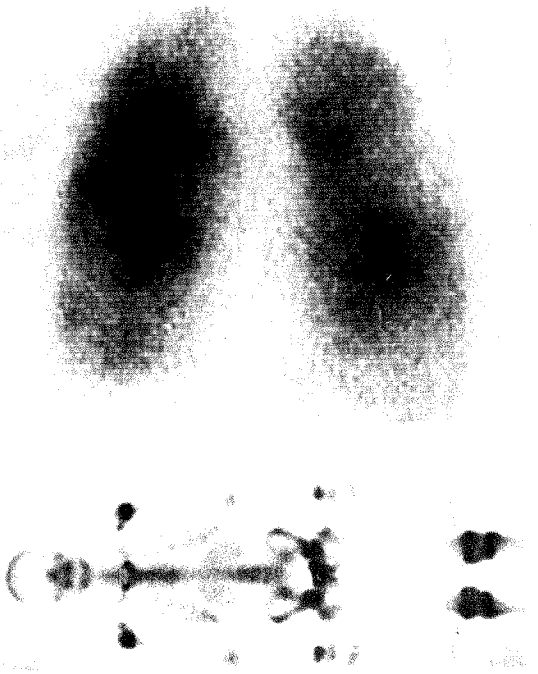
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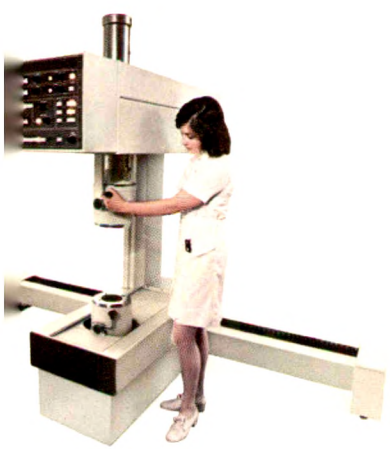
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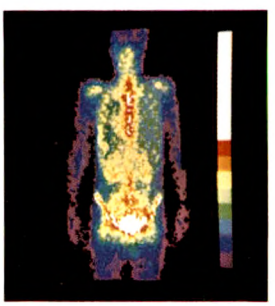
Here's the informatio hospitals are getting with Maxiscan...

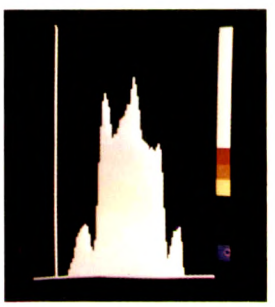
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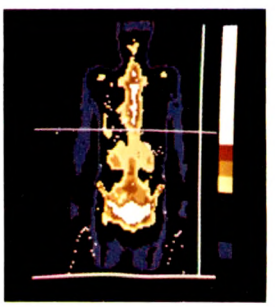
These reproductions of scans, from clinical examinations, illustrate the range of diagnostic information possible with Maxiscan and the Videodisplay Processor.

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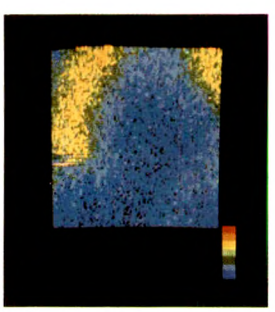


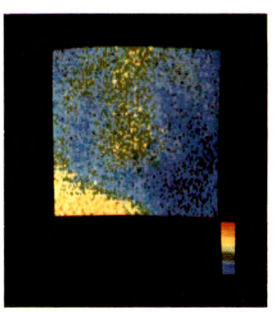


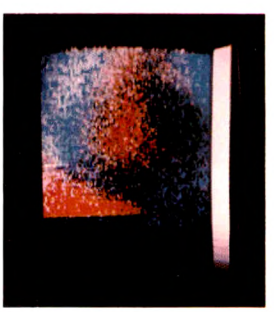




These three images, from a single whole body scan, demonstrate how manipulation of data stored in the VDP electronic memory can enhance desired details and aid diagnosis. The isotope used was ^{99m}Tc Polyphosphate. At left, an anterior view displays raw, unmanipulated data from the memory. At right, smoothed data is shown with a Y axis electronic slice through the area of suspicion. The count profile superimposed over this image and shown separately, center, confirms greater uptake on the right side. The photorecorded image showed only a suspicion of greater isotope uptake.







In a case of suspected pericardial effusion, a transmission scan (left) of the chest was obtained using an Iodine 131 source. An emission scan (center) of the same region was simultaneously obtained with the same probe, 15 minutes after an intravenous injection of ^{99m}Tc labeled albumin. The heart and liver are outlined. Note how the intracardiac activity (central area of center scan) fails to fill the large mediastinal shadow (central blue area of left scan). This discrepancy, between heart size and that of the mediastinum, is more easily seen when these two scans are superimposed (right); a technic easily accomplished on the VDP. The resulting diagnosis, a large pericardial effusion which appears to be predominantly left-sided, was confirmed by the aspiration of 1800 ml. of fluid from an encysted pericardial effusion.

Scans courtesy of Dr. M. J. Chamberlain, University Hospital, London, Ontario.

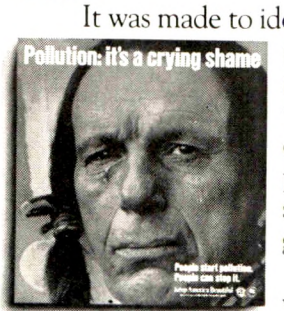
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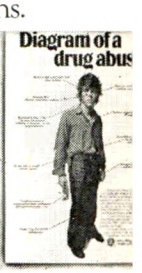


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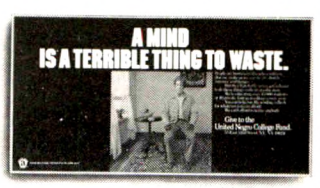




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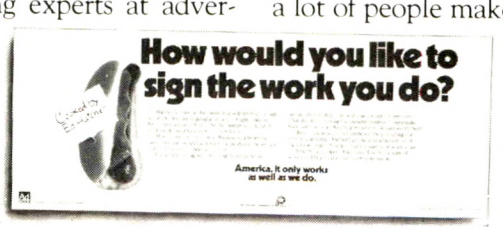
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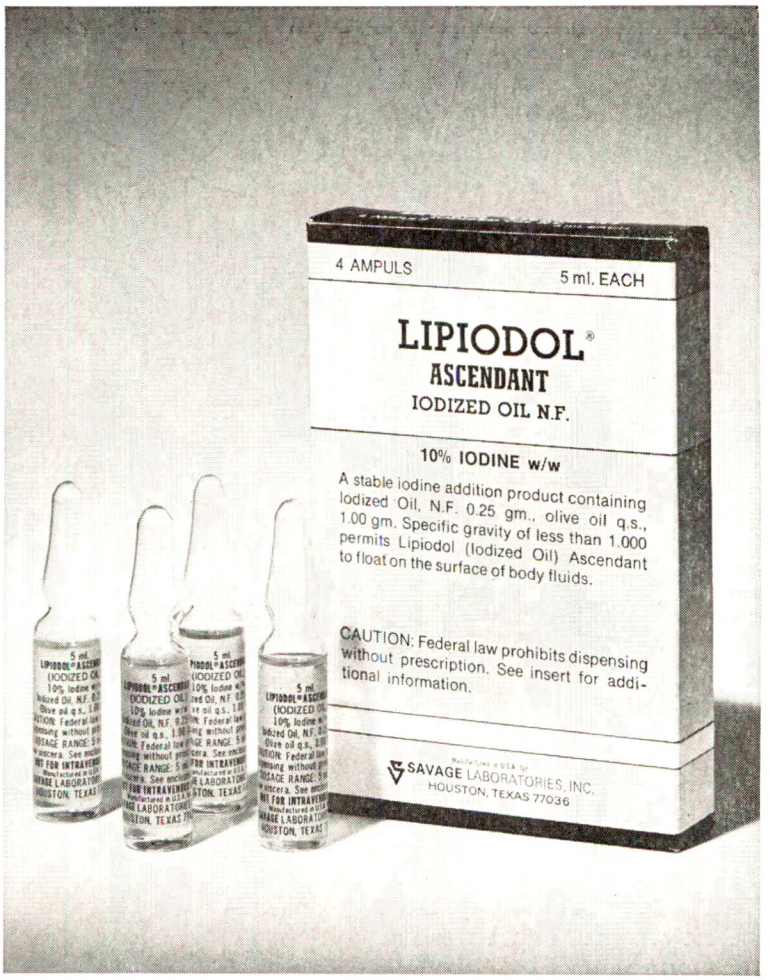
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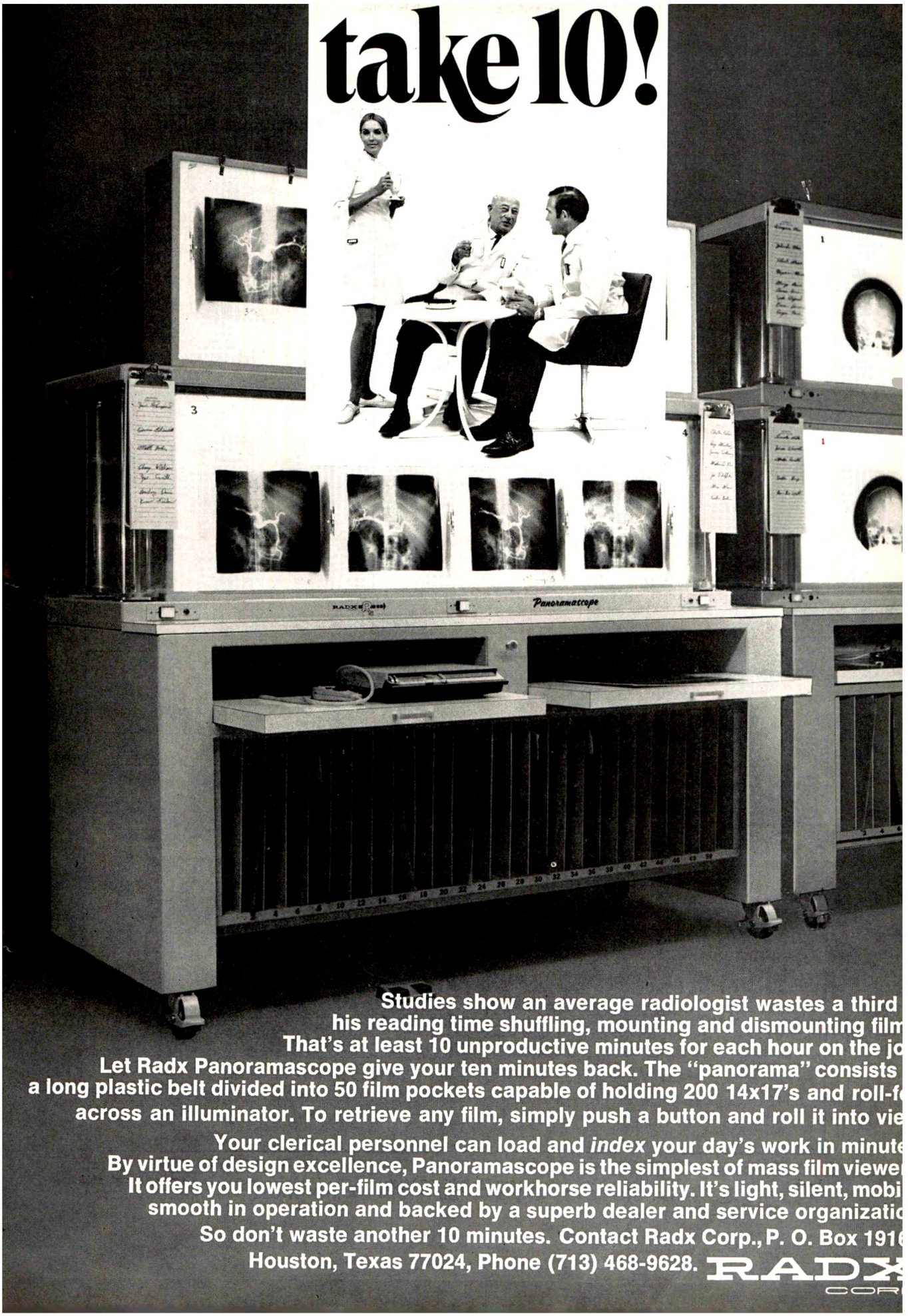
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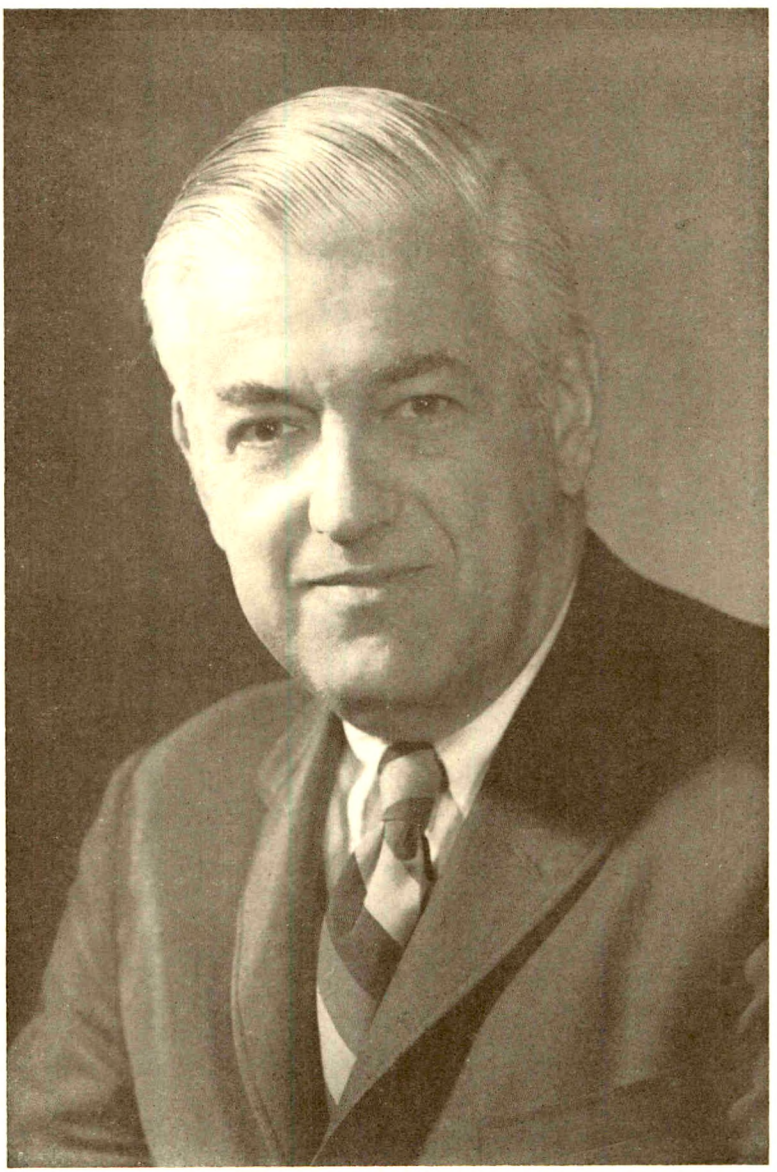
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WILLIAM B. SEAMAN, M.D.

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 119 DECEMBER, 1973 No. 4

AT THIS POINT IN TIME*

THE PRESIDENT'S ADDRESS, 1973

By WILLIAM B. SEAMAN, M.D.
NEW YORK, NEW YORK

I WISH I had the eloquence to adequately describe my appreciation for this honor of serving as President of the American Roentgen Ray Society, the oldest radiological society in North America.

The members of this distinguished society have displayed their leadership, their dedication to the scientific and educational goals of radiology for almost three-quarters of a century. I have always cherished my membership in this group and thus feel doubly privileged for having been selected to serve as your president.

Presidential addresses seem to either "point with pride" or "view with alarm." On re-reading the addresses of some of my illustrious predecessors, I noted that some of the problems pointed out in the past are no longer with us. Radiology is now well recognized as an established clinical specialty, recruitment of young physicians into radiology has been so successful that we may soon be faced with the problem of an excess. Other fears, voiced in the past, seem to be growing in intensity and complexity: for example, the socio-economic problem and the rapidly increasing role of the government and third parties in the practice of medicine. The following quotation seems appropriate:

"It was the best of times, it was the worst of times; it was the age of wisdom, it was the age of foolishness; it was the epoch of belief, it was the epoch of incredulity; it was the season of light, it was the season of darkness; it was the spring of hope, it was the winter of despair; we had everything before us, we had nothing before us."

These words, by Charles Dickens, his opening paragraph in a "Tale of Two Cities," were written to describe the year 1775, but seem to have a certain relevance today. At this point in time, when we will soon be celebrating the seventy-fifth anniversary of the American Roentgen Ray Society, it might be of interest to briefly glance back at where we have been, and then see if we can tell where we are going. Dr. Harold Reineke,[1] in his 1960 Presidential Address, included the following from Oliver Wendell Holmes, "I find that the great thing in the world is not so much where we stand as in what direction we are moving."

[1] REINEKE, H. G. Whither bound? AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *84*, 809.

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, Quebec, September 25–28, 1973.

In March 1900 a small group of midwestern physicians met in St. Louis to organize the Roentgen Ray Society of the United States and to arrange the First Annual Meeting which was held at the Grand Central Palace in New York City on December 14–15, 1900. It is reported that 150 persons attended and that 25 papers were on the program. Since we are meeting today in Montreal, it is worth noting that the name of the Society was changed the next year to the Roentgen Society of America in order to include Canadian physicians, and the second president of the Society was Dr. C. P. Girdwood of Montreal. Professor Girdwood was the first teacher of radiology at McGill University, and the first roentgenogram at the Royal Victoria Hospital was made by him in September 1901. At the Fifth Annual Meeting, held in St. Louis, September 1904, eighteen new members were elected which swelled the membership to 229. The program consisted of eleven papers, a symposium on various aspects of radiotherapy and only four papers on diagnostic radiology. In one of the latter, Dr. Preston Hickey of Detroit, Michigan, attacked the problem of identifying the numerous fine white lines "ramifying from both sides of the sternum." These had been considered to be beginning areas of consolidation, bronchial walls or interlobular pleura. Dr. Hickey, using autopsy and animal material, proved that they were pulmonary blood vessels.

How radiology has changed! In fact, the scope of radiology has been so broadened, that any definition of this specialty based upon x-rays is obsolete. The modern definition of radiology should include new diagnostic techniques utilizing systems other than the x-ray tube and the conventional roentgenogram. The latter can be considered as a system by which the differential absorption of ionizing radiations by internal structures can be displayed. Radiology should concern itself with displays of all types. Any signal which is differentially handled by various structures within the body and can be displayed in such a manner as to convey information about these structures, is properly within the range of diagnostic radiology. Thus, in scanning procedures, although utilizing the emanations emitted from internally deposited radioactive isotopes, the same principles are utilized as for a conventional x-ray. The use of pulsed ultrasound and the detection of infrared radiation are also legitimately within the province of diagnostic radiology. The future will undoubtedly provide additional techniques and it is even conceivable that the film recording system may be eliminated and information stored, sorted and retrieved utilizing computer techniques.

Diagnostic ultrasound is an exciting new branch of radiology. I would like to emphasize that I do consider it a legitimate part of radiology and think all of the radiological societies, including the American College of Radiology, and the American Board of Radiology should formally and officially acknowledge and adopt this important new subspecialty. A refresher course on ultrasound was given during last year's annual meeting and another is scheduled for this meeting. Two papers on ultrasound are included in this year's scientific program. Thus the American Roentgen Ray Society officially welcomes this new important diagnostic modality into the radiologic family.

This broadened scope of our specialty has not been an unmixed blessing. The problems imposed by the development of radiologic subspecialties and the fear of fragmentation has been a recurrent theme echoed by many of my predecessors. Dr. J. C. Cook,[2] in his Presidential Address, entitled "In Union There is Strength" emphasized the important unifying contributions made by this Society at its annual meeting.

Radiological subspecialties are not only here to stay but are essential for our continued survival as a specialty. They have materially contributed to the strength of

[2] Cook, J. C. In Union There Is Strength. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 699.

our specialty. The recent appearance of national societies of gastrointestinal, urologic, skeletal, and chest radiology and ultrasound is an indication of the popularity of this trend. Indeed there is even an International Society of Otoradiology. One of the attractions of radiology to many, and which led to its selection as a career choice, is its diversity and the fact that it involves almost every field of medicine. Radiologists in this group, which includes myself, are reluctant to confine their efforts to only a segment of this fascinating branch of medicine. Yet, I fervently believe, that such subspecialization is essential for the continued survival and growth of radiology. Radiology has become too large and too complex to be mastered by one person. If we are to avoid becoming technicians and continue our heritage as medical consultants, subspecialization must continue to grow.

A number of prominent radiotherapists were queried as to the future of radiotherapy within the American Roentgen Ray Society. With the existence of two organizations devoted entirely to radiotherapy and cancer management in general, plus the Radiation Research Society and an active program of Radiotherapy at the Radiological Society of North America, it was the feeling of many radiotherapists that the American Roentgen Ray Society should devote itself completely to roentgen diagnosis. The radiologist who practices both diagnosis and therapy seems to be a waning species and diagnostic radiology is exploding in all directions, so that this may be sound practical advice. However, we are reluctant to give up this important segment of radiology, so before following their advice we have attempted to revive interest in radiotherapy at this meeting. We are indebted to Dr. Ralph Scott, Chairman of the Executive Council, for organizing a very fine radiotherapy program which will run concurrently with the Wednesday morning session in diagnosis. However, "In Union There is Strength" according to Dr. Cook. Not only political strength, a point not to be underemphasized in today's milieu of pressure groups, professional unions and ever increasing governmental controls, but intellectual and scientific strength. How do we maintain this union in face of ever increasing subspecialty groups? We are pleased that the Society of Pediatric Radiology continues to meet in conjunction with this Society and permits everyone to attend a portion of their meeting. Perhaps other subspecialty groups should be encouraged to do likewise. Is this a trend that should be pursued still further? Should we develop a federation of radiological societies that meet consecutively or even concurrently and let registrants freely attend any program that attracts their fancy? This idea is now being carried out, and with some success, by societies interested in the gastrointestinal tract. Or should we heed the advice of the therapists and become a society whose interests are limited to general diagnostic radiology?

One of the vital issues of the day in medicine is the problem of how to cope with the spate of new developments in medicine. How do physicians remain competent and keep abreast of these explosive additions to medical knowledge. The American Roentgen Ray Society has always been a leader in postgraduate education. Indeed, article 2 of the Constitution of the Roentgen Society of the United States adopted in 1900 states "The object of this Society shall be the advancement of the knowledge of practical x-ray work and allied arts and sciences: the promotion of this branch of surgery and medical science, systematic original research, the uniformity of support to the inventive talent." The scientific program of the annual meeting represents an important annual event in radiological education.

Another important advance in continuing education was pioneered by this Society in 1939, when Dr. B. R. Kirklin, during the presidency of Dr. E. L. Jenkinson, introduced the instructional courses. I would like to pay tribute to Dr. Harold Peterson, Director of the Instructional Course Pro-

gram, for his many years of service in this important area of education and most of all, to all the teachers of these courses for their magnificent contributions to medical education. This year, 58 courses are being offered, a limit imposed more by the physical restrictions of the meeting place than demand. I don't know whether our students or horses are drinking at this fountain but I think we're at least making them thirsty. Where is our future direction in this segment of our activity? Should we continue to expand? Are we competing with the universities? Is there a need for integration and systematization of postgraduate courses on a national basis? We look to the membership for guidance.

I would be remiss if I didn't mention a most important contribution of the American Roentgen Ray Society to postgraduate education: The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, and its very able editor and past president, Dr. Traian Leucutia to whom we are all indebted for his many years of dedication and skillful editorship.

What other changes are desirable to enable the American Roentgen Ray Society to maintain its position as a prestigious, honored organization?

Last year President Dunbar appointed a committee with Dr. James Martin as Chairman, to review the entire structure of the Society, and its activities, including the annual meeting. This committee has been working very hard and its recommendations will be seriously considered. Any suggestions from members and guests will be warmly received.

The closing sentence of Lewis Mumford's book, "The Transformation of Man," is "Every goal man reaches provides a new starting point, and the sum of man's days is just a beginning." What new goals should this Society, soon to be 75 years old, be striving for?

Vol. 119, No. 4

SPLENIC AND HEPATIC EVALUATION DURING INFUSION NEPHROTOMOGRAPHY*

A POTENTIAL SOURCE OF CONSIDERABLE DIAGNOSTIC INFORMATION

By PHILIP M. HATFIELD, M.D.,† *and* RICHARD C. PFISTER, M.D.
BOSTON, MASSACHUSETTS

IN 1970 we reported on the urographic evaluation of 60 patients with polycystic disease of the kidneys and liver.[4] In 1 of these 60 cases, liver cysts were manifested as lucent defects during the total body opacification stage of infusion nephrotomography.

Since that time, we have encountered an additional 3 instances in which extrarenal cysts were clearly delineated by high dose infusion urography. This report summarizes these 4 cases, and the techniques utilized in demonstrating extrarenal cysts during infusion nephrotomography.

REPORT OF CASES

CASE I. This 59 year old diabetic male with a paroxysmal sinus arrhythmia and a permanent demand pacemaker was admitted with recurrent syncopal episodes. The admission chest roentgenogram revealed an unsuspected lung mass in the right upper lobe, and the preoperative intravenous urogram an unsuspected lucent left upper quadrant mass, "probably of renal origin" (Fig. 1). A thick walled cystic splenic mass was subsequently demonstrated by infusion nephrotomography (Fig. 2) and later confirmed by celiac arteriography (Fig. 3, *A* and *B*). An inoperable poorly differentiated squamous cell carcinoma of the lung was found at thoracotomy. The patient received palliative irradiation postoperatively. He remains alive with multiple skeletal metastases 1 year after exploration.

CASE II. This 57 year old male with known polycystic kidneys was admitted with an acute myocardial infarction. The pertinent physical findings included large irregular palpable kidneys and a liver edge that could not be felt, but percussed 10 cm. below the costal margin. Blood urea nitrogen (BUN) 117 mg. per cent, creatinine 4.3 mg. per cent, uric acid 11.8 mg. per cent, calcium 8.7 mg. per cent, phosphorus 4.6 mg. per cent, total bilirubin 0.2 mg. per cent, alkaline phosphatase 1.7 units, SGOT 80, prothrombin time 12.9 min. (control 12.5 min.), albumin/globulin 3.6/2.2 gm. per cent, and cephalin flocculation 1+ at 24 hours, 2+ at 48 hours. Infusion nephrotomograms demonstrated bilateral 20×12 cm. polycystic kidneys and multiple intrahepatic lucencies characteristic of polycystic disease of the liver (Fig. 4). The patient recovered from his myocardial infarction, but expired 18 months later from a cardiac arrhythmia. Postmortem examination was not performed.

CASE III. This 63 year old female with known polycystic disease of the kidneys and liver was admitted with progressive renal failure, severe nocturnal muscle cramps, anemia, and exhaustion. Both kidneys were enlarged and nodular to palpation. The liver was thought to be enlarged clinically, although the liver edge could not be felt. BUN 74 mg. per cent, creatinine 8.4 mg. per cent, calcium 7.6 mg. per cent, phosphorus 5.8 mg. per cent, alkaline phosphatase 2.6 units, SGOT 11, and albumin/globulin 4.2/2.2 gm. per cent. Infusion nephrotomograms demonstrated bilateral polycystic kidneys, the right measuring 20×13 cm. and the left 17×11 cm. and multiple hepatic cysts (Fig. 5). The patient responded to a low protein diet and conservative measures, but died at home 8 months later of progressive renal failure. Postmortem examination was not performed.

CASE IV. This 69 year old female with known polycystic kidneys was admitted with paroxysmal atrial fibrillation and chronic renal failure.

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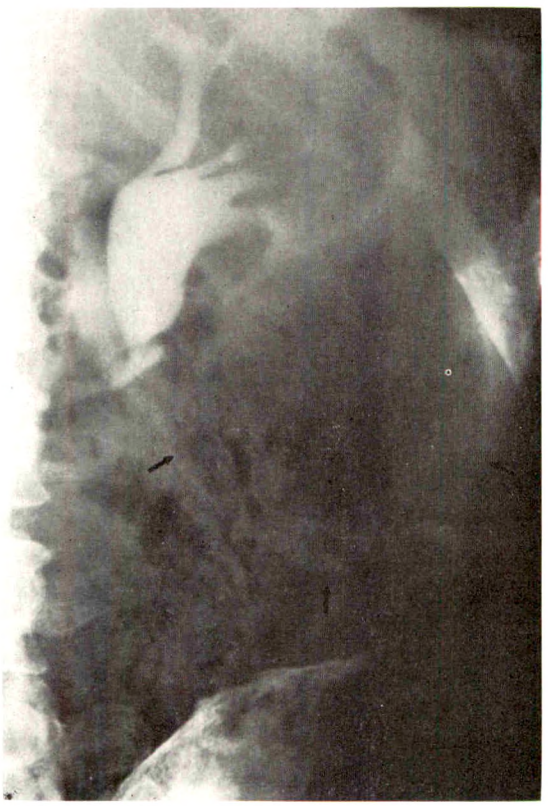


FIG. 1. Case 1. Intravenous pyelogram (IVP) shows lucent mass (arrows), displacing the collecting systems of the left kidney superiorly.

The kidneys were enlarged to palpation and the liver edge was felt 3 fingerbreadths below the costal margin. BUN 63 mg. per cent, creatinine 5.8 mg. per cent, uric acid 8.1 mg. per cent, total bilirubin 0.2 mg. per cent, alkaline phosphatase 17.6 units, SGOT 30, prothrombin time 13.5 min. (control 12.5 min.), and albumin/globulin 4.3/3.4 gm. per cent. Infusion nephrotomograms demonstrated enlarged (20 cm.) polycystic kidneys and multiple liver cysts (Fig. 6), confirmed by technetium 99m sulfur colloid liver scan (Fig. 7). The patient remained stable at her last clinic visit, 5 months after discharge.

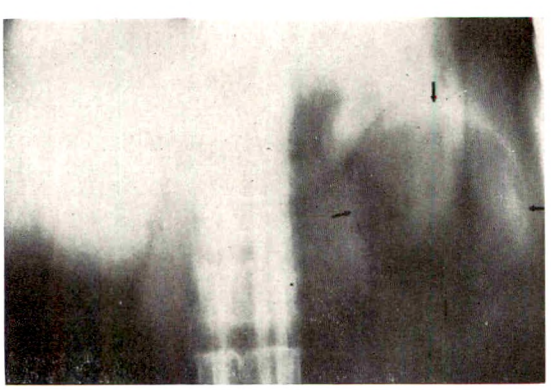


FIG. 2. Case 1. Infusion nephrotomogram shows extrarenal mass (arrows), displacing the left collecting system superiorly.

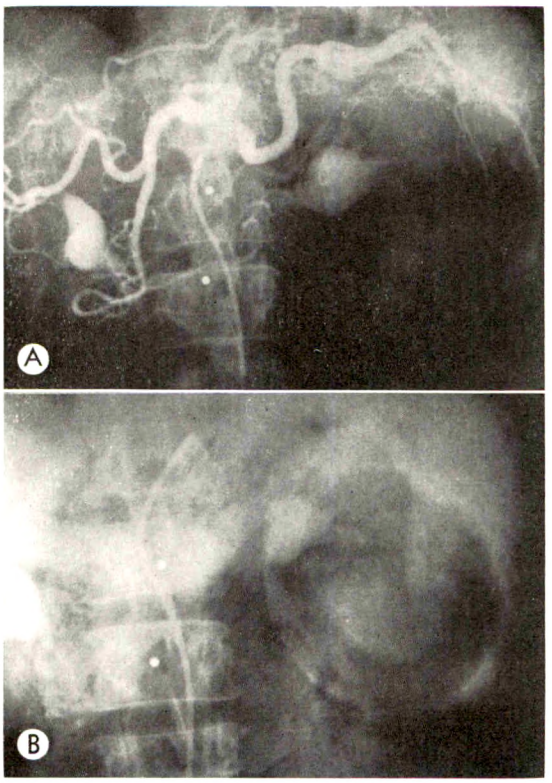


FIG. 3. Case 1. Celiac arteriograms. (*A*) Arterial phase. Superior displacement of the splenic artery by a large lucent mass arising from the inferior portion of the spleen. (*B*) Capillary phase. Thick walled lucent mass arising from the inferior portion of the spleen.

DISCUSSION

Since Evans' introduction of nephrotomography in 1954[2] and the modification by Schencker *et al.*,[10] utilizing the drip infusion technique, high dose infusion nephrotomography has become a widely accepted technique for localizing and differentiating intrarenal masses. O'Connor and Neuhauser[8] first utilized total body opacification to distinguish renal from extrarenal masses in infants. They re-

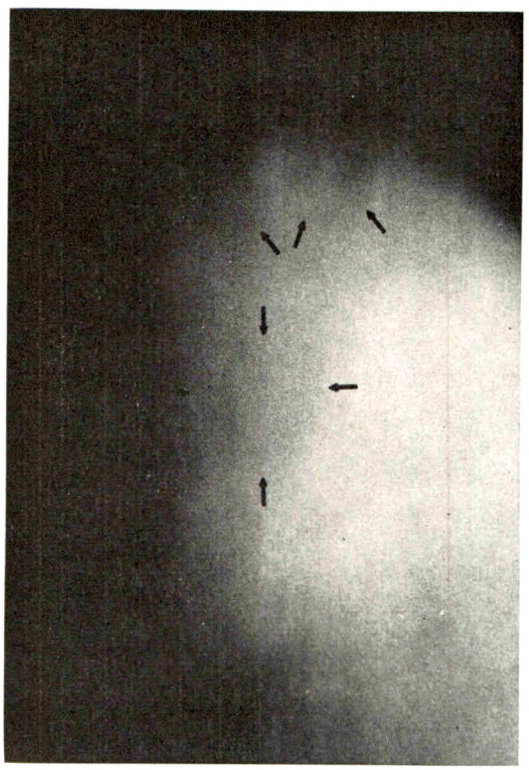


Fig. 4. Case II. Anteroposterior infusion tomogram of the liver demonstrating multiple lucent liver cysts (arrows) during the total body opacification stage.

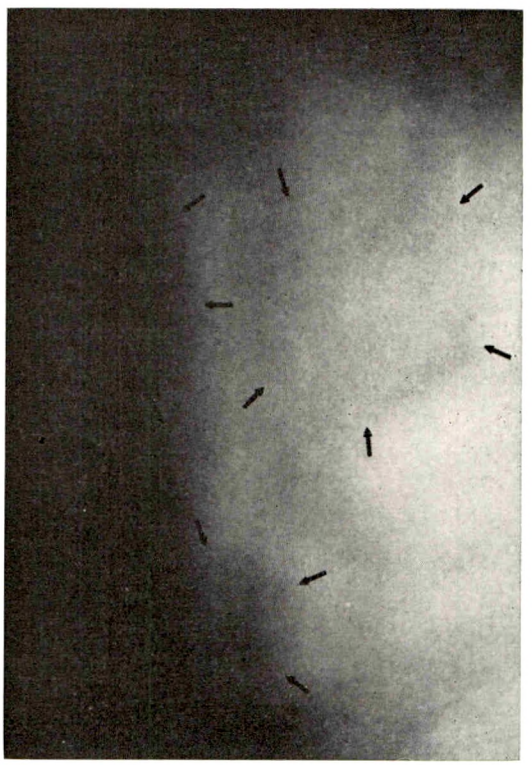


Fig. 5. Case III. Anteroposterior infusion tomogram of the liver demonstrating multiple lucent liver cysts (arrows) during the total body opacification stage.

ported 2 extrarenal masses that were clearly defined by high dose urography—a cavernous hemangioma of the liver and a large cystic intestinal duplication. In a subsequent report from Boston Children's Hospital, Griscom[3] emphasized that total body opacification is invaluable in differentiating abdominal masses in infants, and suggested that the diagnosis of ovarian, hepatic, mesenteric, and choledochal cysts greater than 5 cm. in diameter can often be established by high dose urography. However, he noted that "the phenomenon (total body opacification) is more useful in infants and young children than in adults, because scatter from large volumes of tissue will often obliterate the relatively slight differences in opacity, because children's lesions are relatively larger, and because relatively larger volumes of contrast material can be used routinely."

Although the method has not been routinely applied in adults, our experience suggests that it is applicable, and often yields useful diagnostic information, given meticulous attention to technical detail

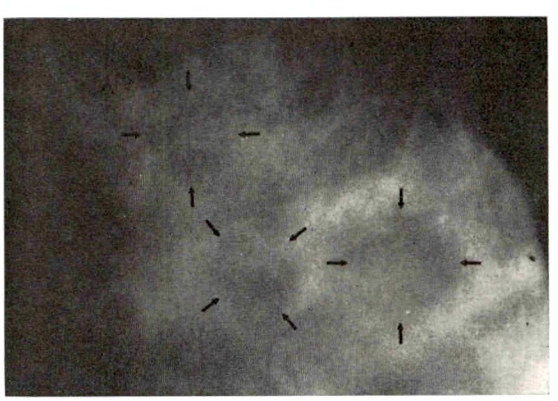


Fig. 6. Case IV. Right posterior oblique projection of the liver, demonstrating multiple lucent cysts (arrows) during the total body opacification stage.

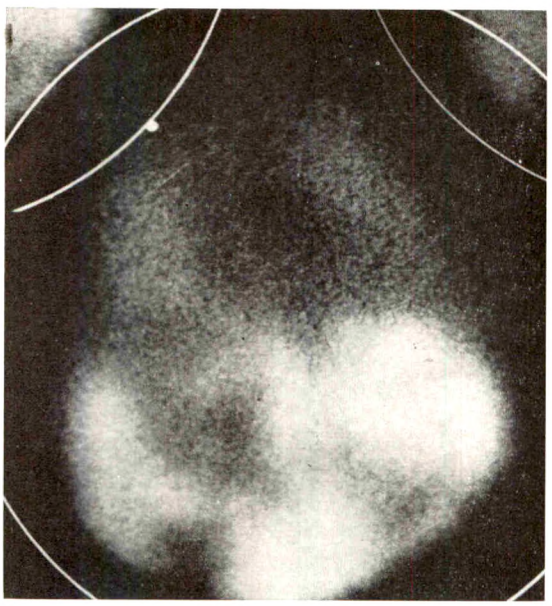


FIG. 7. Case IV. Tc^{99m} sulfur colloid liver scan, anterior view, demonstrating multiple hepatic lucencies characteristic of polycystic disease of the liver.

and a careful search for extrarenal lucencies during each and every infusion urogram.[9]

To date, only 7 extrarenal masses, detected during infusion urography, have been reported in adults. A splenic cyst was visualized by infusion urography in 1966,[5] and a malignant retroperitoneal mesenchymoma in 1968.[11] In a recent review of cystic disease of the kidneys, Elkin and Bernstein[1] published a roentgenogram obtained during the total body opacification stage, demonstrating multiple liver cysts in a patient with polycystic disease of the kidneys and liver.

However, the potential of the method has only recently been recognized. Mitty and Shapira[7] have succeeded in demonstrating extrarenal masses in adults on 4 occasions by infusion urography—a periadrenal lymphatic cyst, a liver cyst, a liver abscess, and a hypovascular splenic metastasis.

The method is straightforward and requires little modification of the standard infusion nephrotomographic techniques.[9] Three hundred ml. of contrast medium (Hypaque 25, Reno-M-DIP) are infused through a No. 17 needle in an antecubital vein. It should take no more than 5 minutes to infuse the entire 300 ml. If the prepackaged tubing provided with the contrast medium will not permit this rapid an infusion, standard blood infusion tubing should be substituted. Anteroposterior tomograms of the abdomen are obtained (10×12 inch films), including the liver and spleen as well as the kidneys. Scout sections determine the appropriate tomographic levels. Usually 5 or 6 sections, between 5 and 12 cm. from the tabletop, suffice. Tomograms are obtained halfway through the infusion when total body opacification becomes intense. If extrarenal pathology is suspected, but not clearly defined, tomograms may be repeated at the end of the infusion, as may appropriate oblique or lateral views. Tomograms of the spleen in the left lateral projection may be particularly helpful in demonstrating splenic cysts.[5] Zonography is the method of choice in nephrotomography, although linear tomography suffices if zonography is not available.[6]

In conclusion, it is clear that both hepatic and splenic cysts in adults may be visualized during the total body opacification stage of infusion urography. The lucencies produced by avascular splenic and hepatic lesions may be subtle. However, meticulous technique and multiple views in the oblique and/or lateral projection(s), will clearly demonstrate extrarenal cystic lesions, and extend the diagnostic yield of the nephrotomographic examination. In so far as the method is safe, and effective, it merits, and no doubt will gain, wide acceptance in the diagnosis of cystic lesions of the liver and spleen.

SUMMARY

The diagnosis of avascular cystic lesions of the liver and spleen in adults may be established by high dose infusion nephrotomography.

Three instances in which multiple cysts

of the liver were demonstrated by infusion nephrotomography, and 1 in which an avascular splenic lesion was clearly defined, and the techniques for visualizing such lesions are discussed.

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RATIONALE AND USE OF VASODILATED EXCRETORY UROGRAPHY IN SCREENING FOR RENOVASCULAR HYPERTENSION*

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OVER the last few years there has been a dramatic increase in medical concern about the diagnosis and treatment of hypertension. The Veterans Administration Cooperative Studies[31,32] have convincingly demonstrated the potential benefits to be realized by effective antihypertensive treatments. However, clinical investigations continue to reveal subgroups of hypertensives for whom unique therapies may be indicated; *i.e.*, propanolol for high renin essential hypertension,[2] spironolactone for low renin essential hypertension,[4] and surgery for renovascular hypertension.[19] Sophisticated diagnostic tests are frequently necessary to cull these subgroups from among the millions of known hypertensives. There may be as many as 5 million hypertensives in the United States alone who have surgically curable secondary hypertension.

Were dollars and diagnostic resources unlimited, each hypertensive might reasonably be given an extensive diagnostic evaluation. This is an unattainable objective in that the number of hypertensives may approach 20–30 million Americans. Screening tests must of necessity be employed. Since the evidence suggests it is the lowering of blood pressure that is beneficial, why not use medical therapy for screening and work-up the medical failures? Due to the large numbers of patients involved, this approach has much to recommend it. However, life-long drug treatment of an essentially asymptomatic disease is quite difficult to achieve and not inexpensive. To the extent that medical therapy is ineffective, patients with surgically curable hypertension are subject to needless risk.

In his role as a diagnostic consultant, the radiologist most frequently faces this uncertain situation in the form of a request for a "hypertensive IVP." It is safe to assume that the referring physician wishes to know if the patient has renal hypertension. There are many forms of renal disease responsible for hypertension.[9] The anatomic information available from an excretory urogram frequently provides important information in the differential diagnosis. However, an examination with even minor risk should not be performed unless the result could influence therapy for the patient. The ideal screening test for renovascular hypertension must identify patients who do not have surgically correctable renovascular hypertension as well as those who may.

We have previously reported, in preliminary form,[36] that Vasodilated Excretory Urography (VEU) provided a more accurate reflection of the true renal hemodynamic state than the hypertensive intravenous pyelogram (IVP). The purpose of this report is to extend those observations and to discuss the renal physiology which makes VEU the best screening test presently available.

INFORMATION AVAILABLE FROM EXCRETORY UROGRAPHY

The presence of a significant renal hemodynamic defect is suspected during excretory urography from either a difference in

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† Assistant Professor of Pharmacology and Radiology.

renal size or a disparity in contrast medium excretion. Renal length is usually measured on the preliminary film (the least discriminant in the entire study), but may be measured at any time. On the other hand, contrast medium excretion is considered to be a variable, where timing is crucial.[21] Actually both renal size and contrast medium excretion are physiologic variables. Before diagnostic significance can be accorded either, one should understand the physiologic basis for the "standard ideas" of delayed appearance, delayed hyperconcentration, spidering, etc.

Disparity in pelvocalyceal opacification following bolus injections of contrast media and positive split function tests are assumed to result from excessive reabsorption of water in the affected kidney.[10,21,27] Since diatrizoate and iothalamate salts are, for the purposes of rapid-sequence urography, distributed and excreted like inulin,[24] it is tempting to believe that contrast media excretions may provide the same information as inulin concentration in split function testing.

There are several reasons for suggesting that the physiologic processes at work during bolus excretory urography may be more complex. First, split function tests are performed under clearance conditions, and renal physiologists have vigorous objections to interpreting any renal clearances obtained under rapidly changing circumstances.[34] Most pertinent to the conditions of excretory urography is the normal heterogeneity of nephron lengths. Short nephrons will excrete substances such as inulin and contrast material more quickly than long nephrons. The magnitude of this phenomenon, known as nephron delay time, can be appreciated following bolus injection of inulin in normal man. Only 1 per cent of the resulting immediate glomerular filtrate reaches the bladder in 1 minute. Longer nephrons are slower, so that 21 per cent of the initial filtrate requires 4 minutes to reach the bladder.[5] For contrast media, the phenomenon is clearly revealed in that peak excreted contrast concentrations are reached at 15–30 minutes, whereas the peak filtered concentrations are achieved within the first circulation time.[8,17] Thus the earliest opacification of the pelvocalyceal system is accomplished by only a fraction of the nephrons of that kidney. The only effective compensation for nephron delay is constant plasma concentration so that each filtered volume starts with the same composition. The influence of hemodynamic factors in nephron delay time is unknown.

A second important difference exists between measured inulin concentrations and subjective estimates of pelvocalyceal opacification. Significant differences in inulin concentration range from 100–500 per cent.[27] Density of pelvocalyceal opacification depends upon the amount (volume times concentration), rather than concentration alone.[8] In addition, 2–4 fold differences in urine concentrations of contrast material may be impossible to distinguish at urography.[22]

Thirdly, mannitol, an osmotic diuretic, tends to eliminate the differences in inulin concentrations in positive split function tests.[27] The contrast agents are very similar to mannitol in their hemodynamic and diuretic effects upon the kidney.

The physiologic conditions are considerably more stable in the later films of the study when plasma concentrations of contrast media are falling less rapidly and the stressful stimuli attending the bolus injection have waned. One might expect delayed hyperconcentration to reflect the excessive tubular reabsorption caused by a significant stenosis. In fact, delayed hyperconcentration is considerably less discriminant than delayed appearance time.[1,20]

In view of these considerations, one wonders how a disparity in contrast medium excretion can ever accurately reflect the hemodynamic lesion. Yet, contrast disparities do indicate the presence or absence of significant stenosis with an accuracy well beyond chance expectations. It seems possible that the vasodilator properties of intravenous contrast agents enhance the

excretion rate from the kidney with normal vasodilator reserve and are not as effectively diuretic in the presence of significant stenosis. Earley and Friedler[11] have shown that vasodilator diuresis due to acetylcholine can be significantly diminished by aortic coarctation.

MATERIAL AND METHOD

This report is based on 250 VEUs performed on hypertensive patients referred to the Radiology Department for a "hypertensive IVP." They were routinely prepared for urography by overnight dehydration, cathartics, and clear liquid diet. Following the preliminary roentgenogram, 50 ml. of conray 400* was injected into the antecubital vein within 20–30 seconds. Subsequent roentgenograms were taken at 1, 2, 3, 5, 8, and 15 minutes. Following the 3 minute film, 50 mg. of ethacrynic acid (edecrin, Merck Sharp and Dohme) was injected intravenously over a period of 1–2 minutes. About one-third of the patients noted pain at the injection site, probably due to the low pH of the diuretic solution. The painful response lasted less than 2 minutes. Because of the rapid diuresis after ethacrynic acid (ECA), it was found necessary to infuse a solution of 50 ml. conray 400 in 200 ml. of saline by intravenous drip to maintain renal silhouette visibility. Renal silhouettes, using the medial tangent method,[14] were outlined with a wax pencil directly on the resulting roentgenograms and their areas determined with a compensating polar planimeter.

RESULTS

1. MEASUREMENTS OF RENAL SIZE

The first prerequisite for performing an experiment is to determine if the parameters of interest can be reproducibly measured. Many radiologists are dubious that renal size changes of the order reported here can be accurately detected. On the contrary, when the renal silhouettes are visible, very accurate measurements can be made. Thus, Klatte *et al.*[20] estimated an error in length of 1.2 per cent and width 2.4 per cent on independent measurements by 3 observers. Vuorinen and Wegelius[38] achieved a highly significant difference in renal length times width before and after mild osmotic diuresis (glucose) when the average difference was only 2.4 cm.^{2}, or about 3 per cent.

Actual measurement of silhouette area is inherently more accurate than length times width because of the marked increase in the number of decision points. The correlation between silhouette area, length, width, and length times width for 40 roentgenograms is shown in Table 1. Of course, no method can accurately measure poorly defined silhouettes. The earliest film following contrast medium injection had to be used for minimum size in 41 kidneys in the 250 studies because preliminary renal shadows could not be identified with confidence, even in retrospect.

2. THE NORMAL RESPONSE

Figure 1 reflects the time course of a typical VEU response in a hypertensive patient with a normal arteriogram and no lateralization of renins. The transient decrease in renal size with the painful response to ECA is seen at 5 minutes. The normalized response for 200 patients with the presumptive diagnosis of essential hypertension is depicted in Figure 2. The 2, 5 (no pain), and 15 minute areas are all different from the preliminary area ($p < .001$).

Any agent that effectively decreases renal resistance without causing systemic pressure (the distending force) to fall

TABLE I

CORRELATION OF SILHOUETTE AREA WITH LINEAR MEASUREMENTS OF RENAL SIZE

	Correlation Coefficient		
	Length	Width	Length×Width
Area	.65	.51	.83

* Sodium iothalamate, Mallinckrodt Pharmaceuticals, St. Louis, Missouri.

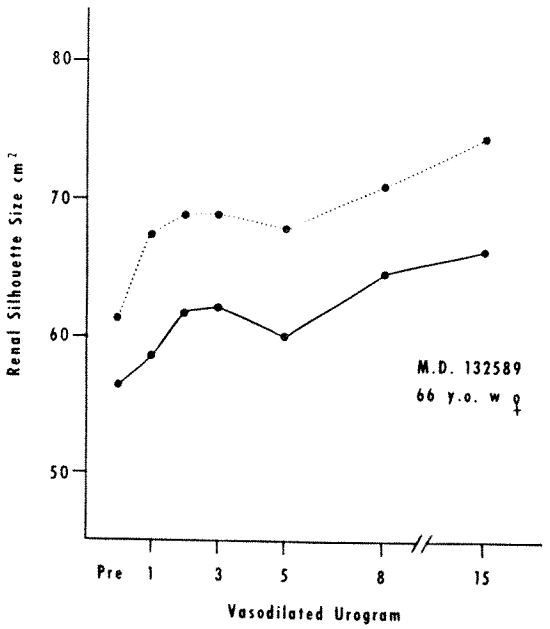


FIG. 1. Typical size changes during the vasodilated excretory urography (VEU) protocol in a hypertensive patient with no arterial stenosis. Right renal response is represented by the solid line, left renal response by the dotted line.

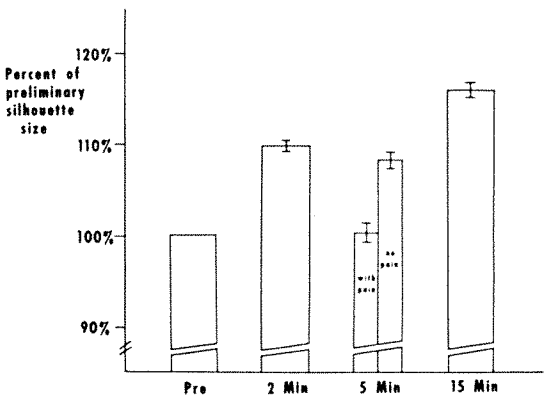


FIG. 2. Average response in 200 hypertensive patients with essential hypertension. The preliminary renal size is normalized to equal 100 per cent. Each bar represents the mean per cent of preliminary size ± 2 S.E.M.

would cause renal size to increase. The response to ECA is not specific since the average size increase with contrast agents is highly significant. However, our experience with the early renal swelling associated with the contrast bolus shows it to be diagnostically less reliable than the more potent and longer lasting ECA response. Nausea, pain, cardiovascular changes, and psychic stress appear to cause the unchanged or even decreased renal sizes seen in 10–15 per cent of the early films.

In the absence of these stimuli, renal size increases an average of about 10 per cent the first 2 minutes. When delineation of renal shadows is inadequate on the preliminary film, the earliest possible adequate nephrogram should be used instead. For the reasons above, one must accept the possibility of a spurious baseline reading, but, on the *average*, the following corrections can be used: 30 seconds—2 per cent; 1 minute—5 per cent. Not infrequently, maximal vasodilatation is realized with contrast media and the 15 minute silhouettes are not much larger than at 2 minutes.

We consider that a size increase (maximum/preliminary × 100) greater than 10 per cent reflects adequate vasodilator reserve and, therefore, normal renal hemodynamics. The validity of this result is shown in Tables II and III. A positive arteriogram is defined as the presence of an anatomic stenosis measuring 30 per cent or greater. A significant stenosis would require lateralization of renins (ratio of 1.4 or

TABLE II

PER CENT AREA INCREASE VERSUS LENGTH AND CONTRAST DISPARITY FOR 250 EXAMINATIONS*

Area Increase (individual kidneys)	Length Difference (examinations)	Contrast Disparity (examinations)	Positive Arteriogram (individual kidneys)	Renin Lateralization (examinations)
1. Greater than 10 per cent n = 415/489	12/250	8/250	4/72	0/42
2. Less than 5 per cent n = 37/489	14/33	10/33	18/22	16/18

* Size increases not measurable for 11 kidneys; 37 kidneys increased 5–10 per cent. See text for further detail.

TABLE III

ACCURACY OF DIAGNOSTIC PREDICTION OF SIGNIFICANT STENOSIS BASED ON PER CENT SIZE INCREASE

	True	False Positive	False Negative
1. Normal VEU	72/72	—	0
2. Suspicious VEU	16/18	2/18	—

greater) in addition to the anatomic stenosis. Minor urographic signs were not included in this assessment. As evident in Table II, 4 kidneys had a normal size increase in the presence of stenosis that did not result in lateralization of renins.

In this series, size increases of 5–10 per cent were measured in 37 kidneys. For 32 of these, renal size was not assessable on the preliminary film. The uncertainties of preliminary size make these responses nondiagnostic. For the remaining 5 kidneys, 3 had roentgenographic evidence of pyelonephritis and 1 patient had biopsy-proven diabetic glomerulosclerosis.

3. PATIENTS WITH SIGNIFICANT STENOSIS

Table II shows that 37 kidneys failed to increase by 5 per cent. Arteriography and selective renins were obtained in 20 of the original 33 patients. Accuracy assessments are based on these 20 patients. Length differences were measured on the preliminary film. The methods included a shortened rapid-sequence protocol, and contrast disparities were assessed only on the films prior to ECA since the influence of this loop diuretic on contrast medium washout in the presence of significant stenosis is unknown. Thus the accuracy comparison of VEU *versus* rapid-sequence is not completely fair to the proponents of the latter.

The typical responses seen for patients with unilateral stenosis or bilateral stenosis are demonstrated in Figures 3 and 4, respectively. None of the 4 patients with bilateral blunted responses were detectable by urographic criteria. Only 2 patients were studied by arteriography and their renins did not lateralize.

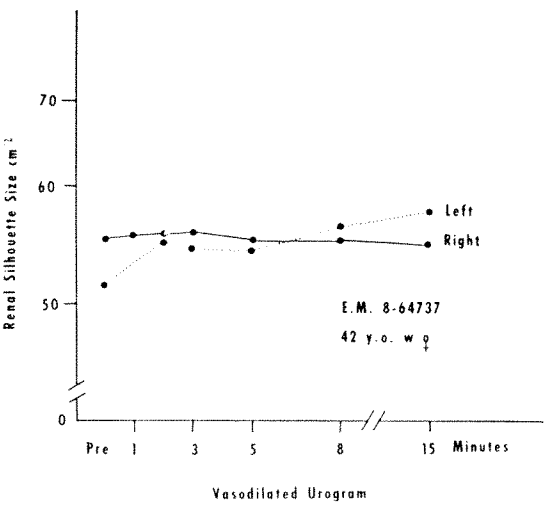


FIG. 3. The VEU response in a case with *left* fibromuscular dysplasia. Previous work-up includes 3 normal IVPs and 3 normal split function tests. She is markedly improved 8 months after renal by-pass grafting.

It is difficult to know what criteria to use as a standard of comparison to determine the "accuracy" of the diagnosis short of surgical cure. All 4 patients with unilateral stenosis treated surgically have had an excellent response, although the average follow-up is less than a year. The agreement of VEU with arteriography and selective renal vein renin ratios for unilateral stenosis only is as follows:

a. In 14 patients all 3 diagnostic modalities indicated the presence of a significant stenosis.
b. Twice, the abnormal VEU was associ-

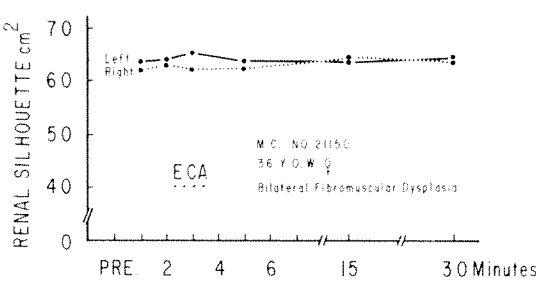


FIG. 4. The VEU response seen with bilateral significant stenosis. The patient had had 2 negative IVPs before this study led to confirmatory arteriography.

ated with normal renin and arteriographic studies.

c. On 2 occasions, the arteriogram was normal but renins lateralized to the side predicted by the VEU.

For the latter, small vessel disease would explain the apparently normal arteriogram. Including these 2 studies as true positives in Table III is debatable, since biopsy confirmation has not been possible. For all patients, the agreement of VEU predictions with other tests performed in the same patient is depicted in Figure 5.

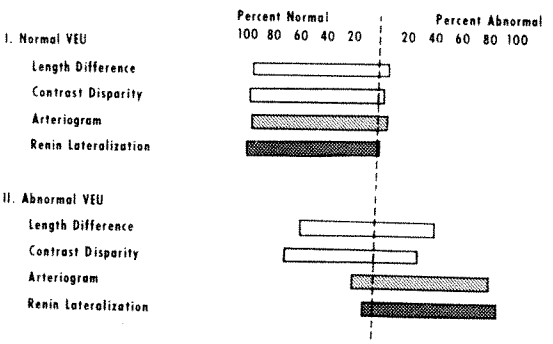


FIG. 5. Concordance of diagnostic tests with normal and abnormal VEU. This is a graphical summary of the data from Table II. Note that the predictions from dynamic changes in renal size are in much better agreement with the abnormal arteriograms and selective renal vein renins than are length difference or contrast disparity.

4. THE MECHANISM OF THE RESPONSE

In our preliminary report, we speculated that local regulatory mechanisms might decrease renal resistance as a compensatory response to renal arterial stenosis. The concept that a vasodilator reserve characteristic for each organ determines the degree of stenosis that is significant for the organ has been considered elsewhere.[35] Since vasodilator reserve is minimal for the kidney, stenoses near 50 per cent suffice to reduce resting renal blood flow, whereas 95 per cent stenosis may be required for skeletal muscle arteries.

In a series of dog experiments, we have examined the correlation of several renal parameters with renal size, either with or without induced renal arterial stenosis. These results can be summarized as follows:

a. The intravenous administration of clinical doses of contrast media causes a decrease in measured renal resistance of approximately 25 per cent in nembutal anesthetized dogs. The characteristic renal vasoconstriction seen with arteriographic doses is not demonstrated.[3] Infusions of contrast material into the renal artery cause renal vasodilatation throughout infusions lasting as long as 15 minutes.

b. Clinical doses of contrast media and ethacrynic acid increase renal size, subcapsular pressure[15] and urine volume, without significantly affecting glomerular filtration rate.

c. Increasing vascular stenosis progressively decreases vasodilator response by the kidney. As dilator reserve is lost, the usual increase in renal size is proportionately attenuated, the subcapsular pressure rise with contrast media and ethacrynic acid is markedly diminished, and diuretic response to either agent is blunted.

DISCUSSION

The results of our clinical and laboratory work support the investigations of Swann who first commented upon the "erectile" nature of the kidney.[6,29,30] The kidney is composed of 3 very distensible compartments: vascular, tubular, and interstitial. The relative pressure within each compartment strongly determines its volume. The distending pressure comes from the systemic circulation, while the several resistances in the kidney determine the extent to which any given distending pressure is transmitted. These resistances are subject to nervous, humoral, drug, and local regulatory influences and thus renal size is a dynamic variable.

The observations that renal size reflects the state of renal vessels can be traced to Starling.[28] Any stimulus that causes hypotension or a strong sympathetic discharge raising renal resistance can be expected to

cause rapid decreases that can be as large as 40–50 per cent.[6,18,26] The rapid decrease in renal size in response to pain reflects the characteristic increase in renal resistance seen in the so-called "defense reaction."[12]

Increases in renal size have generally been recorded with agents that cause renal vasodilatation and/or diuresis.[25,26,33,36,37] Vuorinen and Wegelius[33] appear to have first suggested that drugs might induce changes in renal size. Whether local vasodilatation associated with inflammatory processes or transplant rejection explains the transient increases in renal size seen with the oliguria of the glomerulonephrides, acute tubular necrosis, or transplant rejections[34] remains to be seen.

Our clinical application of induced changes in renal size is critically dependent upon those changes being large enough for accurate detection and their dependence upon hemodynamic events that occur principally on the arterial side. Actual measurement of renal volume[16] may be more sensitive than the silhouette area described herein. However, for screening purposes, satisfactory accuracy seems possible from single plane estimates of renal size.

The concepts of vasodilator reserve deserve more consideration in evaluating the significance of arterial pathology, since a functional rather than anatomic interpretation is obtained. By applying these precepts, the significant anatomic stenosis for the kidney is approximately 50 per cent. It is also at this level that the rapid-sequence IVP tends to become more frequently positive.[1]

The use of VEU has both theoretic and practical advantages over rapid-sequence studies as the screening test for hypertensives. The theoretic advantages of the dynamic test include: (1) the ability to detect bilateral circulatory defects; (2) only lesions significant enough to cause almost total loss of vasodilator reserve are detected, whether large or small vessel disease is responsible; and (3) some idea of vasodilator reserve in the contralateral kidney is obtained.

Many authors have noted that surgical results can be strongly dependent on the circulatory state of the "normal" kidney. In regard to the latter, we are intrigued by the observation that patients in whom contralateral renal vasodilatation occurred during trimethaphan hypotension were surgically curable by nephrectomy, whereas no patient who failed to show vasodilatation was benefited by nephrectomy.[13] The analogy to dynamic testing of vasodilator reserve is obvious.

The practical advantages include the superior accuracy and considerable economy. Fewer expensive hospital work-ups are initiated for false positive studies. The test itself results in a halving of roentgenograms taken and a similar reduction of technician time.

The following protocol is recommended for routine use:

1. Bowel cleansing with cathartics and clear liquid diet.
2. Nothing by mouth for 8–12 hours.
3. Obtain preliminary roentgenogram, determine if the silhouettes of both kidneys are visible
   a. if not, plan to obtain a coned roentgenogram of kidneys 30 seconds after contrast medium bolus injection.
4. Inject intravenous contrast medium bolus in the usual amounts.
5. Obtain 5 and 10 minute full field roentgenograms without compression to define the anatomy of the kidneys, pelves, ureters, and bladder.†
6. After satisfactory delineation of urographic aspects of the study, inject 50 mg. ECA (or 40 mg. furosemide, Lasix) intravenously along with a dilute contrast medium infusion (approximately 5 mg./ml. iothalamate) to maintain the nephrogram.

† Others may prefer their own routine procedure for demonstrating urinary tract morphology. Flexibility is desirable and possible with one constraint. The combination of contrast agent and ethacrynic diuresis makes it increasingly difficult to maintain a satisfactory nephrogram if step 6 is delayed much beyond 10 minutes.

7. Obtain coned film of kidneys 10 minutes after vasodilator injection.
8. Estimate observed renal silhouette increase by planimetry, or length times width, on films from Steps 3 and 7.

VEU is still recommended only as a screening test and must be followed by arteriography, selective renal vein renins, and/or split function testing prior to surgery. Our experience with the normal VEU response in patients with incidental stenosis is too sparse to be definitive. Interestingly enough, Dorph and Oigaard[7] have recently confirmed both the increased accuracy of dynamic size changes over contrast disparity and the normal size increase with insignificant stenosis on a retrospective analysis of several hundred urea-washout studies. Since hypertonic urea is both a good renal vasodilator and a fair diuretic, the search for better screening tests has come full circle. Hopefully, the physiologic bases of such tests are better understood from more recent research.

SUMMARY

The use of dynamic increases in renal size as determined by the technique of vasodilated excretory urography (VEU) is compared with modified rapid-sequence pyelography in a series of 250 hypertensives.

The physiologic constraints of contrast disparities in screening for significant renal arterial stenosis are discussed.

Normal and abnormal responses to VEU are defined as well as laboratory evidence that renal size and renal hemodynamic states are closely linked. Thus, changes in renal size elicited by vasodilators can be used to assess renal vasodilator reserve and, thereby, the presence or absence of significant stenosis. Size changes are shown to be more accurate than contrast or length disparities.

None of the kidneys that increased by 10 per cent or more had significant stenosis; all of the kidneys with significant stenosis increased by 5 per cent or less.

The use of VEU has many advantages in screening hypertensives for renovascular hypertension that is potentially curable.

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DIRECT MAGNIFICATION INTRAVENOUS PYELOGRAPHY IN THE EVALUATION OF MEDULLARY SPONGE KIDNEY*

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MEDULLARY sponge kidney is a well known pathologic entity today with an over-all incidence of about 0.5 per cent identifiable on excretory urography. It is important to differentiate the lesion from more serious entities such as renal papillary necrosis, renal cystic disease with congenital hepatic fibrosis, medullary cystic disease, and diseases causing nephrocalcinosis. In addition, the entity of medullary sponge kidney itself is of clinical significance in that patients may be stone formers or show evidence of hematuria. The identification of patients with medullary sponge kidney, therefore, is of more than academic interest. In 1963 Palubinskas[14] demonstrated that the range of dilated tubules extended from obvious roentgenographically identifiable ectatic tubules to those which could not be differentiated from a so-called pyramidal blush, which may be related to the increased concentration of contrast material in the renal pyramids, particularly with higher doses of newer excretory urographic agents. He showed that considerable uncertainty arose among many radiologists in the identification of those cases having a minimal degree of ectasia. In individual patients there was, however, a range of ectasia from roentgenographically obvious medullary sponge kidney to an appearance indistinguishable from a pyramidal blush from calyx to calyx within the same kidney. Pathologic confirmation demonstrated that at least some patients with a so-called pyramidal blush do have a more benign form of renal tubular ectasia.

During the past 2 years at Misericordia and Fordham Hospitals, we have attempted to utilize direct magnification roentgenography and other techniques to extend the range of roentgenographically identifiable tubular dilatation in frank cases of medullary sponge kidney, and in those cases exhibiting a so-called pyramidal blush.

METHOD

After identification of a case of suspected medullary sponge kidney on initial excretory urography, a repeat study was performed on another day utilizing direct magnification roentgenography. The initial magnification technique consisted of 1.8:1 direct magnification roentgenography utilizing a 0.3 mm. focal spot high speed roentgenographic tube, 3 phase generator, 100 ma. and approximately 80 kv., with an air gap for reduction of secondary radiation. Standard cassettes containing DuPont Hi Speed screens, and DuPont Hi Plus screens, and vacuum enforced rapid film changer cassettes containing DuPont Hi Speed and Radelin STF screens were used for recording. Initially it was thought that infusion intravenous pyelography, or high dose urography with 100 cc. of 76 per cent methylglucamine diatrizoate, would produce better visualization of the ectatic tubules. This proved disappointing, since it resulted in greater renal pyramidal and parenchymal opacification thereby decreasing the contrast between ectatic tubules and the renal parenchymal background in known cases. Ultimately, our standard dose of 50 cc. of 60 per cent methylglucamine diatrizoate, or 50 cc. of 50 per cent sodium diatrizoate became the dose employed, and proved satisfactory. The results were still not optimal because the contrast between small dilated tubules and the remainder of the renal pyramids is not great, particularly in heavy patients.

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Since direct magnification roentgenography at a 1.8:1 magnification factor (considered optimal for a 0.3 mm. focal spot), and 100 cm. target film distance does not result in absorption of all scattered radiation utilizing the air gap technique, in later cases an 8:1 or 6:1, 103 line grid was added.

The final technique consisted of DuPont Hi Plus screens and a 6:1 grid for films collimated to the size of each kidney, followed by coned postage stamp size fields of individual pyramids utilizing air gap technique only.

Several patients were also tomographed to determine whether tomography of the renal pyramids added any additional information.

At one institution, Misericordia Hospital, High Speed screens are utilized, and at the other, Fordham Hospital, Par Speed screens are utilized for ordinary intravenous urography. A comparison of identifiable ectatic tubules on studies made with each of these techniques was evaluated in comparison to the tomographic studies and magnification technique.

RESULTS

Individually dilated tubules in the renal pyramids were definitely more easily identifiable on intravenous urograms made with Par Speed screens than with High Speed screens. Linear tomograms of ordinary intravenous urography and infusion nephrotomograms with a precision tomographic unit did not result in identification of any greater number of individually dilated tubules than intravenous urograms obtained with Par Speed screens.

Results with direct magnification intravenous urography were initially disappointing because of the inadequate contrast due to the high dose urographic techniques first employed, and the lack of contrast inherent in short object-film air gap magnification roentgenography of an entire kidney. The reduction of the amount of contrast material to that used in ordinary intravenous urography and the addition of a grid to the direct magnification technique resulted in increased contrast in the renal pyramids, and identification of greater numbers of individually dilated tubules than could be identified initially.

Finally, the use of DuPont Hi Plus screens with films coned to the size of a postage stamp with air gap alone, resulted in the best definition and contrast. There was definite demonstration of individually dilated tubules in patients showing only a "pyramidal blush" in specific renal pyramids, and in several patients who had obvious roentgenographically identifiable medullary sponge kidney in other renal pyramids.

Several cases have also been encountered in which all renal pyramids showed only a "pyramidal blush" on conventional films, and magnification studies revealed individually dilated tubules.

DISCUSSION

The identification and differential diagnosis of medullary sponge kidney has prognostic and some therapeutic significance. It is possible to differentiate this entity from renal papillary necrosis, renal cystic disease with associated congenital hepatic fibrosis, and medullary cystic disease, by age, history, blood urea nitrogen level, intravenous urography, etc.

The so-called "benign renal tubular ectasia" may progress to the symptomatic form, and is difficult to distinguish from a normal pyramidal opacification (Fig. 1).

While our results initially were disappointing because of a number of incorrect assumptions as to the best method of increasing the contrast between the dilated tubules and the remainder of the renal pyramids, the more recent cases demonstrate that it is possible to identify additional renal pyramids containing dilated tubules by this method, and early cases of medullary sponge kidney which cannot otherwise be differentiated from a pyramidal blush.

Obviously, it will not be possible to differentiate all cases of "benign renal tubular

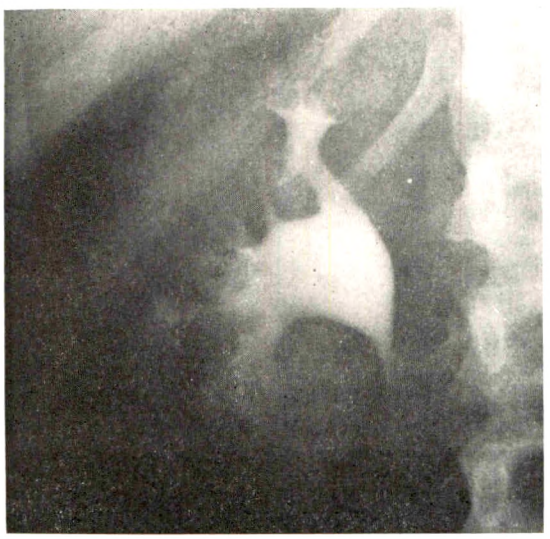


Fig. 1. Intravenous pyelogram shows a dense blush in the renal pyramids. No discrete, individually dilated tubules are visible.

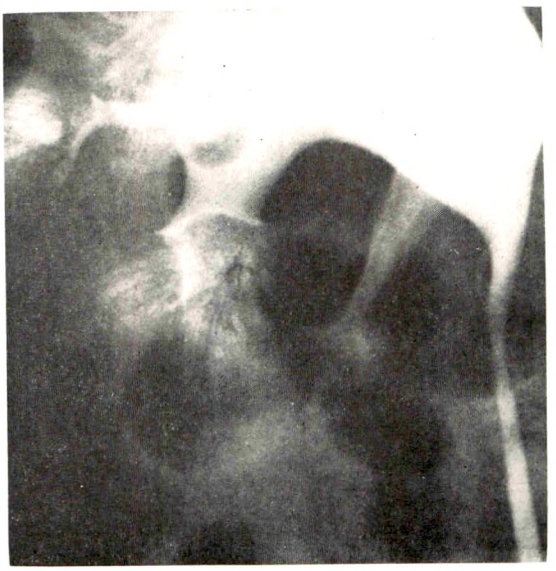


Fig. 2. A coned roentgenogram of the lower pole collecting system of the right kidney utilizing direct magnification shows the presence of discrete, individually dilated tubules characteristic of medullary sponge kidney.

ectasis" from "pyramidal blush" in the foreseeable future. However, 3 and 4 times magnification roentgenographic techniques for abdominal roentgenography are now in the experimental state. The key to further differentiation between the 2 above described entities will be a technical one: how to achieve maximum contrast at maximum magnification; *i.e.*, how to keep the effective focal spot size small, the tube loading not excessive, and the kilovoltage low enough and the field size small enough that air gap technique can be utilized as the sole means of reducing scattered radiation. The problem of spurious resolution due to interference from a fixed grid when dealing with structures as small as minimally dilated tubules mandates that the grid be removed, and air gap technique be employed at magnifications greater than 2:1. Cold cathode needle emission tubes may have some place in investigation of this problem, in that we are dealing with rapid serial roentgenography.

The optimal quantity of intravenous contrast media to differentiate between dilated and nondilated tubules also has not been investigated thoroughly.

Despite improvements in this technique (Fig. 2), it is doubtful that we will be able to demonstrate that all pyramidal blushes represent a benign form of renal tubular ectasia.

CONCLUSIONS

1. Individually dilated tubules in renal pyramids which show only a so-called pyramidal blush in patients with roentgenographically obvious medullary sponge kidney, can be demonstrated by direct magnification intravenous urography.

2. This technique also leads to positive identification of patients with medullary sponge kidney not identifiable by conventional means.

3. With the universally available 0.3 mm. focal spot magnification tubes, the best technique appears to be a combination of air gap and DuPont Hi Plus screens at a 1.8:1 magnification ratio, keeping the field size to 2.5×2.5 cm. at the skin entrance surface, with only a moderate amount of intravenous urographic contrast material introduced for optimal results.

4. Improvements in magnification technique in the near future will further dif-

ferentiate between benign forms of renal tubular ectasia and a so-called pyramidal blush if the latter is a true entity at all. It is doubtful, however, that this differentiation can be made in all cases despite foreseeable technical improvements.

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UROGRAPHIC ANALYSIS OF THE DEVELOPMENT OF POLYCYSTIC KIDNEY DISEASE*

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POLYCYSTIC kidney disease has been variously analyzed and characterized. The work of Osathanondh and Potter[13] has been central in the discussion of this entity since the publication of their paper based on microdissection. It is now commonly accepted that their Type 3 is the cystic disease of the kidney which is described as polycystic kidney disease. Cysts of the liver, and less commonly pancreatic, pulmonary and splenic cysts are associated. Hypertension and intracranial aneurysms are another aspect often leading to early death before an attained age of approximately 60.[1] Analysis of these kidneys has usually demonstrated absence of the papillae, abnormalities of the calyces, and dilatations of any of several portions of the nephron. This is distinguished from their Type 1 renal cystic disease with hyperplasia of the interstitial aspects of the kidney found in infantile polycystic disease.[13]

The disease process is apparently latent for many years and then becomes manifest in renal parenchyma which has developed and functioned normally. The cyst formation usually occurs synchronously and proceeds at approximately the same rate. The increase in renal size generally takes place before there is functional destruction, and uremia is identified in 38 per cent of the patients within 10 years of the first identified symptom.[1] It is impossible to tell who will have a more benign course, although there may be a certain familial uniformity in the course of the disease.

The complications of polycystic disease are a frequent cause for the establishment of the diagnosis. This is particularly true of hypertension. Calculi are identified in 11 to 14 per cent of the patients, with urinary tract infection occurring in one-third to one-half of the patients.[1,15] The hematuria that is a common complication of polycystic kidney disease may be initiated by exercise, urinary tract infection, trauma or the calculi present.[1] It can be entirely spontaneous and be gross, painless hematuria or microscopic. Pain can result from hemorrhage into one or more of the cysts. Renal carcinoma can arise in association with polycystic kidney disease, but it is a rare occurrence and will not be clinically suspected until surgical exploration or at autopsy. It might be demonstrated, however, by angiography as suggested by McFarland *et al.*[9] Angiography is also of use in the diagnosis of polycystic kidney disease when other methods have failed.[4,10]

Unilateral polycystic kidney disease is an extremely rare occurrence; however, this may happen when there is agenesis of the contralateral kidney or incomplete development probably due to a unilateral renal insult in early life, such as infection or obstruction. This last will often be called "hypoplasia," as in the report of Oppenheimer and Narins.[12] There have been several descriptions of unilateral polycystic disease as well which have, on further study, been demonstrated to be simply unilateral manifestations of a bilateral disease. Several of these patients have had nephrectomy with subsequent development of the polycystic elements in the opposite kidney which at surgery had appeared grossly to be normal in size, shape and general appearance.[2]

MATERIAL AND METHOD

The purpose of this study was to see if changes in renal size on serial urography had any correlation with the clinical manifestations of polycystic kidney disease. To

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TABLE I

POLYCYSTIC KIDNEY DISEASE DATA

	No. of Patients	Family History	Uremia	Hypertension	Death	Hematuria	Female	Male	Age at Diagnosis (yr.)		Observation Period (yr.)	
									Average	Range	Average	Range
Enlarged	9	4	6	8	2	4	5	4	38.5	19–47	11	2–21
Minimal Enlargement	8	4	3	5	0	5	2	6	39.5	24–44	5.1	2–11
Not Enlarged	3	2	2	2	0	1	1	2	26	3.5–38	13	8–18
Total	20	10	11	15	2	10	8	12	34.8	3.5–47	9.7	2–21

this end our material of 509 cases at the Cleveland Clinic was surveyed in order to select those cases in which there had been repeated examinations of the urinary tract so that comparisons could be made. Twenty patients with examinations separated from 2 to 21 years were selected. A summation of this data is presented in Table 1. The changes in renal size were divided into three categories: no change; increase in length of 1 to 2 cm.; and increase in length of greater than 2 cm. (Fig. 1, *A* and *B*).

DISCUSSION

Three of the patients manifested no change in kidney size over a period of observation ranging from 8 to 18 years. Two had renal failure and hypertension and 1 had hematuria when first examined. The age at discovery ranged from $3\frac{1}{2}$ to 38 years with the younger patient quite unusual and the youngest of our observation for this entity. Surgically obtained biopsy proof of his disease was available and he has suffered retarded physical maturation.

Eight patients had a small change in renal size over an observation period of 2 to 11 years with a range in age at identification of 24 to 44 years. Three of these patients had manifested renal failure, whereas 5 of them had hypertension. A similar number suffered gross hematuria. Two of these patients had surgical proof of their problem, with liver cysts identified in one.

The 9 patients observed to have the greatest change in the size of their kidneys during a period of observation varying from 2 to 21 years had the highest percentage of renal failure, hypertension, morbidity and the mortality of 2 individuals during this period; 7 of these patients had surgical proof of their disease with 2 identified to have liver cysts as well.

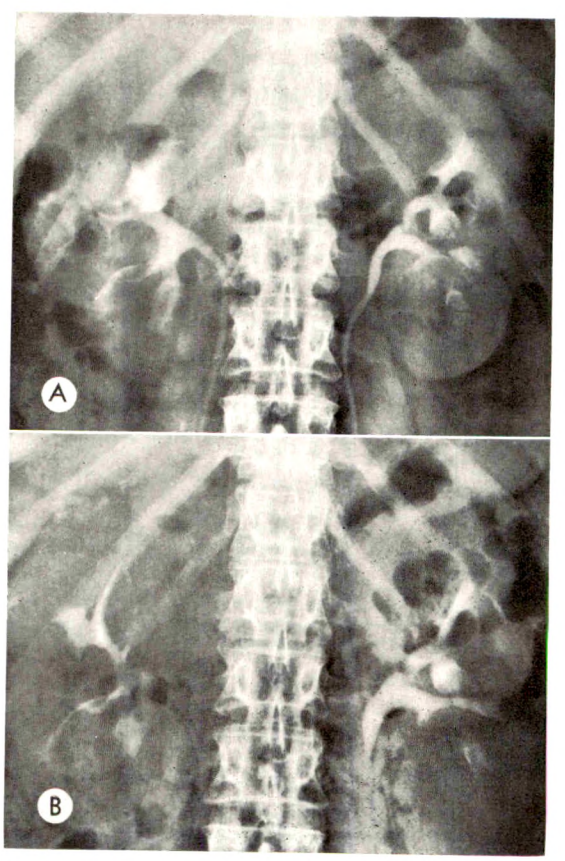


FIG. 1. Typical example of change in renal size. (*A*) Urogram of 1965 shows bilateral kidney length to be 13 cm. (*B*) Repeat urogram obtained 4 years later shows renal length now to be 18 cm. This increase in size was paralleled in this patient by an increase in the severity of the uremia, the hypertension and general debilitated state.

There are an enormous number of variables implicit in a retrospective study of the type reported here and there are many allusions in the literature to the fact that the size of the cyst is in no way dependent on the age of the patient, and in many instances is not a reflection of the severity of loss of renal function.[1,7] It has been observed earlier that there is an onset of the increase in the volume of the cyst before there is clinical evidence of functional impairment of the kidney.[2] One might observe pronounced distortion in the internal renal architecture on urography with relatively good kidney function or slight intrarenal changes and markedly diminished renal function. We would conclude from these previous studies that it is relatively difficult to make an assessment of future status on the basis of a single observation and that each case must be treated in an individual fashion. The analysis of our data, however, would suggest that separated observations demonstrating an increase in the size of the cysts is a more ominous observation than one of minimal or no change.

The kidney that appears to be entirely normal on urography during the early phase of life does not reveal, by urographic means, the abnormal pathology or suggest the portent for the development of obvious polycystic kidney disease in later life. Some authors have speculated, on the basis of experimental evidence demonstrating the production of characteristic polycystic kidney disease in fully mature rats by the chronic administration of diphenylamine, whether the disease might be due to a toxic, hereditary, metabolic defect which produces secondary changes in the kidney.[3,14]

We would agree with Hatfield and Pfister[6] that it is difficult to distinguish, in the presence of normal renal function, polycystic kidney disease from multiple simple retention cysts. The urographic appearance is helpful in this regard, however, as the cysts of polycystic kidney disease tend to be of uniform size and to cause an elongation of the collecting system as well as an elongation of the longitudinal length of the kidney itself. All of our patients had characteristic changes of polycystic kidney disease on the urogram and the majority of them did have renal failure which would be unexpected in the multiple simple retention cysts.

The enlargement of the kidneys ascends in frequency with advancing age, occurring in only 11 per cent of the patients at age 35 and at age 50 is observed in 55 per cent of patients. At age 65 the bilateral enlargement occurs in 74 per cent of the patients.[1]

Higgins[7] has indicated that 75 per cent of polycystic kidney disease is undiagnosed ante mortem. Only 2 of our patients were asymptomatic at the time of diagnosis. All of the remaining had had signs of hematuria, colic, urinary tract infection, or hypertension. The development of hypertension with or without renal failure, however, generally results in shortening of life.[1] It is for this reason that we would recommend the use of urography in the assessment of the hypertensive patient for the proper assignment of a diagnosis. It has been recommended that infusion urography be used; however, it is our impression that the production of a nephrogram during the usual rapid injection, minute sequence filming used for our hypertensive urograms is quite sufficient to demonstrate the presence of cysts which might not be of such a size as to be easily identifiable on the urographic phase (Fig. 2, *A–C*).[5,6]

Occasionally one may encounter an instance of a lesion that appears transitional between medullary sponge kidney disease and polycystic disease.[11] This suggests some relationship as proposed by Osathanondh and Potter.[13] The clinical separation and separate treatment of these two problems is rarely difficult, however.

Aspiration biopsy of the cyst may reveal a clear serous fluid or one that is dark brown or yellowish-red following hemorrhages.[1] The fluid can contain calcium deposits, cholesterol or be viscid from inflammation. The customary criteria applied in distinguishing between cysts and tumors when aspirating simple serous cysts would

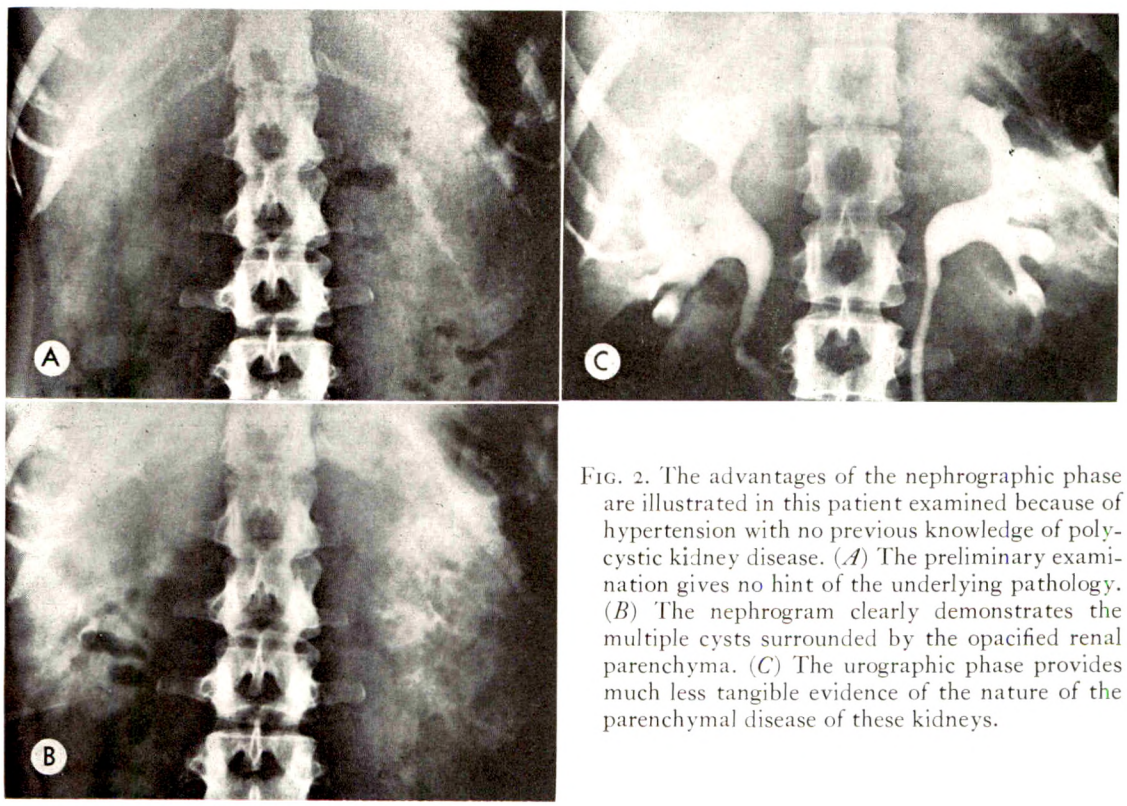


FIG. 2. The advantages of the nephrographic phase are illustrated in this patient examined because of hypertension with no previous knowledge of polycystic kidney disease. (*A*) The preliminary examination gives no hint of the underlying pathology. (*B*) The nephrogram clearly demonstrates the multiple cysts surrounded by the opacified renal parenchyma. (*C*) The urographic phase provides much less tangible evidence of the nature of the parenchymal disease of these kidneys.

not apply in this situation and would be misleading, causing the examiner to diagnose the presence of neoplasm.[8]

It was the purpose of this investigation to determine if the urographer could identify some correlation between advancing renal size and clinical complications. It is our conclusion that this is possible and that renal enlargement in separated urograms has a more ominous significance than no change or slight increase in size. This is consistent with the histologic manifestations of polycystic kidney disease in which the functioning parenchyma is displaced by a rather large number of tightly-packed cysts so that the cystic volume eventually predominates over the solid parenchyma.[2]

SUMMARY

Twenty patients with polycystic kidney disease of the adult type were studied as a result of available examinations over varying periods of time.

Correlations were made between advancing renal size and the presence of clinical complications.

It was ascertained that an observed increase in renal size was in fact correlated with the severity of the disease and could be used as an indicator of probable shortened life expectancy.

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ROENTGENOLOGIC TRIAD OF CONGENITAL MULTICYSTIC KIDNEY*

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CONGENITAL multicystic kidney is a dysplastic kidney, characterized by unilateral involvement and manifested roentgenographically as a nonfunctioning kidney. Pathologically, the normal renal tissues are completely replaced by a conglomeration of various sized cysts. There is no identifiable renal parenchyma accounting for the nonfunction. The dysplastic renal disease is usually discovered in infancy or childhood and presents itself as an asymptomatic flank mass. It is the most common cause of an abdominal mass in infancy and furthermore, it is the most common manifestation of cystic renal disease in infants and children.[6,7,12] A few cases may persist into adult life, often discovered incidentally during the work-up of other urologic problems.[1,2,4,7,13,14,19]

Although the disease characteristically involves one kidney only, contrary to previous belief, there is a significantly increased incidence of associated congenital abnormalities in the contralateral kidney and ureter, reported to occur in about 30 per cent of the cases.[7] Early roentgenologic recognition and evaluation of this entity, therefore, is obviously important, not only in establishing the primary diagnosis, but also to detect the associated abnormalities of the contralateral kidney. Appropriate corrective surgical procedures must be performed before the renal function of the only remaining kidney becomes compromised. Even though congenital multicystic renal disease is pathologically distinct, the urographic appearance is that of a nonfunctioning kidney, which cannot be readily differentiated from congenital unilateral hydronephrosis, Wilms' tumor in children and renal cell carcinoma in adults, renal vein thrombosis, renal artery occlusion, and xanthogranulomatous pyelonephritis. There are, however, characteristic distinguishing features of congenital multicystic kidney in various diagnostic roentgenologic procedures which make up the composite features for the roentgenologic triad of diagnostic findings. Differentiation of congenital multicystic kidney from other renal lesions which cause unilateral nonfunction is extremely important. The prognosis is excellent in the case of an uncomplicated congenital multicystic kidney, which should not be mistakenly diagnosed as any of the other more serious renal diseases that are considered in the differential diagnosis.

The purpose of this presentation is to emphasize the importance of roentgenologic recognition of this entity which can readily be diagnosed by employing a combination of 3 basic diagnostic roentgenologic procedures to reveal the characteristic pathologic features of congenital multicystic renal disease. In this report, a survey of the literature on pathologic and clinical features is made and the diagnostic findings in 6 proven cases are presented to establish the pathologically correlated roentgenologic features of this entity. The roentgenologic triad thus created is diagnostic. Awareness of these diagnostic features will help in making the correct diagnosis and prevent an unnecessary surgical intervention for this innocuous congenital disorder with excellent prognosis, which is considered by far as the most common cause of abdominal masses in infancy.

ROENTGENOLOGIC FEATURES

PLAIN ROENTGENOGRAM AND INTRAVENOUS UROGRAM

The preliminary survey roentgenogram of the abdomen usually shows a mass lesion in the region of the kidney on the involved

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side, displacing the adjacent bowel loops (Fig. 1*A*). It is very useful to obtain a lateral view of the abdomen, where the mass can be seen arising from the renal fossa posteriorly and extending anteriorly (Fig. 1*B*). The mass effect is characteristically seen in infants and children; it may also be seen in adults, but this finding is not always constant. In approximately 27 per cent of the adult patients there are characteristic ovoid or ring-like densities, outlined by a thin peripheral shell of calcium in the region of the kidney (Fig. 2*A*; and 5*A*).[13] Not uncommonly, there is more than one calcified ring-like density.

The kidney shows no function in the intravenous urogram. There is usually compensatory hypertrophy of the remaining kidney (Fig. 2*B*). If the disease is complicated by the association of other congenital abnormalities of the contralateral kidney and ureter, the manifestations of the underlying anomaly may be seen in the intravenous urogram. The most common associated abnormality is hydronephrosis, either of the entire collecting system or localized to the renal pelvis and calyces due to ureteropelvic junction obstruction (Fig. 3; and 5*B*).

RETROGRADE PYELOGRAM

The ureter in the retrograde pyelogram is almost always atretic, especially in its proximal half. The appearance of such a ureter in the retrograde pyelogram is that of small caliber hypoplastic appearing ureter which ends blindly in its proximal portion (Fig. 4). There is no filling of the renal pelvis or calyces. When such an abrupt blind ending ureter is not recognized during retrograde pyelography and additional force is used in injecting the contrast medium, extravasation into the retroperitoneal space invariably occurs (Fig. 5*D*). Such an incident is observed not infrequently, especially in adult patients (Cases II and III). The appearance of the visualized portion of the ureter shows multiple pseudosacculations or pseudodiverticula (Fig. 2*C*; 4; and 6*A*). According to one report[1] these pseudosacculations are simply submucosal cyst formations, similar to von Brunn's epithelial cysts of ureteritis cystica, which on gross inspection appear as polypoid nodulations in the lumen of the ureter. The presence of such pseudodiverticula or pseudosaccules in the ureter in the retrograde pyelogram, when seen together with an atretic proximal ureter, is believed to be characteristic of a dysplastic kidney.

ABDOMINAL AORTOGRAM

Usually there is no identifiable renal artery and no collateral vessels on the affected side (Fig. 6*B*). At times, especially in infants and children, there may be a very hypoplastic thread-like artery, seen in the general location of the renal artery, but the vessel is so small that it cannot be positively identified (Fig. 1*C*). During the nephrographic phase there is no nephrogram and no renal vein is seen during the venous phase of aortography (Fig. 1*D*; and 6*C*). In the presence of a flank mass or ovoid shells of thin calcifications in the region of the kidney, an absent renal artery without a nephrogram in the abdominal aortogram indicates a congenital multicystic kidney. This feature is believed to be diagnostic and it correlates well with the pathologic features.

DISCUSSION

Congenital multicystic renal disease was first described by Schwartz[18] in 1936 following nephrectomy in a 7 month old infant. There were several names given to this entity which has led to much confusion in the literature; the disease is most frequently confused with true polycystic renal disease. Various synonyms include: unilateral multicystic disease; multicystic renal dysplasia; unilateral polycystic renal disease; polycystic renal disease with abundant stroma; renal dysplasia; hypoplastic multicystic kidney; and congenital unilateral multicystic kidney.[6,19] In 1955, Spence[19] popularized this disease and classified it as a distinct entity, which is to be distinguished from polycystic kidney. A congenital multicystic kidney, unlike poly-

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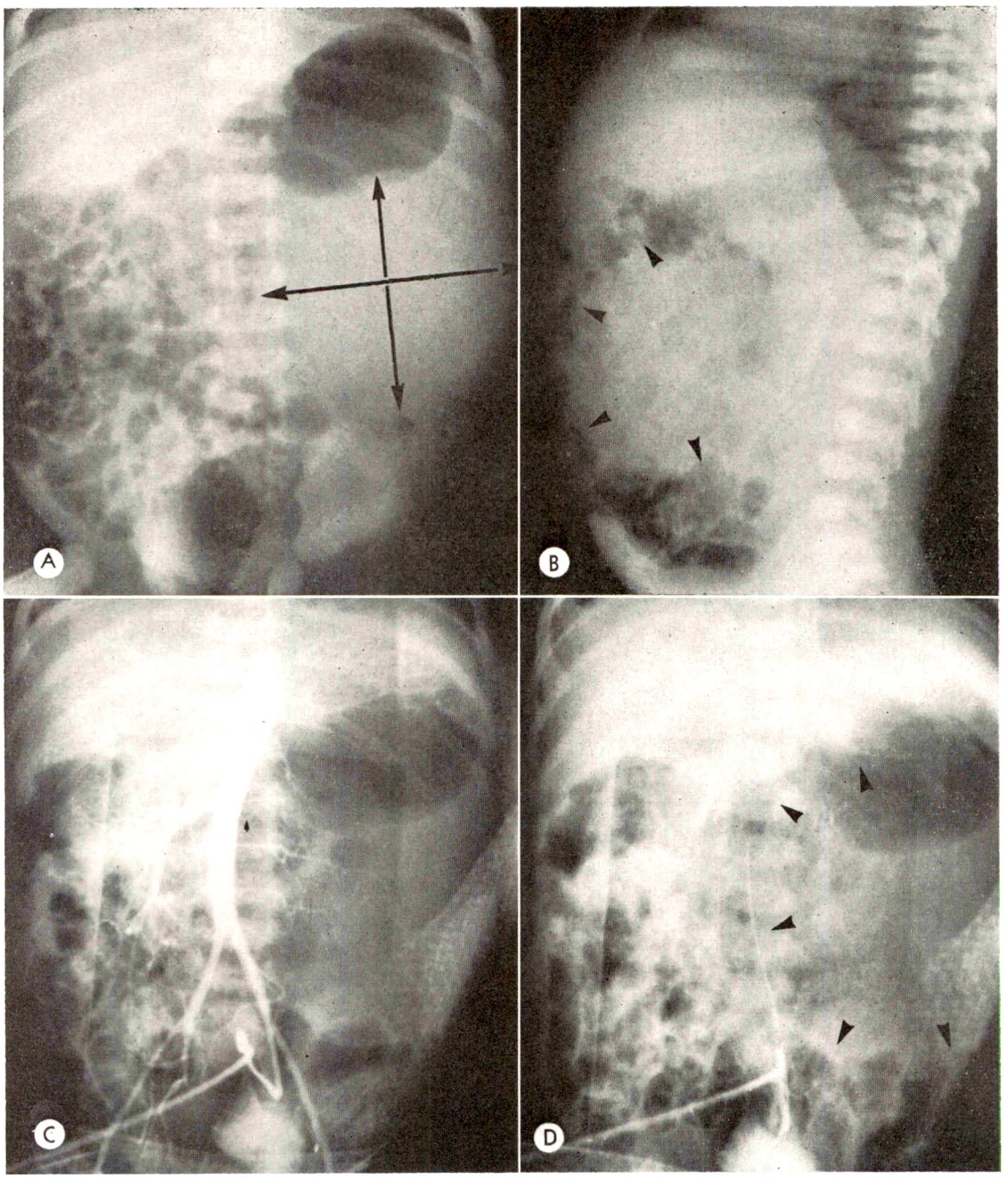


FIG. 1. Case 1. (*A*) Intravenous urogram in anteroposterior view reveals a large left flank mass displacing the gas-filled bowel loops. No renal function is present on the left side. (*B*) Lateral view shows the extent of the flank mass. (*C*) Abdominal aortogram (*via* indwelling umbilical arterial catheter). Arterial phase shows a large avascular left flank mass indenting and displacing the abdominal aorta to the right. The arrow indicates a probable thread-like hypoplastic renal artery. (*D*) Late nephrographic phase demonstrates the avascular mass, but there is no nephrogram and the renal vein on the left side is absent.

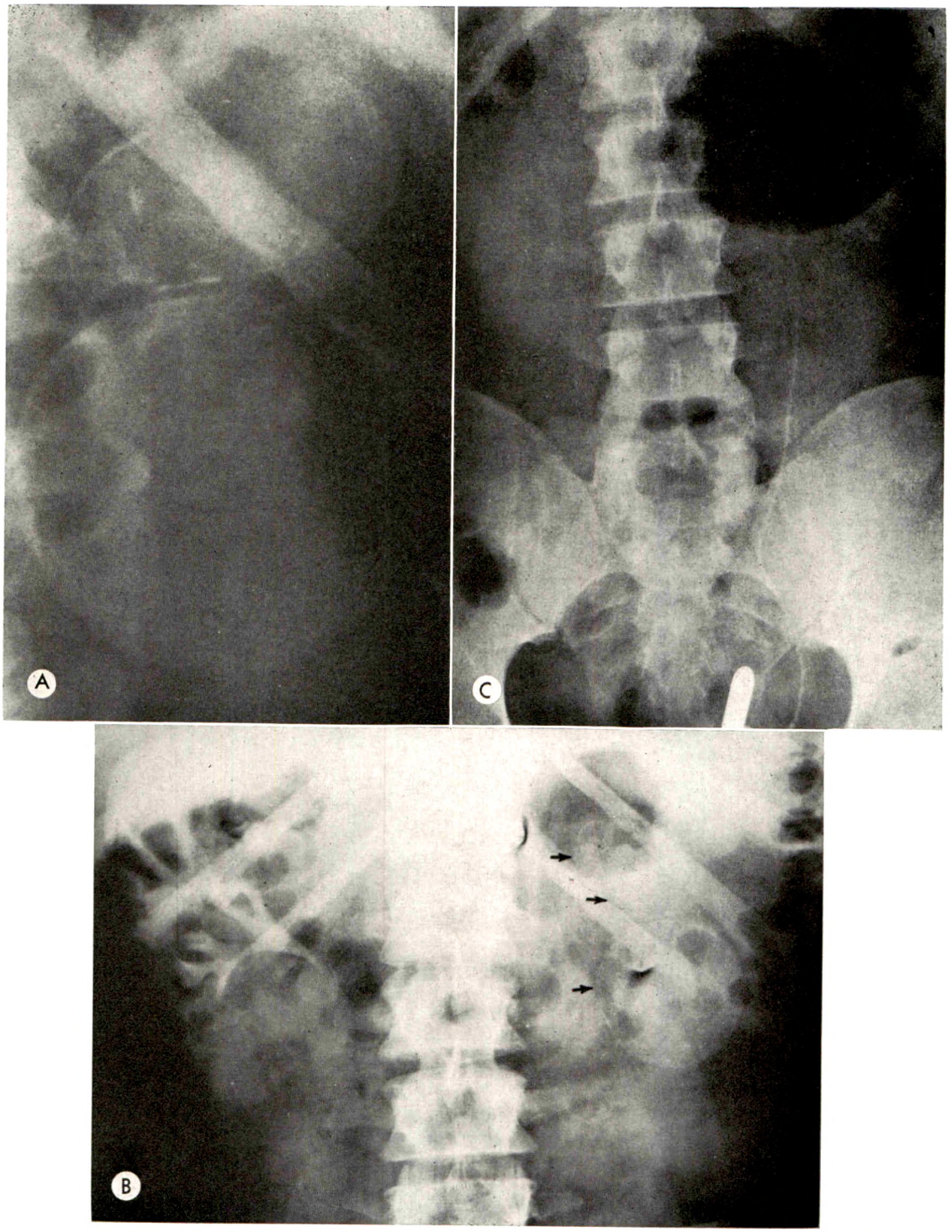


Fig. 2. Case 11. (*A*) Close-up view of shell-like calcified rings seen on the plain roentgenogram. (*B*) Intravenous urogram shows no renal function on the left side and compensatory hypertrophy of the right kidney. (*C*) Left retrograde pyelogram demonstrates atretic proximal ureter. Classical beaded appearing pseudosacculations of the distal ureter are seen.

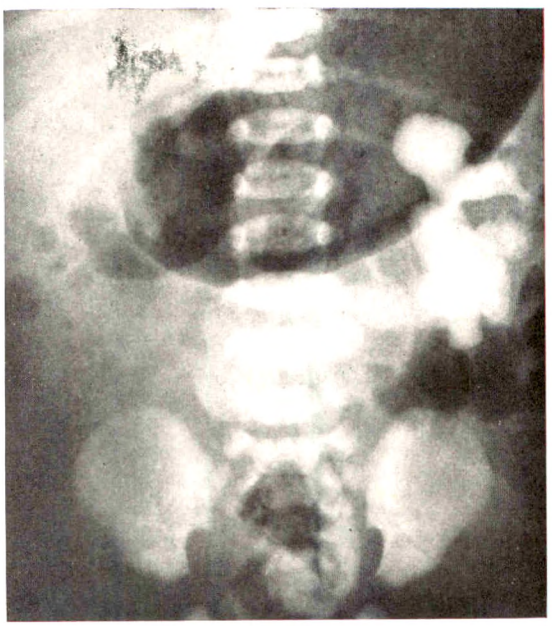


FIG. 3. Case III. Intravenous urogram shows no function of the right kidney, and hydronephrosis and hydroureter on the left side.

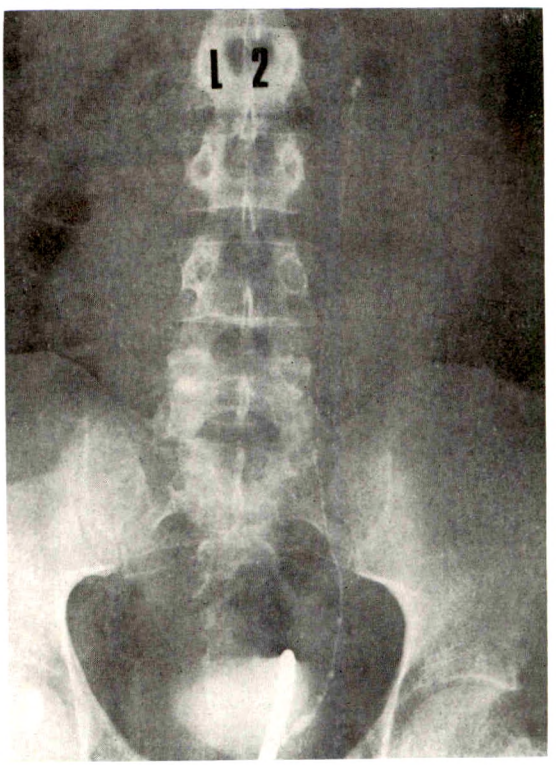


FIG. 4. Case IV. Left retrograde pyelogram shows atretic proximal portion and the characteristic pseudodiverticula or pseudosacculations in the patent segment of the ureter.

cystic renal disease, is neither a familial nor an hereditary condition characterized by unilateral involvement. It is a congenital lesion believed to have been acquired *in utero* and formed as a result of disturbed differentiation of nephrogenic tissues during the embryonal life. The end result is disorganized renal parenchyma with persistence of mesonephric primitive renal tubules and maldeveloped glomeruli which are inappropriate for the functional requirements of the postnatal renal apparatus. It is important not to confuse congenital multicystic kidney with true polycystic kidney disease, because the former, especially when uncomplicated, has an excellent prognosis and the latter is a renal disease with a grave prognosis which is ultimately fatal.

There are many theories for the etiology and pathogenesis, but none has been proved universally acceptable.[9–11,17] Regardless of the exact etiology, the important and practical point we now know is that the congenital multicystic kidney is a distinct clinicopathologic entity resulting from a congenital defect(s) due to fetal maldevelopment, not on an hereditary basis, and is characterized by unilateral involvement.

Grossly, the congenital multicystic kidney consists of an innumerable number of varying sized cysts which are not connected internally, but are held together by loose connective tissues. The topographic appearance of such a kidney is like that of a cluster of grapes. Calyces are absent and the ureter is almost always atretic in its proximal part or it may be entirely absent. Histologically, the cyst walls are lined by tall cuboidal epithelium and the cysts usually contain clear or pale yellow fluid. There is an abundant undifferentiated mesenchymal stroma amongst the cysts, which retains its embryonal primitive features. Foci of primitive cartilage and lymphoid tissues may be found. In general, normally developed parenchymal tissues cannot be identified, either grossly or

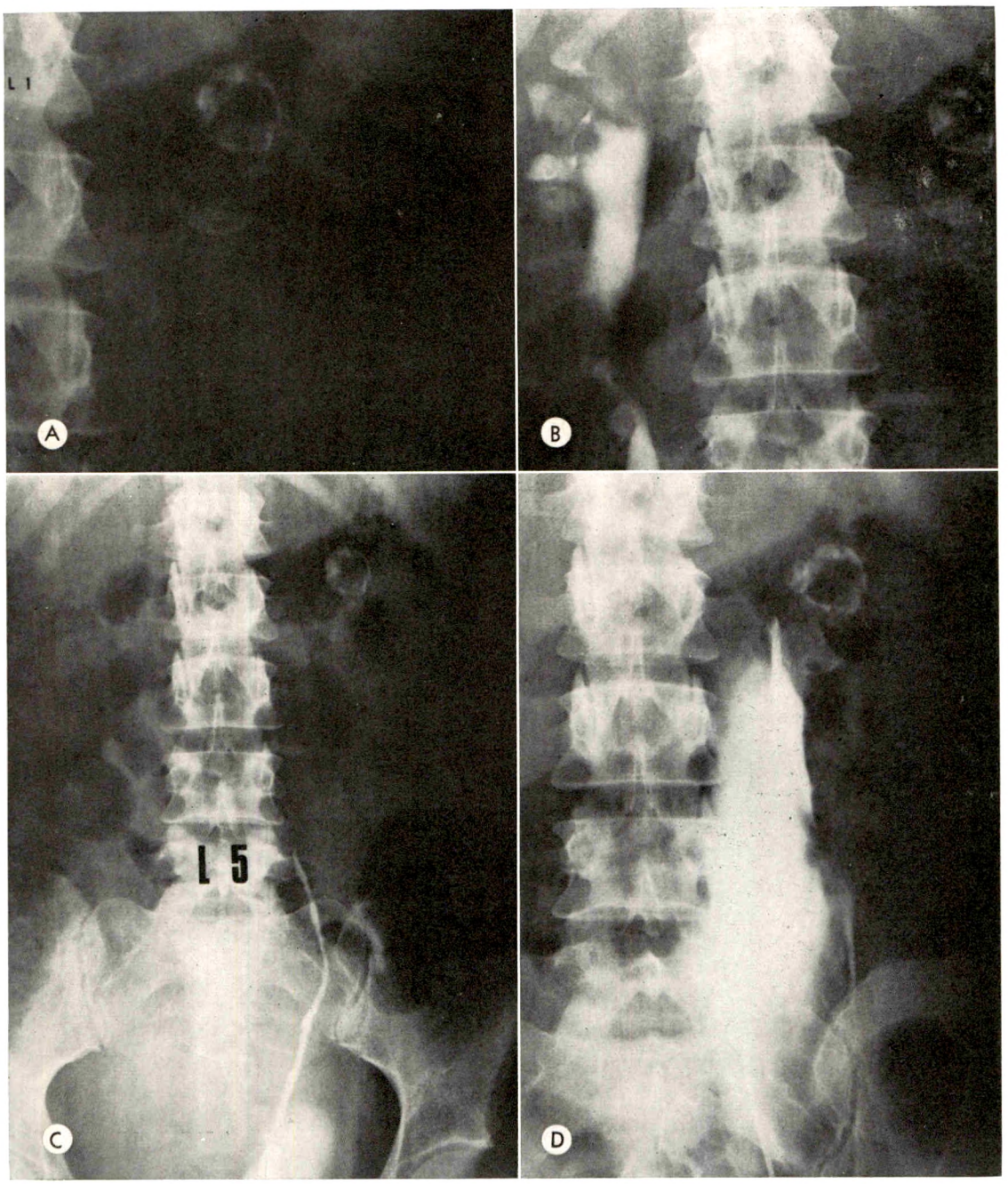


Fig. 5. Case v. (*A*) Close-up roentgenogram shows 3 or 4 ring-like calcified densities in the left flank. (*B*) Intravenous urogram demonstrates no function of the left kidney and a small right kidney with chronic pyelonephritis and hydronephrosis secondary to ureteropelvic function obstruction. (*C*) Left retrograde pyelogram shows patent distal ureter with some pseudodiverticular formations and atretic proximal ureter. (*D*) Extravasation from the atretic ureter when more pressure was used in injecting the contrast medium.

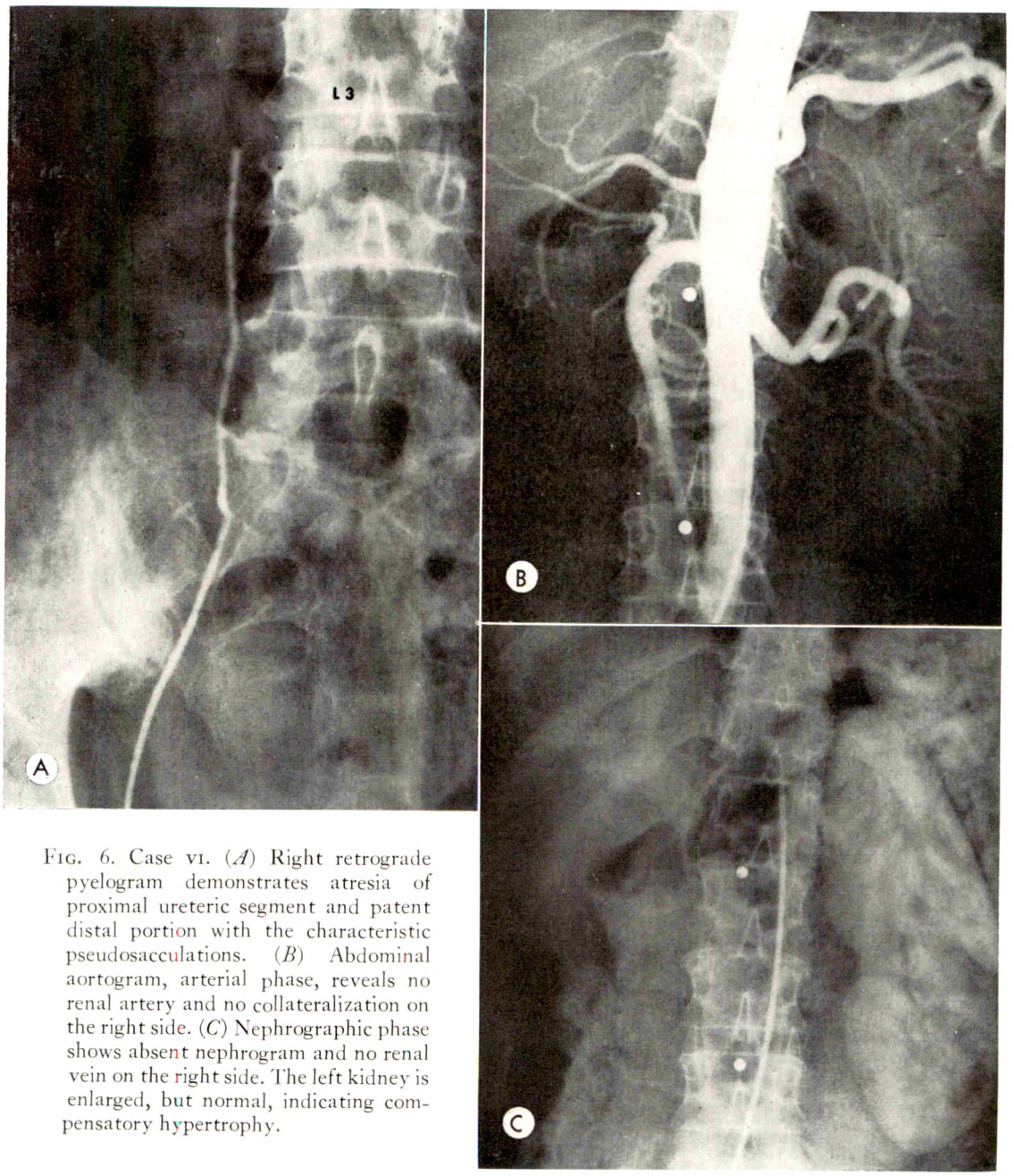


Fig. 6. Case vi. (*A*) Right retrograde pyelogram demonstrates atresia of proximal ureteric segment and patent distal portion with the characteristic pseudosacculations. (*B*) Abdominal aortogram, arterial phase, reveals no renal artery and no collateralization on the right side. (*C*) Nephrographic phase shows absent nephrogram and no renal vein on the right side. The left kidney is enlarged, but normal, indicating compensatory hypertrophy.

histologically. The renal artery is almost always absent or, when present, is thread-like and so small that it cannot be accurately detected. The intrarenal vasculature is insignificant and disorganized.[12]

Associated anomalies occur in the contralateral kidney in about 30 per cent of the cases that Greene and his associates described.[7] Among the various associated anomalies of the contralateral urinary tract are ureteropelvic junction obstruction, malrotation of the kidney and segmental ureteral disparity.[7]

The prognosis of congenital multicystic

kidney when uncomplicated is excellent. Males and females are equally affected and there is no predilection for the side of involvement. Urinalysis and other pertinent laboratory studies are usually normal. Malignant transformation has not been reported nor have hypertension or other systemic diseases of renal origin been seen. The clinical data of the 6 proven cases of this series are listed in Table I.

Extirpation of the nonfunctioning dysplastic kidney has been the treatment of choice. The main reason for surgical intervention is to confirm the clinically suspected diagnosis of congenital multicystic kidney, since the features of this entity and other more serious renal disorders can be essentially alike on clinical grounds and there has been no accurate means of diagnosing this entity short of surgery. All the available published data document that there is no known evidence, indicating that such avascular dysplastic kidney *per se*, if left alone, causes harmful effects or complications to the body. Since the main purpose of surgically removing such a harmless anatomic structure has been to confirm the clinically suspected diagnosis, surgery may be avoided, if there is an accurate way of making the absolute diagnosis of this entity.

By employing intravenous urography, retrograde pyelography and abdominal aortography, an accurate roentgenologic diagnosis can be made, provided the combined positive features of congenital multicystic kidney are detected by these methods. Each of these methods detects only one pathologic manifestation of congenital multicystic renal disease and it is essential to utilize all 3 diagnostic disciplines to make the absolute positive diagnosis. The roentgenologic features of the diagnostic triad thus formed are pathognomonic for congenital multicystic renal disease (Table II). A plain roentgenogram and intravenous urogram permit detection of the presence of renal function, a renal mass or the characteristic ring-like calcifications. A retrograde pyelogram enables detection of the

TABLE I

CLINICAL DATA

Case	Age and Sex	Symptoms or Clinical Diagnosis	Blood Pressure	Urinalysis and Pertinent Laboratory Data	Status of Contralateral Kidney	Treatment
I	Newborn	Palpable left abdominal mass	Normotensive	Normal	Normal	Nephrectomy
II	33	Intermittent abdominal pain for 4 months (subsequently discovered to have cholecystitis and cholelithiasis)	Normotensive	Normal	Normal (compensatory hypertrophy)	Nephrectomy
III	Newborn	Palpable right flank mass	Normotensive	Normal	Hydronephrosis and hydroureter	Nephrectomy
IV	44	Prostatitis	Normotensive	Microscopic hematuria	Normal (compensatory hypertrophy)	Nephrectomy
V	35	Chronic pyelonephritis and renal failure	Hypertensive	Pyuria, azotemia, positive urine culture	Atrophic chronic pyelonephritis; ureteropelvic junction obstruction	Bilateral nephrectomies and allograft renal transplantation
VI	48	Stress urinary incontinence	Normotensive	Normal	Normal (compensatory hypertrophy)	Nephrectomy

TABLE II

ROENTGENOLOGIC TRIAD OF CONGENITAL MULTICYSTIC RENAL DISEASE

1. Unilateral nonfunctioning kidney on the excretory intravenous urogram with a flank mass or with shell-like calcifications on the plain roentgenogram
2. Absent or atretic proximal ureter on the retrograde pyelogram
3. Absent or hypoplastic renal artery, no collateral arterial supply and no nephrogram on the abdominal aortogram

presence of an atretic ureter. The abdominal aortogram reveals absence or hypoplasia of the renal artery. Since a congenital multicystic kidney by definition is a dysplastic kidney with no function, with an atretic or absent ureter, and with absent renal vasculature, these 3 diagnostic steps are tailored to bring out the specific functional and pathologic features characterizing congenital multicystic kidney.

There is a group of renal lesions to be considered in the differential diagnosis, which present with unilateral nonfunction or nonvisualization of the kidney in the intravenous urogram, with or without an associated palpable flank mass. True polycystic kidney, although previously known to be confused with congenital multicystic kidney, is now more or less recognized as a bilateral renal disorder and, therefore, should not enter in the differential diagnosis of a unilateral nonfunctioning kidney. There are several renal or perirenal lesions which may cause unilateral nonfunction of a kidney. The most common among them, from the point of view of differential diagnosis, are: congenital unilateral hydronephrosis; Wilms' tumor in children and renal cell carcinoma in adults; renal vein thrombosis; renal artery occlusion; xanthogranulomatous pyelonephritis; and renal agenesis. However, the diagnostic findings of the "roentgenologic triad" described in this report (Table II) are present only in the congenital multicystic kidney, so that the other renal lesions may be eliminated on this basis.

SUMMARY

Congenital multicystic kidney is the most common cause of an abdominal mass in infancy. The disease may persist into the adult life and be incidentally discovered. Although it became a distinct pathologic entity in 1955, clinical and roentgenologic diagnosis of a congenital multicystic kidney could not be made with certainty. This is because the diagnostic features consisting of a unilateral nonfunctioning kidney with or without a palpable flank mass are also observed in Wilms' tumor in children and renal cell carcinoma in adults, congenital unilateral hydronephrosis, renal vein thrombosis, renal artery occlusion, renal xanthogranulomatous pyelonephritis and renal agenesis. In view of lack of a diagnostic procedure to make an accurate diagnosis, these patients have been subjected to surgical exploration to confirm the clinical suspicion of congenital multicystic kidney, so as to make certain that one is not dealing with the more serious or malignant renal diseases considered in the differential diagnosis.

Roentgenologic detection of pathologically correlated diagnostic features can be made by employing the intravenous urogram, retrograde pyelogram, and abdominal aortogram, in combination. These procedures bring out selectively functional and pathologic features of congenital multicystic kidney. The diagnostic features each procedure displays complement the other two, to establish the pathognomonic triad of roentgenologic signs for congenital multicystic renal disease which is outlined in Table II.

These diagnostic procedures not only make the primary diagnosis of congenital multicystic kidney possible, but also reveal the nature of the underlying congenital anomaly of the contralateral urinary tract, which occurs in 30 per cent of these patients. Consequently, appropriate thera-

peutic measures may be undertaken to prevent deterioration of the only functioning kidney.

Emphasis is placed on the roentgenologic recognition and an awareness of the nature and significance of this innocuous renal lesion which, when left alone, is not known to be harmful to the body. If one is cognizant of the diagnostic value of the roentgenologic triad described in this report, an accurate diagnosis can be made and consequently surgical intervention for exploration can be averted. This is important since congenital multicystic kidney is the most common cause of abdominal masses in children.

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ANTEGRADE PYELOGRAPHY IN A HORSESHOE KIDNEY*

By E. W. L. FLETCHER *and* M. G. W. KETTLEWELL
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ANTEGRADE pyelography is a well proven method of demonstrating the urinary collecting system when excretory urography fails to do so and when retrograde pyelography cannot be performed.[1–8]

We have recently performed this examination on the nonfunctioning side of a horseshoe kidney.

REPORT OF A CASE

A 72 year old man was admitted so the Radcliffe Infirmary with 6 weeks painless hematuria followed by 2 days right loin pain and profuse hematuria. On examination, a firm smooth mass was felt to the right of the umbilicus.

Excretory urography was performed on March 26, 1973 and this showed a normally functioning left side of a horseshoe kidney, while the right side was nonfunctioning and had a poor nephrogram (Fig. 1). A neoplasm of the right part of the kidney was suspected and selective angiography of the 2 left and 3 right renal arteries was performed on April 3, 1973, which showed small right renal arteries with narrow stretched branches around a medially situated cystic area (Fig. 2). These appearances were interpreted as being due to hydronephrosis and right ureteric catheterization prior to retrograde pyelography was attempted. Catheterization of the ureter was unsuccessful and therefore antegrade pyelography was performed on April 4, 1973 to show the site of obstruction.

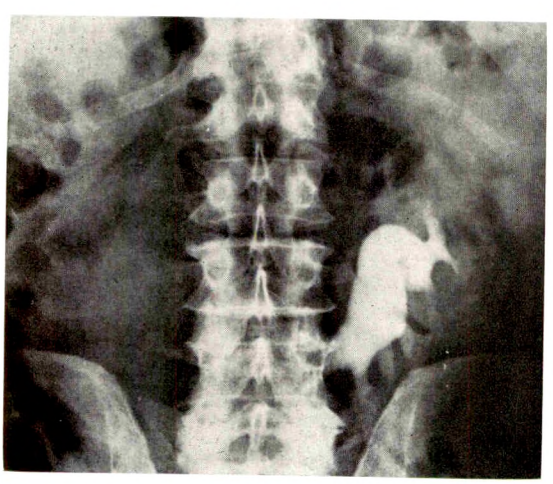


FIG. 1. Excretory urogram shows the normal left half and the nonfunctioning right half of the horseshoe kidney.

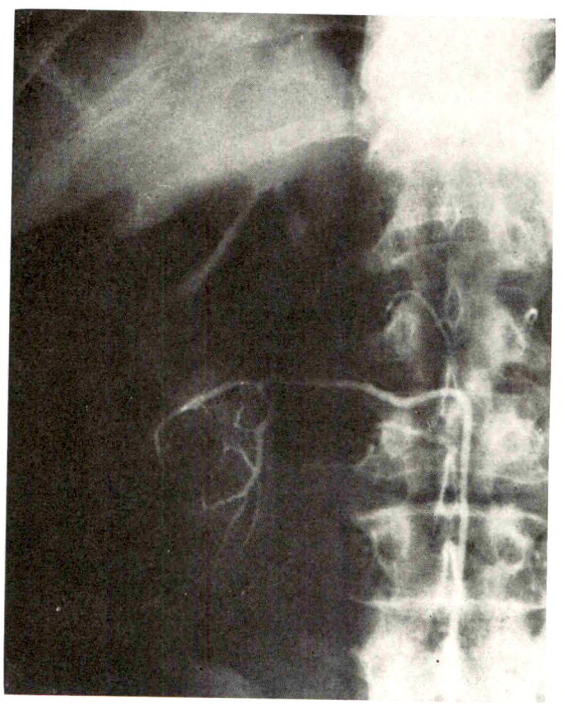


FIG. 2. Selective angiogram of one of the right renal arteries. The arteries are small and splayed around the hydronephrosis.

The patient was placed in a prone position and fluoroscopy showed the right side of the horseshoe kidney was to the right of L_5 vertebral body. This site was marked on the skin of the patient's back and local anesthetic was injected. A 20 gauge Harris lumbar puncture needle 12 cm. long was directed anteriorly through the patient's back. This needle was too short, and a No. 16 end-hole translumbar aortogram needle was introduced to a depth of 14 cm. before changed blood mixed with urine

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was encountered. An amount of 200 ml. of this fluid was removed before 120 ml. of Urografin 60* was injected. Straight and oblique roentgenograms showed dilated calyces and renal pelvis but, although the upper ureter was shown, the site of obstruction was not demonstrated. The needle was removed and the patient stood erect while posteroanterior and oblique roentgenograms were made. These showed occlusion of the right ureter approximately 4 cm. from the pelvi-ureteric junction, with a small tail of contrast material extending further down the ureter (Fig. 3), and the diagnosis of neoplasm of the ureter was made.

A neoplasm of the ureter below the isthmus of the horseshoe kidney with gross right hydronephrosis was found at operation. The hydronephrotic right half of the horseshoe kidney and the neoplastic ureter were removed. Histology revealed an invasive transitional cell carcinoma of the ureter and obstructive hydronephrosis of the right half of the kidney.

* Schering A. G., Berlin.

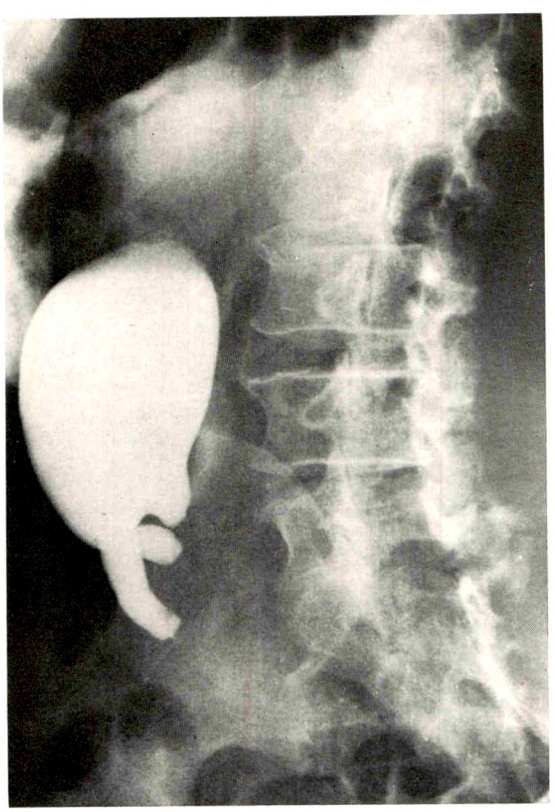


Fig. 3. Antegrade pyelogram. The patient is erect and in an oblique position to show the ureteric obstruction with the 'tail' of contrast medium extending into the ureteric neoplasm.

DISCUSSION

Antegrade pyelography was easily performed on this horseshoe kidney but with a few small modifications of the normal procedure. The calyces and pelvis of the horseshoe kidney lie lower, more medially and more anteriorly than those of the normally situated kidney. The needle has to traverse the thick muscles of the back and the full thickness of the kidney before reaching the collecting system and this requires a needle approximately 16 cm. long. In addition, the transverse process of the fifth lumbar vertebra lies over the calyces and pelvis of the horseshoe kidney, and makes puncture of the collecting system more difficult. When the needle was in the patient's kidney it did not move with respiration and it was difficult to be sure when the renal substance had been entered. No complications of the procedure were encountered.

SUMMARY

Antegrade pyelography was performed on the nonfunctioning side of a horseshoe kidney and revealed the site and nature of the obstruction of the collecting system.

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We would like to thank Mr. J. C. Smith for allowing us to present this patient, and the Medical Illustration Department of the Radcliffe Infirmary for the illustrations.

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RENAL SIZE IN VARIOUS NEPHROPATHIES*

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RENAL size is one parameter in assessing renal disease roentgenologically. The causes of bilateral renal enlargement with minimal or no pelvicalyceal distortion are shown in Table I.

Two years ago one of the authors performed renal arteriography on a patient clinically suspected of having polycystic disease. The excretory urogram revealed bilateral enlarged kidneys with no other apparent abnormality. The arteriogram (Fig. 1) confirmed the very large kidneys with no evidence of intrarenal cysts, and no distortion of the renal arteries or pelvicalyceal system. As the diagnosis was still uncertain, the patient subsequently had a renal biopsy revealing lobular glomerulonephritis.

This case initiated a retrospective and prospective study of renal size in patients who had renal biopsies revealing one of the following entities:

1. Latent glomerulonephritis
2. Lobular glomerulonephritis
3. Lipoid nephrosis
4. Membranoproliferative glomerulonephritis and
5. Membranous glomerulonephritis.

These pathologic conditions consitute a group of diseases which have recently been more precisely defined primarily with the aid of electron microscopy and immunofluorescent staining.[5] A thorough search of the literature failed to reveal any previous radiographic study of renal size in these conditions.

TABLE I

CAUSES OF BILATERAL RENAL ENLARGEMENT WITH MINIMAL OR NO PELVICALYCEAL DISTORTION[2–4,6,8–10,12–14]

1. Acute glomerulonephritis
2. Lymphomas, leukemia, SLE
3. Polycystic disease
4. Bilateral renal vein thromboses
5. Rarely—amyloidosis, sickle cell disease, sarcoidosis

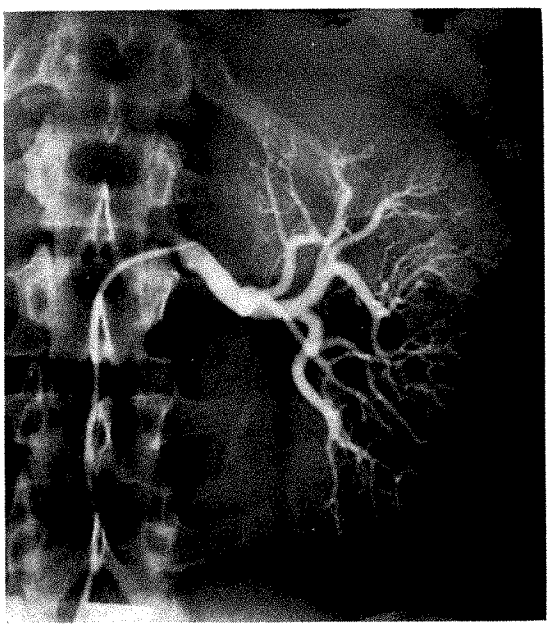


FIG. 1. Selective renal arteriogram of a 25 year old male showing grossly enlarged kidney with no arterial distortion.

METHOD

The clinical findings of the 68 patients studied ranged from no symptoms to the occasional full blown nephrotic syndrome. Most of our patients presented with a minor degree of urinary abnormality such as proteinuria or hematuria. All of the patients had a percutaneous renal biopsy within a week of excretory urography.

Excretory urograms were made using a 40 inch target film distance. Preliminary roentgenograms or early excretory supine urograms were used whenever possible to minimize the effect of compliance.[1] Mea-

* Presented at the Seventy-third Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 3–6, 1972.
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surements were made independently by the 3 authors using the method of Simon[11] (Fig. 2). The length of each kidney was determined in its longest axis. The height of the 2nd lumbar vertebral body was measured including the L2-L3 disk. These latter measurements were made at the posterior margins of the vertebral bodies. The ratio of the renal size divided by L2 plus the disk of L2-L3 was calculated. The normal mean in adults for this ratio is 3.1 ±0.27.

For example, the patient in Figure 3 had grossly enlarged kidneys on excretory urography. Of incidental note are the changes of old pyelonephritis involving the right upper pole, but there is no evidence of a space occupying lesion to account for the increased renal size. The renal ratio was calculated to be 3.8, which is beyond 2 standard deviations above normal. Electron microscopy revealed lobular glomerulonephritis.

In children the kidney lengths were compared to the patient's age using the standard of Hodson.[7]

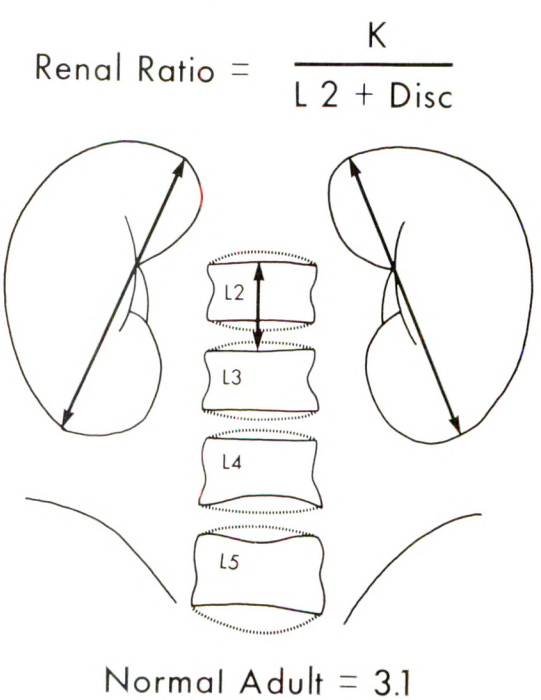


FIG. 2. Normal renal size according to Simon.[11]

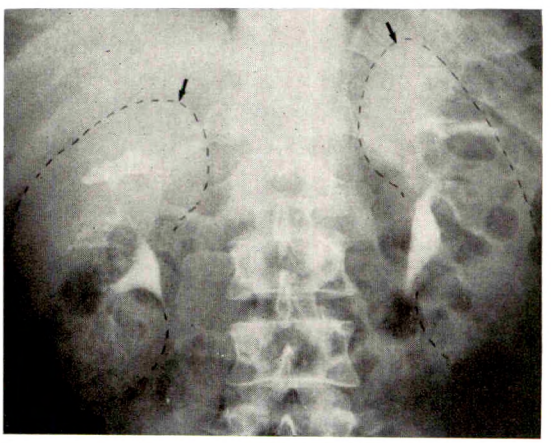


FIG. 3. An intravenous pyelogram of a 59 year old male with minimal proteinuria and biopsy proved lobular glomerulonephritis.

RESULTS

Means, standard deviations and t values were calculated for the test groups as shown in Table II. Graphically the results can be displayed by plotting the probability of occurrence against the specific renal ratios (Fig. 4).

In the case of latent glomerulonephritis we did not expect to find an increase in renal size and, in fact, our group of patients fell entirely within the normal limits. With lobular glomerulonephritis and lipoid nephrosis these curves extended prominently beyond the normal curve and statistically these kidneys were enlarged ($p < 0.025$). However, a moderate degree

TABLE II

RESULTS

	N	$\bar{X}$	DS	t
Lobular glomerulonephritis	18	3.4	.47	2.1(t')*
Lipoid nephrosis	11	3.3	.30	1.8*
Membranous glomerulonephritis	12	3.2	.42	1.3(t')
Membranoproliferative glomerulonephritis	4	3.3	.26	1.5
Latent glomerulonephritis	13	3.2	.12	1.5(t')
Normal[11]		3.1	.27	

N = number; $\bar{X}$ = mean renal ratio; SD = standard deviation; t = t test.

* Enlargement with a significance of $p < 0.025$.

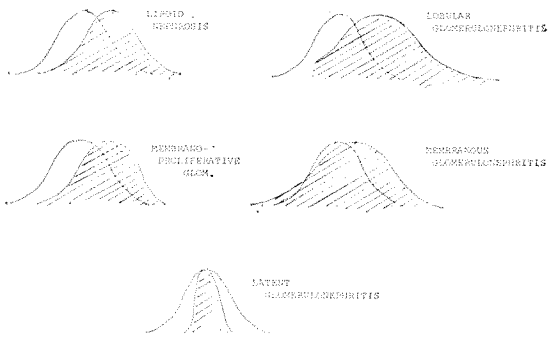


FIG. 4. Results, plotting probability of occurrence *versus* renal ratio.

of overlap exists between these 2 entities and the normal range; thus, a normal renal ratio does not exclude either of these conditions from the differential.

Although the means of membranous and membranoproliferative glomerulonephritis were above normal, statistically they could not be differentiated from the normal group.

DISCUSSION

It is only in recent years that renal biopsy has been used to study these conditions in the hope that their earlier, more accurate diagnosis will lead to better therapeutic regimens. We feel that radiologically this is an important group of conditions to be aware of, as these patients may present asymptomatically with only minor proteinuria or hematuria. The radiologic detection of significantly enlarged kidneys with normal pelvicalyceal systems may prompt the nephrologist to perform early renal biopsy. Thus the excretory urogram may not only exclude gross pathology but also may provide information to expedite biopsy and diagnosis.

No satisfactory explanation for the renal enlargement in lobular glomerulonephritis and lipoid nephrosis is apparent. In neither of these entities did we consistently find the microscopic hypercellularity of acute glomerulonephritis nor the tissue edema of the nephrotic syndrome. The possibility of the enlargement being related to alteration in the tubular cells secondary to proteinuria is presently being investigated.

SUMMARY

Renal sizes in a group of 68 patients suffering from various nephropathies were compared to expected normal values.

Kidneys with lipoid nephrosis and lobular glomerulonephritis were shown to be statistically increased in size, whereas no statistical difference was noted in cases of membranous, membranoproliferative and latent glomerulonephritis. These 2 relatively new entities should be included in the differential diagnosis of enlarged kidneys with no other apparent abnormality on excretory urography.

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The authors wish to express their thanks to Dr. W. H. Chase for providing the biopsy data with their interpretation. Dr. Don Newman was of great assistance in the organization of the paper. Special thanks are extended to Dr. Brenda Fraser who calculated the statistics.

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RENAL ADENOMA: RETROSPECT AND PROSPECT*

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ALTHOUGH small renal adenomas are common pathologic findings at autopsy, the large solitary adenoma is a much less common lesion. Its discovery may lead to considerable confusion due to the existing consensus of opinion, primarily in the English language literature, that size is critical to the pathologic classification of the lesion as benign or malignant. Support of this opinion can be found in recently published textbooks of both pathology and radiology.

It is the purpose of this paper to discuss the large solitary renal adenoma from the standpoint of the pathologic and roentgenographic findings with a historic perspective. The following case prompted our investigative efforts.

REPORT OF A CASE

A 25 year old male presented with the symptoms of left ureteral colic and gross hematuria. His past history was unremarkable with the exception of one episode of pyelonephritis at the age of 12. No roentgenologic examination was performed at that time.

Physical examination was unremarkable with the exception of moderate left costovertebral angle tenderness. Urinalysis revealed gross hematuria. Urine culture was negative.

An intravenous pyelogram demonstrated a normal right kidney, but on the left there was distortion and effacement of the calices in the region of the superior and lateral portion of the kidney. The remainder of the pyelogram was within normal limits. Because of these findings arteriography was performed.

Utilizing the Seldinger technique, abdominal aortography was performed which demonstrated a single artery on the right side and a normal right kidney. On the left there were multiple renal arteries, and along the superior and lateral portion of the left kidney there was a very large mass with considerable neovascularity. Therefore, selective arteriography was performed on the left side. Injection of the superior artery on the left demonstrated absolutely no neovascularity or evidence of the mass (Fig. 1). Selective arteriography performed on the second left renal artery demonstrated a very large mass, approximately 11 cm. in diameter, with considerable neovascularity, some blush, but without evidence of arteriovenous shunting. Demarcation between the parenchyma of the kidney and the mass was extremely sharp (Fig. 2). Because of the neovascularity, an epinephrine study was performed. Epinephrine (10 micrograms) was given intra-arterially, followed by a contrast medium bolus. There was a moderate epineph-

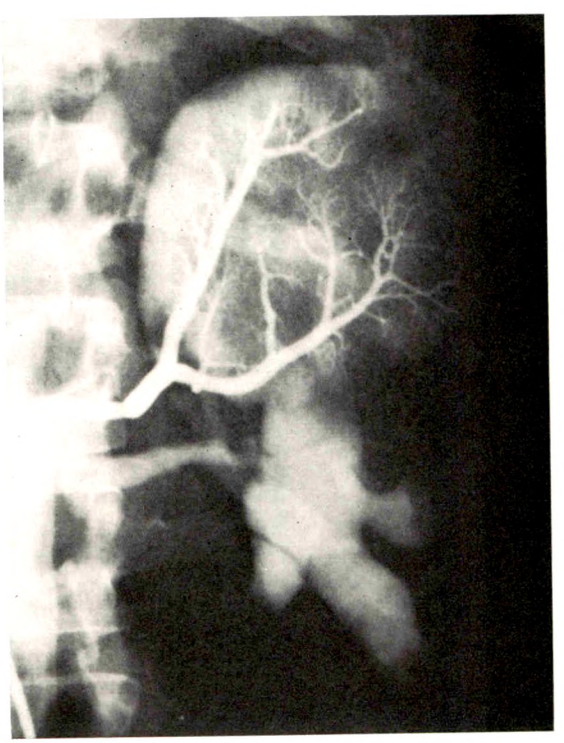


FIG. 1. Selective injection of the first renal artery demonstrates normal vessels and parenchyma without visualization of a mass.

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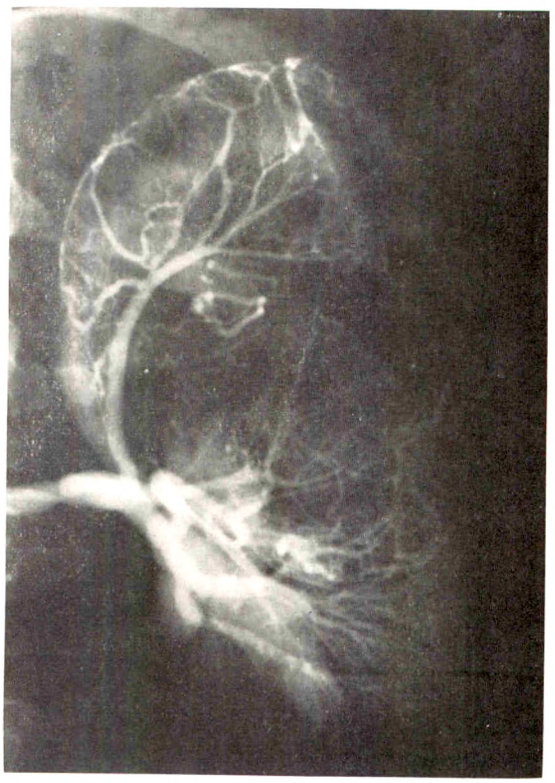


FIG. 2. Selective injection of adjacent renal artery demonstrates an 11 cm. mass with considerable neovascularity and some supply to the adjacent parenchyma. Note the extremely sharp margin between parenchyma and mass.

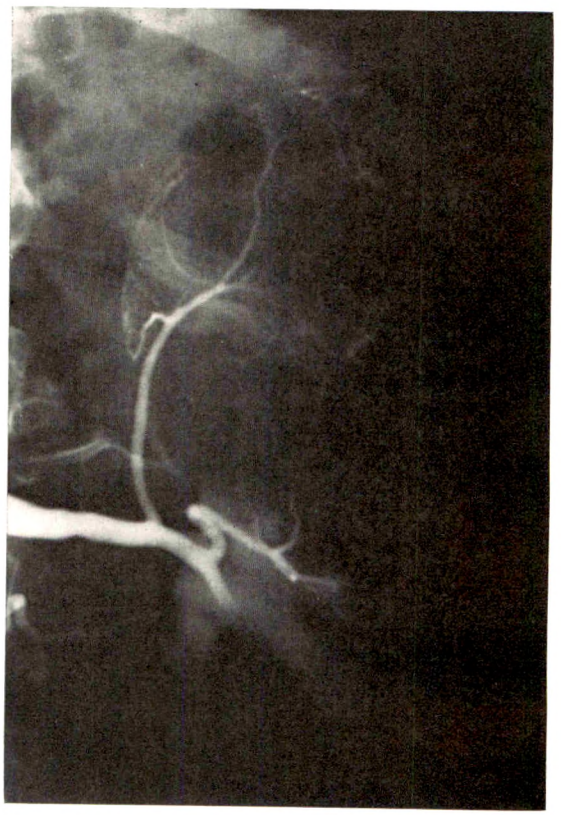


FIG. 3. Post epinephrine arteriogram of the second renal artery shows moderately good epinephrine response of the neovascularity. Some tumor vessels are still seen.

rine response; however, some of the neovascularity was still seen (Fig. 3).

A left total nephrectomy was carried out. In the gross specimen, along the superior and lateral portion of the left kidney, a large neoplasm was found. The tumor had a glistening capsular surface and there was a sharp demarcation of the mass from the normal kidney parenchyma. A section through the tumor showed it to measure 11 cm. in diameter. It was pale tan and somewhat friable (Fig. 4).

The histologic appearance of the tumor was uniform, consisting of fairly large cells with considerable amounts of granular cytoplasm and normal appearing nuclei (Fig. 5). No vascular invasion was seen.

The patient is presently doing well with no evidence of disease 1 year following surgery.

DISCUSSION

The giant renal adenoma is an uncommon lesion and its occurrence has generated several reports.[7,8,19] In the present case, considerable controversy developed because of the benign appearance of the microscopic sections in the face of such a

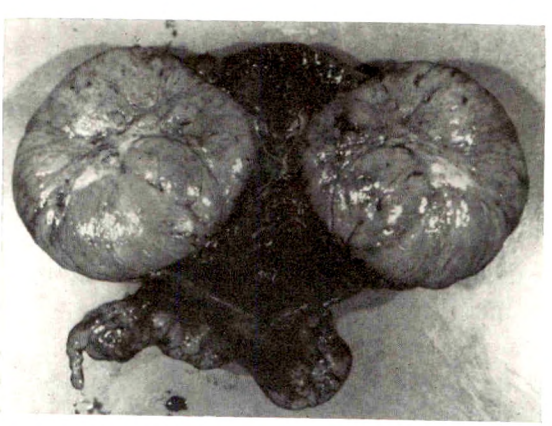


FIG. 4. Gross specimen shows discrete large mass with sharp demarcation between parenchyma and a well-defined capsule.

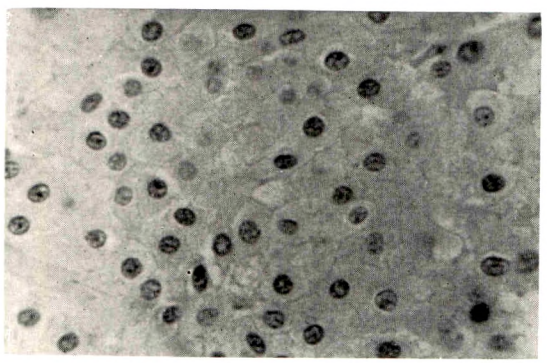


Fig. 5. The histologic pattern is that of a uniform cell type, with each having normal appearing nuclei and considerable amounts of granular cytoplasm.

large renal tumor. Several pathologists were hesitant to make a diagnosis of benign lesion, largely due to the size of the neoplasm. This situation can be attributed to the criterion for malignancy stated by Bell,[3] "I have arbitrarily classified all tumors of this group with the diameter of less than 3 cm. as adenomas." This statement has been cited repeatedly and can be found in similar form in current leading pathology textbooks. Anderson,[2] in his sixth edition of *Pathology*, states, "Some have arbitrarily classified all tumors of this type that are less than 3 cm. in diameter as adenomas." In Robbin's[13] *Pathology* the following statement can be found: "All tumors over 3 cm. in diameter are considered by some as malignant." In a very recent publication by Evans and Bosniak[6] it is stated: "Most pathologists believe that the gross size determined whether a particular lesion should be benign or malignant, using 3 cm. diameter as the critical measurement." The impact of these statements has not been felt as greatly in the foreign language literature. Wagner *et al.*[18] have recently spoken of this point and conclude that tumor diameter is not a valid criterion for establishing the diagnosis of a benign or malignant lesion.

One of the purposes of this paper is to reassess the value of tumor size in the establishment of diagnosis; for, except in the case of renal adenoma, tumor size is rarely used in the field of pathology for the purpose of differentiating benign and malignant lesions. The more conventional criteria for malignancy such as cellular appearance, blood vessel, and/or lymphatic invasion, capsular extension, parenchymal invasion, extension into surrounding renal tissues, and metastases would seem to be more important.

Once the mass has been identified by routine roentgenographic techniques, or by contrast medium study such as intravenous pyelography, much more significant preoperative information can be obtained by nephrotomography and angiography.[8,19] Although not utilized in this case, a chlormerodrin scan can be helpful.[4] The addition of pharmaco-angiography gave additional information; *i.e.*, moderate response of the neovascularity to the intra-arterial epinephrine, which suggested a benign etiology of the mass lesion.[1,8] Although variable responses to epinephrine have been described in benign lesions such as inflammatory disease, angiomyolipomas,[9] lipomas, cysts, and others, a partial response should at least suggest a benign lesion, as should lack of arteriovenous shunting. Interestingly, Grazi *et al.*[7] have recently reported a similar case in which the preoperative diagnosis of adenoma was suggested on the basis of the angiographic findings. Fortunately, they also were dealing with a kidney having multiple renal arteries, and the blood supply of the mass showed no parasitation from the other arteries.

SUMMARY

Giant renal adenoma is a relatively uncommon lesion which may be difficult to diagnose correctly if the criterion of size is given undue emphasis. Rather, cellular appearance, histologic morphology, and presence or lack of blood vessel and/or lymphatic invasion, capsular extension, and retroperitoneal invasion should be strongly emphasized.

A benign lesion such as adenoma should be considered preoperatively when there is a partial or complete response of neovascularity to epinephrine, lack of parasita-

tion of vascular supply from an adjacent renal artery (when present), no demonstration of tumor vessels beyond the margin of the mass lesion, and lack of arteriovenous shunting.

If the possibility of a benign lesion can be raised prior to surgery, a less radical approach to the lesion can be planned.

Ultimately, the clinical course of the patient will judge both the pathologic and roentgenologic diagnoses.

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ROENTGENOGRAPHIC FEATURES OF PRIMARY RENAL CANDIDIASIS*

FUNGUS BALL OF THE RENAL PELVIS AND URETER

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CANDIDA, most commonly a saprophytic fungus found in the digestive tract and vagina, will under certain circumstances become pathogenic.[3,13] When host resistance is altered due to malignancy, diabetes, antibiotic therapy, steroid therapy, chronic disease or a combination of these factors, *Candida* may become an invader and produce one of two clinical syndromes:[6,10,16]

1. *Systemic candidiasis.* The organisms may produce disseminated infection with a resultant fulminating course. Multiple organ involvement in addition to candidemia is found and death is usually due to renal failure.[14]

2. *Primary renal candidiasis.* The second and less serious clinical picture is produced by primary kidney invasion without other organs being affected and without evidence of candidemia.[20]

To date, there have been 12 reported cases of primary renal candidiasis with the emphasis being on the pathogenesis and/or treatment (Table 1).

The purpose of this communication is to review the previous cases and add 3 additional cases, stressing the radiologist's role in the recognition of this entity. Certain roentgenographic features when coupled with the clinical history will allow the radiologist to suggest primary renal candidiasis earlier than most cases have heretofore been diagnosed.

REPORT OF CASES

CASE I (Fig. 1, *A* and *B*). J.W., a 17 year old white male with a 10 year history of severe diabetes, was involved in an automobile accident and sustained cervical spinal cord trauma resulting in quadriplegia 5 months prior to admission. One month prior to admission he suffered an episode of ketoacidosis which necessitated hospitalization. A urinary tract infection, found at that time, was treated with several drugs including chloromycetin and gantanol. After discharge from the hospital he continued on prophylactic antibiotic treatment.

His final admission was to the Emory University Hospital in December, 1964. At that time his diabetes was out of control and again there was evidence of urinary tract infection. He was given keflin and chloramphenicol with no apparent improvement in his clinical condition. On the eleventh hospital day, excretory urography revealed a large left kidney with delayed function and poor concentration. There was no visualization of the right kidney draining structures. A left retrograde pyelogram demonstrated marked hydronephrosis with a large irregular filling defect in the renal pelvis, extending into the infundibula and calyces.

Bilateral nephrostomies were performed and the material removed from the left renal pelvis was noted to be yellowish-brown in color and "putty-like." Histologic examination of this material demonstrated the entire mass to be a fungus ball composed of *Candida albicans*. A perinephric abscess with perforation on the right side was found.

Following surgery the patient was given amphotericin B which resulted in a brief period of improvement, and then a gradual downhill course and death 2 months after his final admission.

CASE II (Fig. 2, *A–C*). C.G., a 59 year old female diabetic, was admitted to Crouse-Irving Memorial Hospital in April of 1969 with a 5 day history of right flank pain and weakness.

Four days prior to admission the patient was noted to have fever ranging from 101° to 103°F. and was seen in the Emergency Room of another hospital, where a diagnosis of pyelo-

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TABLE I

SUMMARY OF CASES REPORTED IN THE LITERATURE

Case No.	Author	Age and Sex	Predisposing Factors	Symptoms	Roentgenographic Findings		Comments
					Intravenous Pyelogram	Retrograde Pyelogram	
1	Gilliam and Vest[8]	56 M	Previous renal trauma	Right renal colic	Right calyceal blunting and hydronephrosis	———	Fungus balls passed per urethra
2	Taylor and Rundle[19]	56 F	———	Abdominal pain and fever	———	———	Fungus balls (bilateral) at autopsy
3	Albers[1]	28 F	Mitral valvular disease	Flank pain	———	———	Fungus ball formation proven at surgery
4	Albers[1]	53 F	———	Flank pain and vomiting	———	Filling defects in calyx, left hydronephrosis	Fungus ball passed without retrograde study
5	Raphael and Badgery[18]	42 F	Mitral valvular disease	———	———	———	Autopsy—fungus ball in right renal pelvis
6	Gillam and Wadelton[7]	51 F	———	Flank pain and fever	———	Filling defects in left renal pelvis and calyces	Fungus ball at cystoscopy and surgery
7	Cowan *et al.*[5]	41 F	———	Flank pain and fever	Left hydronephrosis and calyceal irregularity	Filling defects in left renal pelvis and calyces	Fungus ball passed per urethra
8	Blum[2]	28 M	———	Flank pain and vomiting	Nonvisualization on right	Filling defect in right ureter with obstruction	Fungus material irrigated from right ureter
9	Price *et al.*[17]	32 F	———	Flank pain and abdominal mass	Left ureteral obstruction with hydronephrosis; ? renal mass	———	———
10	Price *et al.*[17]	57 F	Diabetes	Dysuria	———	———	Thirty fungus balls passed per urethra
11	Tennant *et al.*[20]	16 M	Diabetes	Back pain and fever	———	Filling defect in left lower calyx	Fungus ball in renal pelvis at surgery
12	Tennant *et al.*[20]	18 F	Diabetes	Flank pain, dysuria and fever	Poor visualization right kidney	Right caliectasis and hydronephrosis	———
13	Gerle	17 M	Quadriplegia and diabetes	Fever	Nonvisualization on right; poor visualization on left	Large filling defect in left renal pelvis and calyces	Fungus ball at surgery
14	Gerle	59 F	Diabetes	Dysuria, right flank pain and weakness	Poor function on right with hydroureter	Filling defects in calyces and right ureter	Fungus ball at cystoscopy
15	Gerle	45 F	Diabetes	Right flank pain, "passing tissue"	Filling defects right renal pelvis	Confirmed and extending into right calyces	Fungus balls passed per urethra

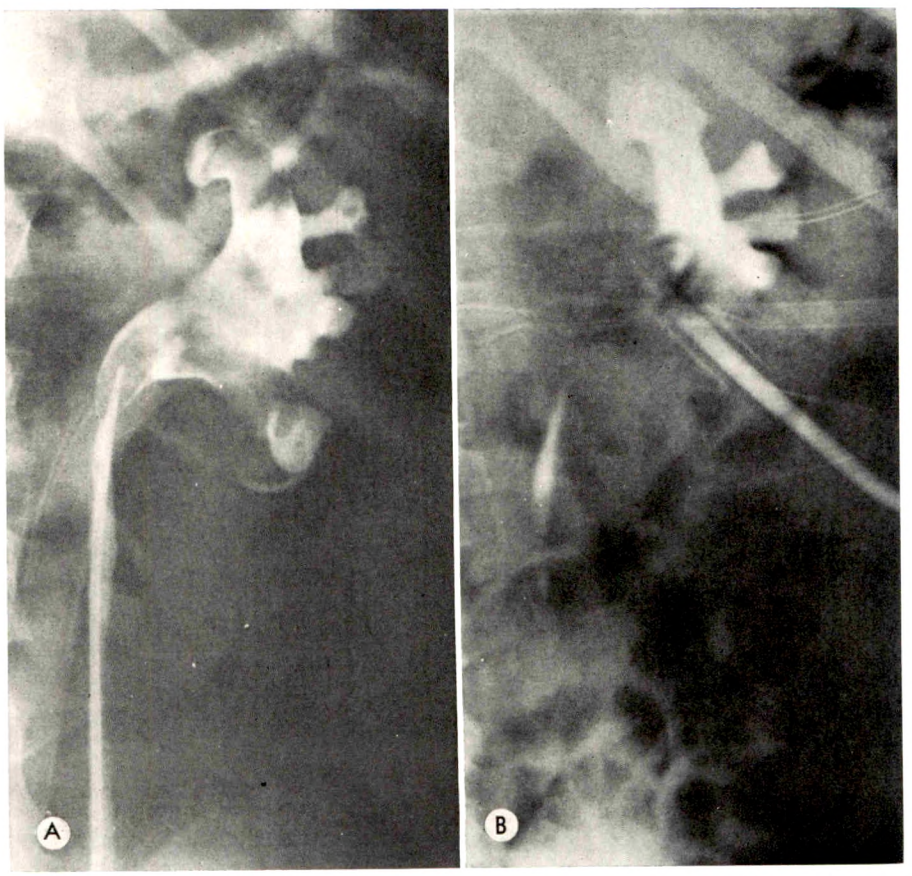


Fig. 1. Case 1. (*A*) Left retrograde study demonstrating a large irregular filling defect in the renal pelvis and extending into the calyces. (*B*) Left nephrostomy contrast study after removal of "putty-like" material proven to be "fungus ball" and composed of *Candida albicans*.

nephritis was made and antibiotics were given.

She failed to respond to the antibiotic regimen and because of increased fever and weakness was hospitalized. A diagnosis of acute pyelonephritis superimposed on chronic renal disease was made. Her blood urea nitrogen was elevated to 90 mg. per cent and on physical examination there was a questionable mass in the right upper quadrant.

Roentgenographic examination of the abdomen demonstrated the presence of gallstones and empyema of the gallbladder was then suspected.

Exploration on the second hospital day revealed cholelithiasis without evidence of cholecystitis.

Following surgery the patient's hospital course deteriorated with her diabetes remaining in poor control and her blood urea nitrogen continuing to rise. Blood cultures revealed *Aerobacter bacillus* and treatment for septicemia and acute pyelonephritis with chloromycetin was instituted. The patient's condition immediately improved with a fall in the blood urea nitrogen and a return of blood cultures to normal. Now for the first time a urine culture demonstrated an overgrowth of *Candida albicans* and several subsequent cultures confirmed this finding. Excretory urography showed the left kidney to be normal with marked delay in visualization on the right and a dilated right ureter. Because of the patient's history of diabetes and infection treated with several antibiotics, plus the roentgen findings of obstructive uropathy, fungus infection with fungus ball formation was suggested by the radiologist.

Cystoscopy revealed thick purulent brown material coming from the right ureteral orifice. Right retrograde pyelography showed marked dilatation of, and a filling defect within, the distal ureter, plus several filling defects in the dilated renal calyces. Cultures of aspirated

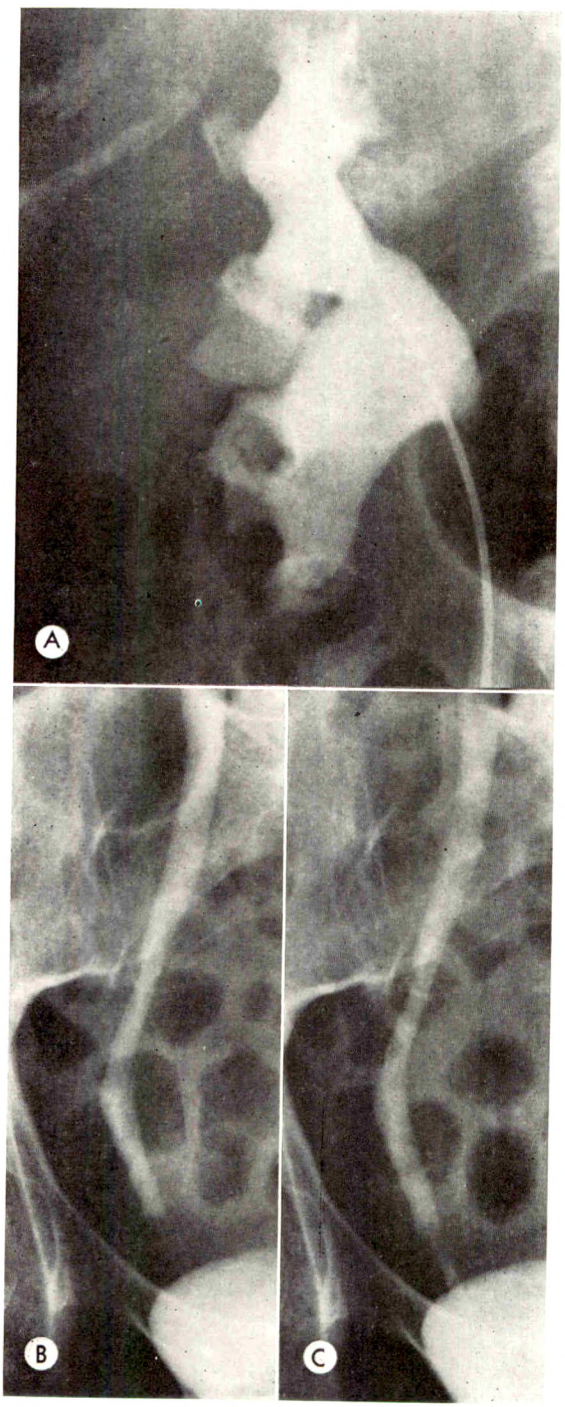


FIG. 2. Case II. (*A*) Right retrograde study demonstrates irregular filling defects in the enlarged calyces and moderate hydronephrosis. (*B* and *C*) The filling defect in the distal ureter was removed and found to be a small "fungus ball" as was the material removed from the kidney.

material from the ureter and renal pelvis revealed *Candida albicans*. The catheter was left in place and amphotericin B irrigations were carried out daily. Repeat retrograde studies done 2 weeks later showed a marked decrease in the size of the calyceal filling defects. The ureter appeared normal.

The patient was discharged 2 months after admission with her diabetes under control and no evidence of fungus infection of the urinary tract.

CASE III (Fig. 3, *A–C*). M.K., a 45 year old diabetic female was admitted to Crouse-Irving Memorial Hospital in April of 1970 because of intermittent right renal colic of 48 hours' duration. She stated that during this time she had passed small amounts of "kidney tissue." She had experienced a similar episode 1 month previously including passage of "abnormal tissue." The symptoms at that time had subsided after 3 days with no antibiotic treatment. Immediately prior to these 2 episodes she had had multiple bouts of pyelonephritis treated with various antibiotics.

After hospital admission the right colicky-type pain persisted and urine cultures taken on admission revealed a "nonpathogenic" yeast. A catheterized specimen revealed a pure growth of "yeast." Some "pieces of tissue" which had been passed by the patient were sent for histologic examination. The initial impression was "possible anaplastic carcinoma." However, on closer study of this and additional material, organized areas of inflammatory cells mixed with fungus were demonstrated.

Excretory urography demonstrated the suggestion of enlargement of the middle and lower portion of the right kidney together with faint and peculiar appearing calcifications in the region of the right renal pelvis. Later roentgenograms demonstrated a probable filling defect in the same area.

Because of the patient's history of diabetes and urinary tract infections having been treated with many antibiotics, plus the known passage of material thought to represent a "fungus ball," right retrograde pyelography was performed. This confirmed a large right renal pelvis filling defect which extended toward many of the calyces.

An indwelling catheter was inserted and amphotericin B irrigations were carried out daily. The culture of urine from the ureteral catheter

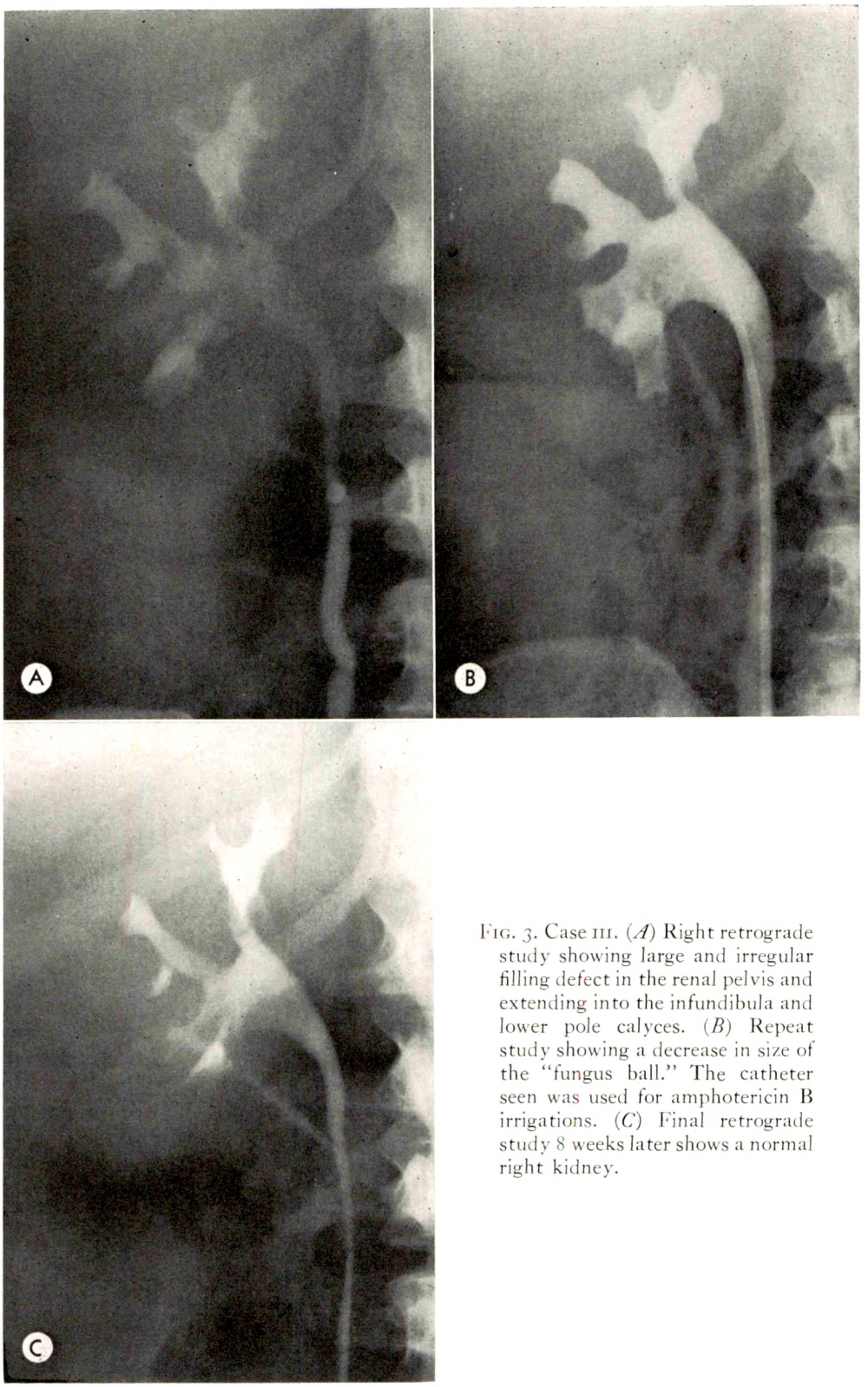


Fig. 3. Case III. (*A*) Right retrograde study showing large and irregular filling defect in the renal pelvis and extending into the infundibula and lower pole calyces. (*B*) Repeat study showing a decrease in size of the "fungus ball." The catheter seen was used for amphotericin B irrigations. (*C*) Final retrograde study 8 weeks later shows a normal right kidney.

taken 5 days later demonstrated *Proteus*, but no evidence of fungus. Approximately 2 weeks later the ureteral instillation of amphotericin B was stopped, and repeat retrograde studies demonstrated a definite decrease in the size of the pelvic "fungus ball."

Nine weeks later the patient was discharged asymptomatic with her diabetes under good control.

DISCUSSION

HISTORIC REVIEW

Berg, in 1846, established the fact that fungus can be responsible for pathologic alteration of tissue and demonstrated that oral thrush was caused by fungus now known to be *Candida albicans*.[11] Shortly after this Virchow demonstrated the invasiveness of this fungus by its presence in the submucosal layer of the esophagus.[12] It has since been learned that *Candida albicans* is not the only pathogen of the *Candida* species. Given the right milieu, almost all of the *Candida* species can become pathogenic. In 1931 Lundquist called attention to what he termed "mycosis renalis" or primary fungal infection of the kidney. He cited 17 cases in the world literature including 11 cases of actinomytosis, 4 cases of candidiasis and an isolated case each of sporotrichosis and cryptococcosis.[15]

PATHOGENESIS OF URINARY TRACT FUNGAL INFECTION

Involvement of the urinary tract by fungus and more specifically the *Candida* species occurs in 3 ways.

(1) *Candida cystitis:* Fungal infection of the bladder alone is much more common than primary renal candidiasis, almost always occurring in females with associated vaginal moniliasis. Some authors believe that renal involvement results from ascending infection starting in the bladder.[20] Fungus ball formation confined to the bladder, producing a large intravesicular mass and demonstrating distinctive roentgenographic features, has been reported.[4] However, it should be pointed out that there has been no clinical or investigative proof that renal candidiasis is a result of an initial bout of monilial cystitis.

(2) *Systemic candidiasis:* Systemic candidiasis occurs in the debilitated host. Disease takes the form of very rapidly progressing infection with most major organs being involved. The kidneys are diffusely involved in 85 per cent of the patients. Candidemia is present initially and throughout the entire course of the disease with death resulting from diffuse pulmonary involvement in addition to the renal failure which is usually found in the end stages of this disease.[11,14]

(3) *Primary renal candidiasis:* This form of renal fungus infection is defined as renal invasion by the *Candida* organism, without associated blood stream or other organ involvement. As a result of investigative work by Hurley and Winner in 1963, there is good experimental evidence that primary renal candidiasis is the end stage of an initial mild candidemia.[12] By utilizing white mice, these investigators have produced a picture virtually identical to the second and third clinical forms of candidiasis as outlined above. Thus, when large doses of *Candida* were rejected intravenously in mice, a fulminating form of candidiasis resulted with most major organs involved. However, with smaller doses of inoculum the findings were limited to the kidneys. Mice from this latter group, when sacrificed early, were shown to have kidney lesions consisting of cortical, renal, tubular and interstitial abscesses. When sacrificing mice from this same group at a later date, the pathologic changes found in the kidneys were those of necrotizing papillitis as well as fungus balls located in the calyces, renal pelvis and ureters. In this latter stage, many of the parenchymal lesions were already noted to have healed. Based on this investigative work it would appear possible that primary renal candidiasis and fungus ball formation occurring in the renal pelvis and ureter as seen in the human may be part of a clinical state representing the final stage of an initially mild candidemia.

DIAGNOSIS (The Radiologist's Role)

In previous communications on the subject of primary renal candidiasis no mention is made of the radiologist's role in

helping to establish an earlier diagnosis. The importance of such a diagnosis is that surgery may be avoided for what has been mistakenly diagnosed as tumor within the renal pelvis. Earlier attention may then be given to the now accepted treatment of this condition with systemic fungicidal medication plus local instillation of fungicidal solution *via* a ureteral catheter.[9,17] Early aggressive therapy of this type often results in dissolution of the fungus material with relief of obstruction and return in many cases to normal function.

It cannot be overly emphasized that part of the radiologist's role is also to recognize the importance of the clinical history. In many of the previously reported cases, as well as in the 3 just presented, there are certain clinical similarities that should alert the radiologist to include fungus ball high in his list of differential diagnoses of filling defects of the renal pelvis and ureter.

These similarities are as follows: (a) history of diabetes or debilitating illness associated with recent urinary tract infections treated with various antibiotics; (b) history of improving for a short time before developing new signs and symptoms of urinary tract infection despite antibiotics; (c) history in some instances of "passing tissue" per urethra; and (d) history of no hematuria.

One further part of the radiologist's role is follow-up retrograde pyelography once the diagnosis of fungus ball has been established. Done at regular intervals, this will demonstrate dissolution of the fungus ball. In 2 of our cases (Cases II and III), pyelography was accomplished under fluoroscopic control with spot roentgenograms which proved a very simple and satisfactory method of carrying out this examination.

ROENTGENOGRAPHIC FEATURES OF PRIMARY RENAL CANDIDIASIS

When the conventional intravenous urography is carried out, unilateral or bilateral nonvisualization may be noted and erroneously ascribed to nonspecific infection or poor renal function and nothing further suggested in the form of a roentgenologic investigation. If a diagnosis of primary renal candidiasis is to be made in this group of patients, additional studies will have to be recommended and should include flooding intravenous pyelography with tomography for better visualization of the drainage structures. If obstructive uropathy is then suggested, retrograde pyelography should be carried out to conclusively demonstrate the presence of filling defects within the renal pelvis, calyces and ureter and thus indicate the presence of fungus ball formation.

As can be seen in Table 1, in 4 of the previously reported cases no mention is made of roentgenologic findings. Of the remaining 11 cases, intravenous urography was performed in 8 and in all but 1 of these cases unilateral involvement, with the opposite side appearing normal, was found. The right kidney was affected in 5 cases, with the left being involved in the other 3. Bilateral involvement was present in 1 case. The roentgenographic abnormality demonstrated in the affected kidney was poor or no visualization in 4 cases, hydronephrosis with distorted calyces in 3 cases, and actual filling defects caused by the fungus ball in only 1 case.

A total of 9 patients had retrograde pyelography performed. In 3 patients in whom intravenous urography did not demonstrate sufficient visualization, the retrograde study proved conclusively the presence of abnormal filling defects representing fungus ball formation. Retrograde studies were performed in 4 patients in whom intravenous pyelography had suggested definite abnormality, and all of these cases conclusively demonstrated the presence of fungus ball formation in the renal calyces, pelvis or ureter, although the diagnosis was suggested by the radiologist in only 2 of these cases. The retrograde study was nondiagnostic in only 1 patient. Retrograde pyelography was the sole study in 2 cases and both demonstrated multiple filling defects in the calyces and pelvis. None of the patients studied had renal angiography and therefore the vascular

changes which might be associated with this entity cannot be commented upon.

SUMMARY

Three cases of primary renal candidiasis (fungus ball of the renal pelvis and ureter) are reported and the previous cases in the literature are reviewed.

The characteristic clinical findings and the roentgenographic features are stressed.

It is hoped that radiologists will become more aware of this entity which in turn will lead to an earlier diagnosis and prompt definitive treatment.

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ANGIOGRAPHY IN GLOMERULAR DISEASE OF THE KIDNEY*

A CORRELATION WITH CLINICAL FINDINGS AND THE STAGE OF THE DISEASE

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GLOMERULONEPHRITIS is, next to pyelonephritis, the most frequent renal disease. Very few investigators, however, have discussed the value of angiography in the diagnosis of glomerulonephritis. In hitherto published reports the angiographic appearance has not been correlated with different stages, or clinical findings of glomerulonephritis;[7] the number of cases has been small; or the technique used for angiography has been nonselective and thus inadequate for demonstration of small vessel detail.[6] Furthermore, recent advances in the treatment of glomerulonephritis with immunosuppressive agents have necessitated a more exact diagnosis of the disease.[2]

We have, therefore, analyzed the angiographic findings in 66 patients with histologically verified glomerular disease of the kidney to determine the diagnostic and differential diagnostic capability of selective renal angiography in glomerulonephritis.

It is obvious that abnormalities in any disease will become more marked with its progression and a mere description of angiographic findings in glomerulonephritis would be of very little value if not correlated with the stage of the disease. In the present paper we have, therefore, also analyzed the angiographic findings in 4 different stages of glomerulonephritis, as classified with regard to the clinical course and renal function, hypertension, hematuria and histology.

MATERIAL AND METHOD

The age and sex of the 66 patients included in the present study are shown in Figure 1. The average age of the patients was 30 years. All patients had the clinical diagnosis of glomerulonephritis. In 2 patients the final histologic diagnosis, however, was: systemic lupus erythematosus; and amyloidosis of the kidney; periarteritis nodosa and Wegener's granulomatosis each in 1 patient.

All patients were examined with transfemoral bilateral, selective renal angiography. Eight to 10 ml. 60% metrizoate (Isopaque coronar, Nyegaard, Oslo) was injected through a red thin-walled catheter (I.D. 1.45, O.D. 2.2 mm.) at a rate of 8–10 ml./sec. Serial roentgenography of every kidney was performed at least in 2 projections with Elema-Schoenander cut film changer. The program for filming was usually 2 films/sec. for 2 seconds, followed by a fifth and a sixth film at 2 second intervals. In several cases 12 films were obtained at the rate of 3 films/sec. for 2 seconds, and 1 film/sec. for a further 6 seconds for better evaluation of the renal circulation time, nephrographic phase and of the renal vein. The nominal focal spot size was 1 mm. The focal spot-patient-film distances resulted in a geometric magnification of approximately 1.25–1.3 times.

All angiographic roentgenograms were reviewed by 2 of us (L. E. and J. K.) without knowledge of the clinical or histologic diagnosis. The following criteria were

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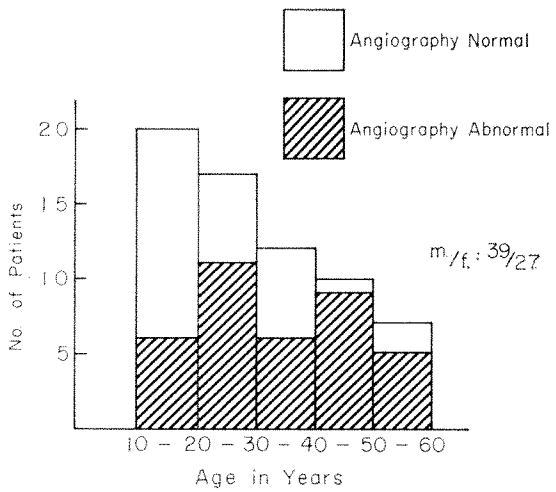


FIG. 1. Age and sex distribution in 66 patients with glomerulonephritis examined with selective renal angiography.

registered as essential for angiographic diagnosis of glomerular disease: kidney size, caliber of the main renal artery; appearance and filling of interlobar, arcuate and interlobular arteries; concentration of contrast medium in the kidney during the nephrographic phase; width of the cortex and definition of the cortical-medullary junction; renal circulation time and filling of the renal vein.

In evaluation of these criteria the kidney size was regarded as normal when its length was 11.5–14 cm., and width 5–7 cm.;[12] the diameter of the main renal artery 5–9 mm.[4] and the width of the cortex 6–8 mm. (According to textbooks of anatomy the width of the normal cortex is 5–7 mm. Gill and Pudvan[7] regarded 6 mm. as normal. We have taken into consideration also the geometric magnification at filming.) Arterial emptying time was considered normal when less than 1.5–2 seconds, and filling of the renal vein was expected 4–5 seconds following injection of the contrast medium.

From the clinical point of view the patients were divided into 4 groups as shown in Table 1. In determination of the renal function an endogenous creatinine clearance of 80 ml./min. (24 hours sampling period) was regarded as the lower limit of normal. Eight patients with acute glomerulonephritis had endogenous creatinine clearance between 2 and 105 ml./min. Forty-three patients had chronic glomerulonephritis but normal kidney function (clearance $\geq$ 80 ml./min.). Thirteen patients had chronic glomerulonephritis with reduced kidney function (clearance between

TABLE I

STAGING OF PATIENTS AND ANGIOGRAPHIC FINDINGS IN 66 CASES OF HISTOLOGICALLY VERIFIED GLOMERULAR DISEASE OF THE KIDNEY

Stage	No. of Patients	No. of Abnormal Angiograms	Kidney Size	Type of Pathologic Findings Renal Artery			Cortical Width	Cortical-medullary Junction
				Main Stem	Interlobar Arteries	Arcuate and Interlobular Arteries		
Acute	8	6	Increased 5	Dilated 5	Dilated 6	Incomplete filling 5	Increased 3	Indistinct 5
Chronic (clearance $\geq$ 80 ml./min.)	43	18	Increased 1	Normal 43	Dilated, Tortuous 15	Incomplete filling 16	Reduced 2	Indistinct 16
Chronic (clearance 51–79 ml./min.)	13	11	Reduced 2	Normal 12 (fibromuscular hyperplasia) 1	Dilated, Tortuous 8 Narrowed (amyloidosis) 1	Incomplete filling 9	Reduced 3	Indistinct 9
Advanced chronic (clearance $\leq$ 50 ml./min.)	2	2	Reduced 1	Narrowed 1	Narrowed 2	Incomplete filling 2	Reduced 2	Indistinct 2
Total	66	37						

50 and 80 ml./min.) and in 2 patients with advanced chronic glomerulonephritis the endogenous creatinine clearance was 50 ml./min. or less.

Histologic diagnosis was obtained by means of percutaneous needle biopsy, usually not more than 30 days prior or post angiography.

RESULTS

In 29 patients the angiographic findings were within the limits of the criteria regarded as normal. In 37 patients some pathology at angiography was found. The incidence of pathologic angiograms was higher and the changes were more marked in patients with reduced kidney function. The average age of patients with pathologic angiography was higher than the age of patients with normal angiography (33.6 or 25.5 years, respectively).

As the frequency and the type of pathologic changes varied with the stage of glomerulonephritis, the findings will be discussed separately for each clinically and histologically staged group of patients. A case with the findings characteristic for each stage is presented in Figures 4 to 6. Figures 2 and 3 show normal angiography in a young and an aged adult for comparison.

ACUTE GLOMERULONEPHRITIS (Fig. 4, *A–D*)

Six of 8 patients with acute glomerulonephritis had pathologic angiograms. In 5 patients the caliber of the renal artery was wider than 9 mm.; the interlobar arteries were dilated and stretched, and the arcuate and interlobular arteries were incompletely filled, or not filled at all. In these 5 patients the kidneys were enlarged and during the nephrographic phase the cortical-medullary junction was poorly demarcated. The cortex had increased width in 3 patients. The circulation time in the kidney was normal and the renal vein was visualized in all patients. The pathologic changes showed regression following immunosuppressive therapy with corticosteroids and azathioprine.



Fig. 2. Normal selective renal angiogram in a young adult.

CHRONIC GLOMERULONEPHRITIS WITH NORMAL KIDNEY FUNCTION (Fig. 5, *A* and *B*)

Pathologic findings at angiography were present in 18 out of 43 patients, or in 42 per cent. The kidney size remained normal in all cases but 1. The main renal artery had normal width, but the interlobar and arcuate arteries were dilated and tortuous with irregular segmental narrowing in other areas. Filling of arcuate and interlobular arteries was incomplete and the cortical-medullary junction was indistinct in 16 patients. The width of the cortex was reduced in only 2 patients.

CHRONIC GLOMERULONEPHRITIS WITH REDUCED RENAL FUNCTION

In this group, pathologic angiograms were present in 11 out of 13 cases, or in 85 per cent. The kidneys were reduced in size in 2 patients. The vascular and nephrogenic changes were of the same type as in patients with chronic glomerulonephritis with normal renal function, but they were more advanced and more frequently pres-

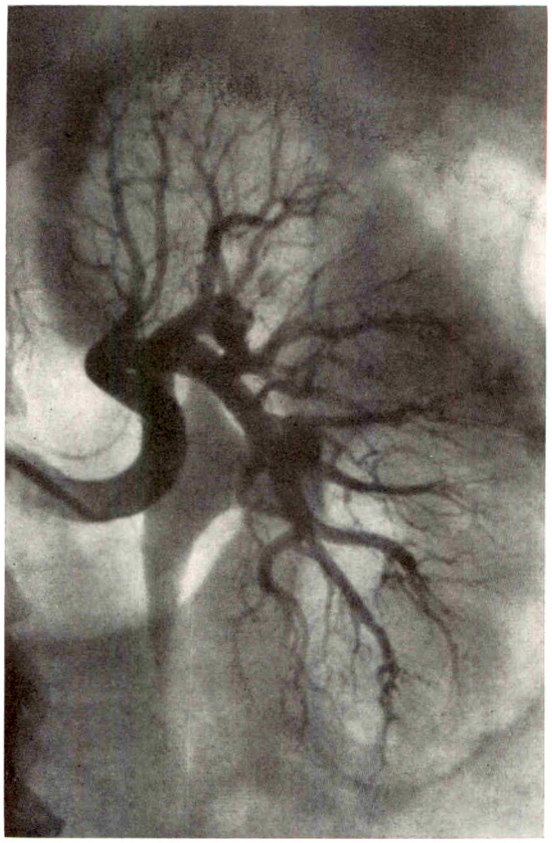


FIG. 3. Selective renal angiogram, normal for aging population (70 year old man).

ent. One patient who—contrary to others—had general narrowing of the interlobar arteries, had amyloidosis. Renal circulation time and filling of renal vein were normal except in 1 patient with associated renal vein thrombosis.

ADVANCED CHRONIC GLOMERULONEPHRITIS (Fig. 6)

Only 2 patients had advanced chronic glomerulonephritis with endogenous creatinine clearance of 50 ml./min. or less. The kidney size and the width of the main renal artery were reduced in 1 patient. The interlobar arteries at this stage were generally narrowed. The arcuate and interlobular arteries were not filled, the width of the cortex was reduced and the cortical-medullary junction was indistinct.

In all stages both kidneys were always affected by the disease, although slight differences in vascular changes in the right and the left kidney occasionally could be found. There was no focal scarring or compensatory parenchymal hyperplasia as in chronic pyelonephritis. The size of both kidneys was approximately equal in 63 of 66 patients. Urography was normal in all patients except in 1 with severely reduced renal function (patient with acute glomerulonephritis, clearance 2 ml./min.).

ELEVATED BLOOD PRESSURE

A blood pressure of 160/100 mm. Hg or higher was present in 4 patients out of 29 who had normal angiography, and in 9 out of 37 patients with pathologic angiographic findings. This difference is statistically not significant.

HEMATURIA

Twenty patients had more than 50 million red blood cells (RBC) in the 12 hour sediment. In 14 of these 20 patients widening and tortuosity of interlobar arteries were present. Forty-six patients had less than 50 million RBC in the 12 hour sediment, and in 17 of these patients changes in interlobar and arcuate arteries, or incomplete filling of interlobular arteries were found ($p < 0.05$, nearly significant difference between the frequencies of vascular changes present in low and high degree hematuria).

RENAL FUNCTION

In 7 patients who had reduced cortical width at angiography, the creatinine clearance was 72 ± 17.5 ml./min. In 56 patients with normal cortex, the creatinine clearance was 105 ± 4.3 ml./min. ($p < 0.02$, almost significant). No statistical correlation was found between vascular abnormalities and renal function.

GLOMERULONEPHRITIS ASSOCIATED WITH RENAL ARTERIOLOSCLEROSIS

A total of 32 patients had incomplete filling of arcuate and interlobular arteries at angiography. Twelve of these patients had histologically glomerulonephritis and vascular changes characteristic for arteriolosclerosis. In only 2 of 34 patients who

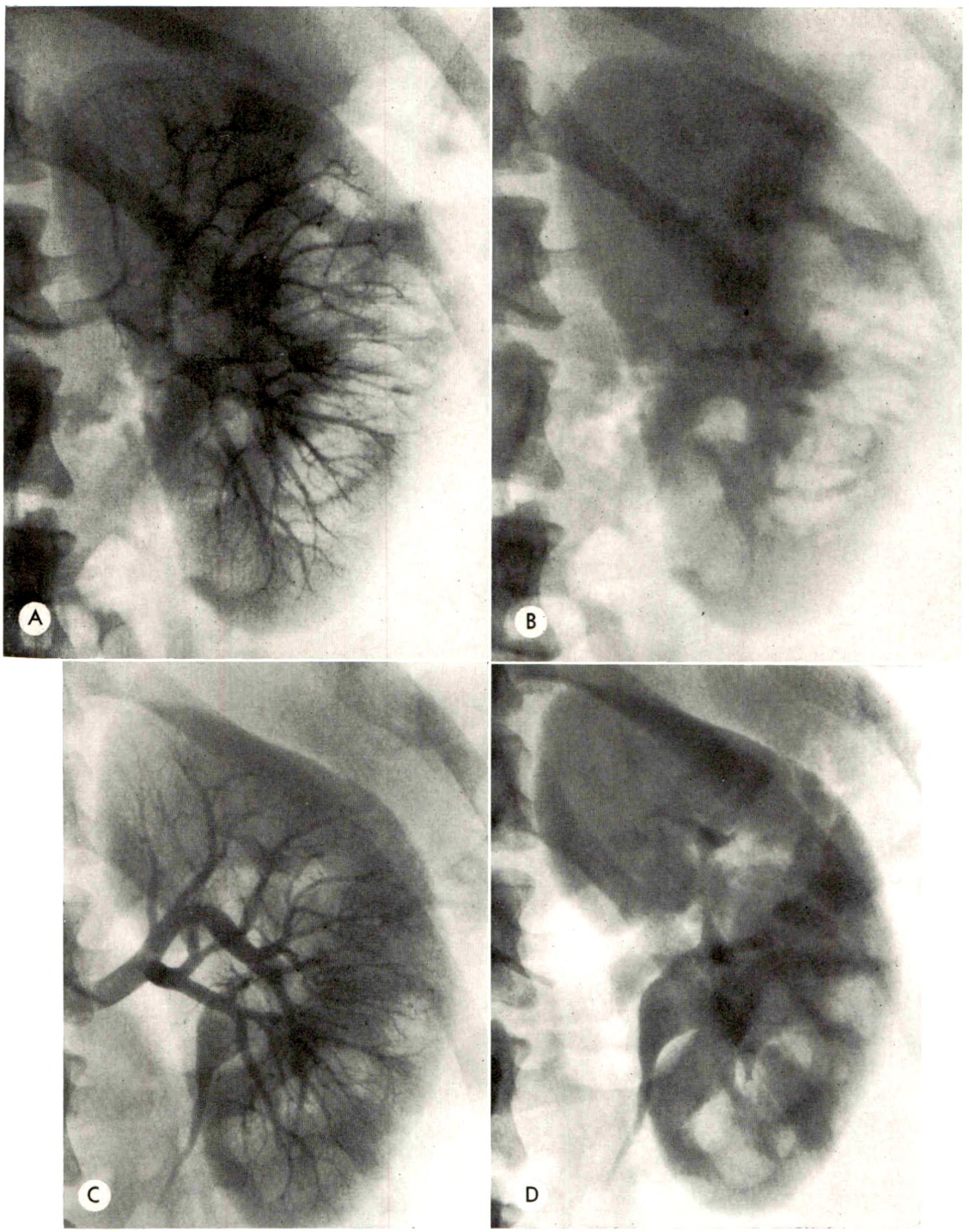


FIG. 4. Angiography in acute glomerulonephritis; 20 year old patient; endogenous creatinine clearance 74 ml./min. Blood pressure 130/80 mm. Hg.

(*A*) Enlargement of the kidney (17×9.5 cm.), stretched arteries with some incomplete filling of arcuate and interlobular vessels. The cortex had normal width in this case. (*B*) During the nephrographic phase the cortical-medullary junction cannot be defined. The renal vein is filled.

(*C* and *D*) One year after treatment with corticosteroids and azathioprine, there is decrease of kidney size (16×8 cm.) and improvement of the nephrogram. (*C*) Arterial and (*D*) nephrographic phase.

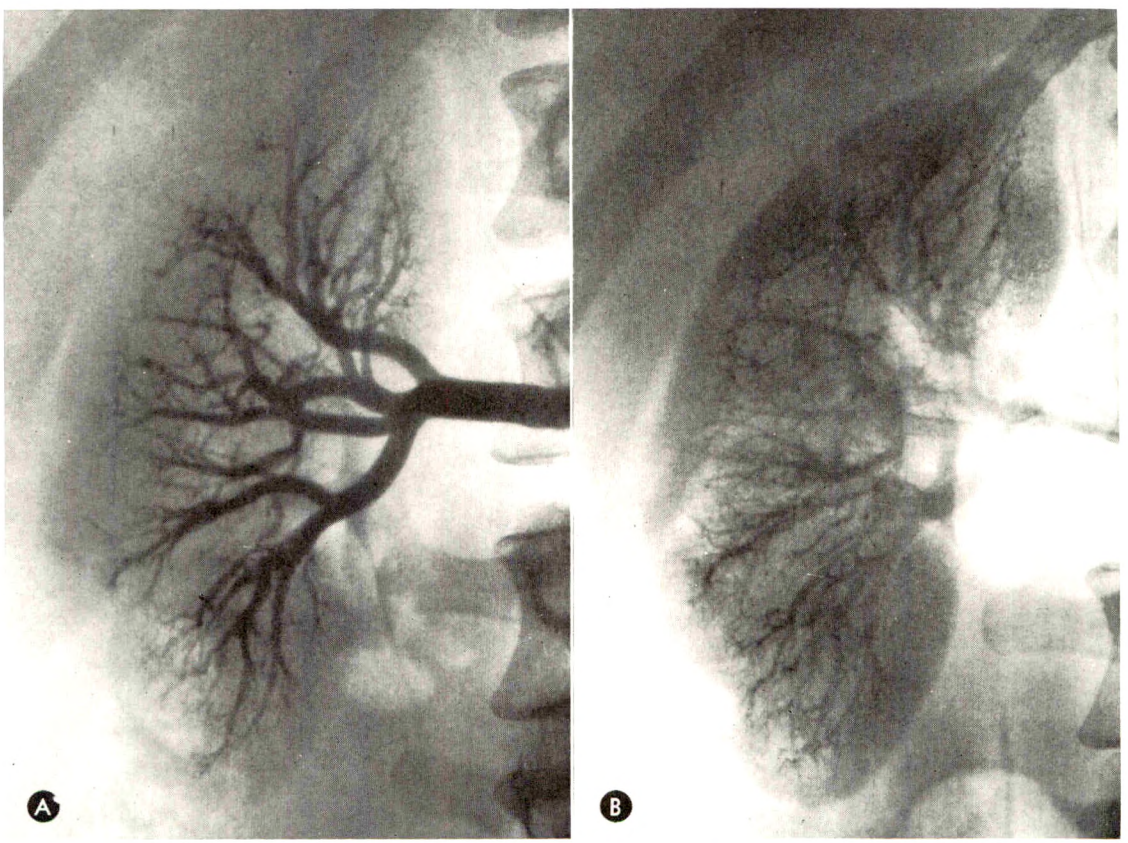


FIG. 5. Selective renal angiography in a 22 year old patient with chronic glomerulonephritis. Microscopic hematuria since 1962, blood pressure 165/110 mm. Hg. Endogenous creatinine clearance 115 ml./min. Histologically, hyalinization of several glomeruli and focal arteriolosclerosis. Renal angiography in 1966 was normal.

Angiography in 1969. (*A*) Early arterial phase. Tortuosity and dilatation of peripheral interlobar arteries, interruption in the course of vessels. (*B*) Late arterial phase. Similar changes in arteries at cortical-medullary junction. Irregular focal narrowing of the arterial lumen laterally in the lower pole. Lack of normal, smooth "tapering" of the arteries. Only focal filling of the cortical arteries. The width of cortex is not reduced.

had normal arcuate and interlobular arteries at angiography, glomerulonephritis was associated with arteriolosclerosis ($p < 0.005$, significant).

DISCUSSION

The diagnosis of glomerulonephritis is clinical. It is supported by a variety of laboratory tests from simple urine analysis to more sophisticated clearance studies. Ultimately, the diagnosis is made by percutaneous renal biopsy. From the diagnostic procedures, urography which is valuable in chronic pyelonephritis, proved to be non-diagnostic in glomerulonephritis. The kidney size can be determined in most cases on preliminary roentgenograms.

In *acute glomerulonephritis* the cortex and kidneys are edematous. No or incomplete filling of cortical vessels is seen. Dilatation of the renal arteries seems to indicate increased flow to the kidney in an attempt to improve cortical perfusion. The effectiveness of immunosuppressive therapy resulting in regression of kidney size, edema of the cortex and normalization of vascular changes can be followed angiographically (Fig. 4, *A–D*). In our series no

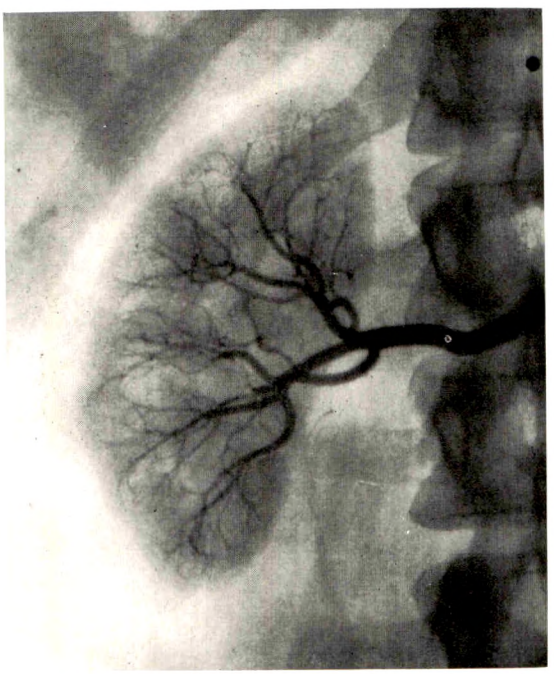


Fig. 6. Selective renal angiogram in advanced chronic glomerulonephritis. A 37 year old female with a microscopic hematuria and proteinuria since 1961. Blood pressure 135/95 mm. Hg. Endogenous creatinine clearance 50 ml./min. Angiography in 1969: Both kidneys were reduced in size (10.5×5 cm.), no local scarring. Narrowing of the main renal artery and interlobar arteries was present. There was lack of normal "tapering" and incomplete filling of arcuate and cortical arteries; the width of the cortex was reduced (early arterial phase). In the nephrographic phase the cortical-medullary junction was indistinct.

patient was in acute renal failure, or showed changes as described by Hollenberg *et al.*[10] Filling of the renal vein excluded venous thrombosis.

In *chronic glomerulonephritis* with normal kidney function, angiographic changes —dilatation and irregularity of the peripheral interlobar arteries, incomplete filling of the arcuate and interlobular arteries with indistinct cortical-medullary junction as a sign of poor perfusion of the cortex— were present in only 42 per cent of cases. These changes became more marked and were more frequent when the renal function was reduced. In advanced stages of chronic glomerulonephritis with severely reduced creatinine clearance the interlobar arteries became narrowed, also probably secondary to increased interstitial fibrosis.

The presence of vascular changes was statistically nearly significant in severe hematuria, but not in elevated blood pressure, or impaired renal function. The reduced cortical width, however, seemed to be a valuable criterion for estimation of renal function.

Vascular changes similar to those found in glomerulonephritis may also be caused by aging (Fig. 3) but they do not occur in a normotensive population under 50 years of age.[3] In our series 86 per cent of patients with pathologic angiographic findings were younger than 50 years of age. There was increased frequency of pathologic angiograms in the higher age groups (Fig. 1), indicating that abnormalities might have been caused by glomerular disease as well as by aging. In microangiographic studies Ljungquist[11] has indeed shown that the number of degeneratively changed glomeruli increases with age, and that in clinically benign hypertension and arteriolosclerosis a higher number of aglomerular arterioles, irregular arterioles and interlobular arteries is present. In malignant hypertension and advanced arteriolosclerosis these changes are more marked and lead to reduction of the cortex as they do in advanced glomerulonephritis. Hollenberg *et al.*[9] have confirmed these *in vitro* observations[8,11] in clinical studies.

Although the primary site of the disease in glomerulonephritis, as opposed to arteriolosclerosis caused by hypertension, is different, angiographic changes in these entities are reflected in more proximal vessels than glomeruli and arterioles. These structures cannot be evaluated with our present angiographic technique. The angiographic differential diagnosis between chronic glomerulonephritis and arteriolosclerosis (with few exceptions, Fig. 8) is impossible not only for radiologic-technical reasons, but also because of the simultaneous presence of both diseases (found in

14 cases in our series). If, however, in a patient with renal disease the arcuate and interlobular arteries have normal appearance, the presence of arteriolosclerosis is unlikely.

Differential diagnosis between chronic glomerulonephritis and pyelonephritis can be made as the criteria for angiographic findings in chronic pyelonephritis have been well established[13] (Fig. 7), and because this disease also reflects from intravenous urography.

Vascular changes in collagen diseases, such as in polyarteritis nodosa, are characterized by local stenotic areas and microaneurysms.[1,5] In our series collagen disease was present in 4 cases, but none of these showed the characteristic angiographic appearance as described in collagen disease.

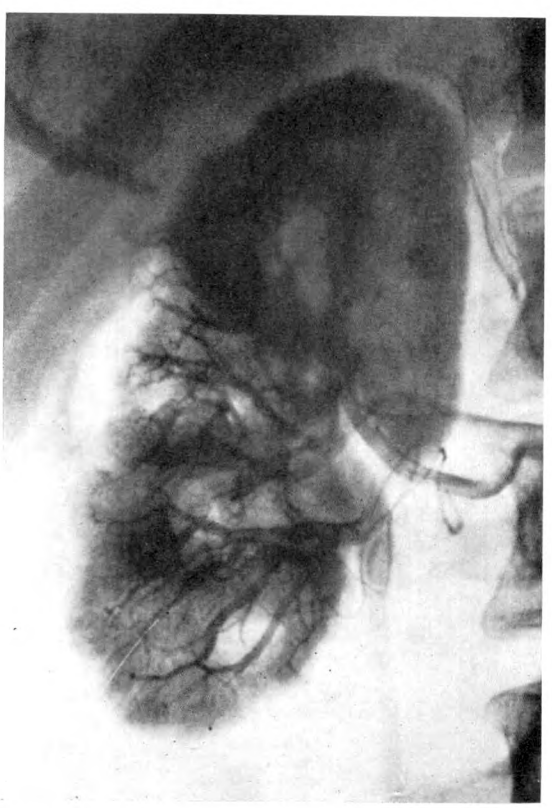


FIG. 7. Chronic pyelonephritis in a 56 year old male. There is a markedly and irregularly reduced renal parenchyma in the middle part and lower pole, while the upper pole of the kidney is less involved by the disease and shows focal compensatory hyperplasia.

Constrictions and dilatations of renal arteries were thought to be caused by a collagen disease in 1 patient, but histologic examination showed only arteriolosclerosis (Fig. 8). Such changes were not found in any case of chronic glomerulonephritis.

SUMMARY

The angiographic findings in 66 patients with histologically proven glomerular disease were analyzed and correlated with the

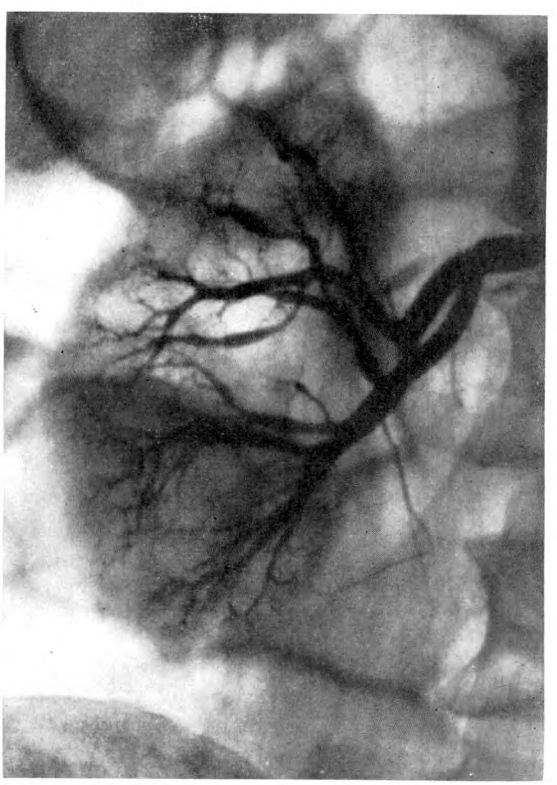


FIG. 8. Selective renal angiogram in a 72 year old male with arteriolo- and arteriosclerosis of both kidneys and reduced renal function. The kidney is decreased in size (10×6 cm.), and the width of the cortex is reduced. The surface is irregular without compensatory parenchymal hyperplasia. Localized stenosis in peripheral and also proximal interlobar arteries, tortuosity and marked irregularity of peripheral branches without filling of arcuate and cortical arteries are present. The changes are different from those in chronic glomerulonephritis, but similar to those which may be found in periarteritis nodosa. The left kidney in this patient had normal size, but showed the same type of arterial changes as the right kidney.

stage of the disease, renal function, blood pressure, and hematuria.

With progression of the glomerular disease, when associated with arteriolosclerosis, and in older patients pathologic angiograms are more frequently present and the vascular and cortical changes are more marked.

The vascular changes are more frequently present also in marked hematuria, while reduced cortex indicates reduced renal function.

Intravenous urography was found to be of no help in the diagnosis of glomerulonephritis.

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RENAL VASCULARITY IN THE DOG—ALPHA-ADRENERGIC BLOCKADE AND ACUTE HEMORRHAGE*

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ACUTE hemorrhagic shock in the dog produces marked changes in the renal angiogram consisting usually of: reduction in caliber of the main renal, segmental, and interlobar arteries as well as of the intrarenal veins and the main renal vein. Renal blood flow is diminished as judged by prolonged circulation time (slowing of the vascular phases) and increased reflux of the contrast medium from the renal artery into the aorta at time of selective angiography. A frequent finding is irregularity of the nephrogram suggesting intrarenal redistribution of blood away from the cortex.[4,7]

Angiograms in dogs made hypotensive by ganglionic block are different from those of hypovolemic hypotension. In a study of Arfonad-induced hypotension the renal angiogram usually showed diminution in caliber of the main renal artery with dilatation of the segmental and interlobar arteries.[3] The main renal and the intrarenal veins remained unchanged in size as compared to the control angiograms. In almost all experiments the nephrogram was smooth in outline with no evidence of shunting of blood away from the cortex.

The renal vascularity thus responds differently to these different modes of hypotension. In Arfonad-induced hypotension the renal vessels do not constrict and there is no evidence of diversion of blood away from the cortex into the medullary circuit. In hypovolemic hypotension there is renal vasoconstriction and redistribution of blood away from the cortex, possibly mediated by increased activity of the sympathetic nervous system. In an attempt to define the role of adrenergic stimulation, the acute hemorrhage experiments have been repeated after alpha-adrenergic blockade.

Phenoxybenzamine hydrochloride (Dibenzyline) effects prolonged blockade of alpha-adrenergic receptors. Vick[10] has reported the beneficial effect of the administration of Dibenzyline to monkeys in endotoxin shock, supposedly by inhibition of the action of excess endogenous catecholamimes and histamine. Hardaway *et al.*[5] indicated that early renal failure in hypovolemic shock is due to inadequate perfusion of the renal capillaries; in patients not responding to fluids alone, phenoxybenzamine effected a dramatic improvement with an increase in blood pressure and urine flow. Kane[6] demonstrated angiographically that Dibenzyline produced renal vasodilatation in 2 monkeys subjected to hemorrhagic shock. Novelli *et al.*[9] reported that hemorrhagic shock produced vasodilatation in the selectively alpha-blocked kidney. Nickerson[8] warned that the administration of Dibenzyline to hypovolemic animals or patients may cause precipitous further fall in blood pressure and respiratory arrest.

METHOD

Selective renal angiography was performed in dogs with Thorotrast as the contrast medium.[2] A red Kifa catheter was passed into the renal artery by the Seldinger technique after percutaneous puncture of a femoral artery. In order to reproduce injection and timing factors in serial angiography, an automatic injector was used, which not only injected the same amount of Thorotrast each time at the

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same rate, but also initiated the roentgenographic exposure at the same time in the course of the injection. The dose of Thorotrast was 0.5 ml. per kg. Direct magnification radiography (2 to 2.5 times magnification) was obtained, using an x-ray tube with high speed rotating anode and an effective focal spot of 0.3 mm. (Dynamax 61). The magnification technique allowed improved study of the smaller intrarenal vessels; however, the x-ray tube limits precluded rapid serial filming. Exposures were made at 1 per second or 2 per three seconds for a total of 6 films in the usual study. Blood pressure was recorded continuously from a catheter in the lower aorta. The dogs were anesthetized with sodium pentobarbital administered intravenously.

Dibenzyline,* 2 mg. per kg. diluted in 200 ml. of saline, was given intravenously on the period of 30 minutes.

Acute hemorrhagic shock was produced by rapid bleeding from the femoral artery into a warmed graduated bottle containing heparin solution. When reinfused, the blood was passed through a fine metal filter to prevent the introduction of clots.

The protocol consisted of the following steps: control angiogram; administration of Dibenzyline; angiogram 30 minutes after completion of the Dibenzyline infusion; bleeding to the desired level of hypotension followed by an angiogram; administration of epinephrine intravenously (15 μg. per kg. in 5 ml. saline) in about 10 seconds, followed by an angiogram 45 seconds later; rapid reinfusion of the blood followed by a final angiogram.

RESULTS

Experiments were done on 5 dogs ranging in weight from 12 to 22 kg. The average control mean blood pressure was 146 mm. Hg—a level not unusual for dogs under sodium pentobarbital anesthesia.[1] Post-hemorrhagic systemic mean blood pressure levels were in the range of 23 to 60 mm. Hg at the time of study by 7 angiograms. Five of the studies were done at a blood pressure level of 44 to 51 mm. Hg; the other 2 were at 23 and 60 mm. Hg. To produce these levels of hypotension by bleeding after alpha-adrenergic blockade, an average of 30 ml. of blood were removed per kg. of body weight. This compares with bleeding of 39 ml. per kg. to produe similar levels of hypotension in another group of dogs without alpha-adrenergic block. This difference is to be expected since the dogs pretreated with Dibenzyline are less able to respond by vasoconstriction during the time of blood volume loss and are thus less able to maintain systemic blood pressure.

EFFECTS OF DIBENZYLINE

Under the conditions of these experiments alpha-adrenergic blockade in itself effected hemodynamic changes. In the control state the animals apparently had a degree of vasoconstriction, very likely produced or exaggerated by the anesthesia. Corcoran and Page[1] had reported that sodium pentobarbital causes an increase in systemic blood pressure with resultant constriction of renal afferent arterioles; it was proposed that these changes may be due to excitation of the sympathetic nervous system. In the 5 dogs the mean blood pressure decreased after Dibenzyline, from an average level of 146 mm. Hg to one of 96 mm. Hg. As evidence of lessening of vasoconstriction after Dibenzyline, the angiograms showed changes as compared with the control (Table 1), chiefly an increase in size of the renal vessels (Fig. 1, *B* and *F*). Using a difference of 10 mm. in either length or width as an indication of change in renal size, the kidney showed no change in 4 animals and an increase in size in the other animal.

HEMORRHAGE AFTER DIBENZYLINE

The changes in the appearance of the renal angiograms after hemorrhage as compared to the pre-bleeding angiograms are summarized in Table II. The main renal

* The Dibenzyline used in this study was kindly supplied by Smith, Kline and French Laboratories, Philadelphia, Pennsylvania.

TABLE I

CHANGES WITH DIBENZYLINE COMPARED WITH CONTROL
(5 dogs)

	Increase	Decrease	Unchanged
Main renal artery	2	1	2
Segmental arteries	5		
Interlobar arteries	5		
Intrarenal veins	2	1	2
Main renal vein			5
Flow			5

TABLE II

CHANGES WITH HEMORRHAGE AFTER DIBENZYLINE
(5 dogs)

	Increase	Decrease	Unchanged
Main renal artery		3	2
Segmental arteries		2	3
Interlobar arteries	2	1	2
Intrarenal veins		3	2
Main renal vein		2	3
Flow		3	2

and segmental arteries remained unchanged or showed a slight decrease in caliber. The interlobar arteries varied in their response, although again they tended to remain unchanged or dilate slightly (Fig. 1*C*). The main renal and intrarenal veins showed no change or a slight decrease in size (Fig. 2*B*). As judged from angiographic criteria, renal blood flow appeared to have diminished only slightly. After hemorrhage the kidney decreased in size in 2 of the 5 dogs. These findings differed from those previously reported[4] in hemorrhage without Dibenzyline pre-treatment, where all renal vessels showed diminution in size and where angiographic criteria indicated marked diminution in renal blood flow. Also, in the unmodified hemorrhage group many of the studies showed evidence of diversion of blood away from the cortex. In the Dibenzyline group only minimal irregularity of the cortical nephrogram was seen in 2 dogs (Fig. 1*G*; and 2*B*); the other 3 dogs showed homogeneous regularity of the cortical nephrogram despite the hypotension. The difference in the 2 groups in respect to evidence of redistribution of intrarenal blood flow is most likely due to the effect of Dibenzyline in lessening or precluding the constriction of the small peripheral renal vessels following hemorrhage. The levels of hypotension were similar in the 2 groups. A less likely explanation for the difference in appearance of the nephrogram would be the difference in magnitude of bleeding in the 2 groups to reach the desired levels of hypotension-average of 30 ml. per kg. in the Dibenzyline group as compared to 39 ml. per kg. in the dogs without alpha-adrenergic block.

EFFECTS OF EPINEPHRINE

With the intravenous administration of epinephrine during the hypotensive state all dogs showed a similar response in respect to systemic blood pressure. Within 30 seconds of the epinephrine administration the pressure fell, usually 10 to 15 mm. Hg with gradual restitution so that the pre-epinephrine levels were reached within 5 minutes. Epinephrine has both alpha-adrenergic and beta-adrenergic stimulating effects; with block of the alpha-adrenergic receptors by the Dibenzyline, epinephrine's beta-adrenergic effects are manifest resulting in further hypotension rather than the usual epinephrine-induced increase in blood pressure.

Previous studies[2] had demonstrated that this dose of epinephrine (15 μg. per kg.) administered intravenously to normal dogs produced a marked increase in renal vascular resistance with narrowing of the major branches of the renal artery (segmental arteries) near the site of branching of the main renal artery with poor opacification of the narrowed interlobar arteries. These epinephrine effects were even more pronounced in hypovolemic animals. In our present studies the hemorrhaged dogs pretreated with Dibenzyline showed a markedly different response to epinephrine (Table III). Mean systemic blood pressures for the 5 animals at the time of the epinephrine angiogram were 18, 30, 31, 33, and 37 mm. Hg. As compared to the pre-

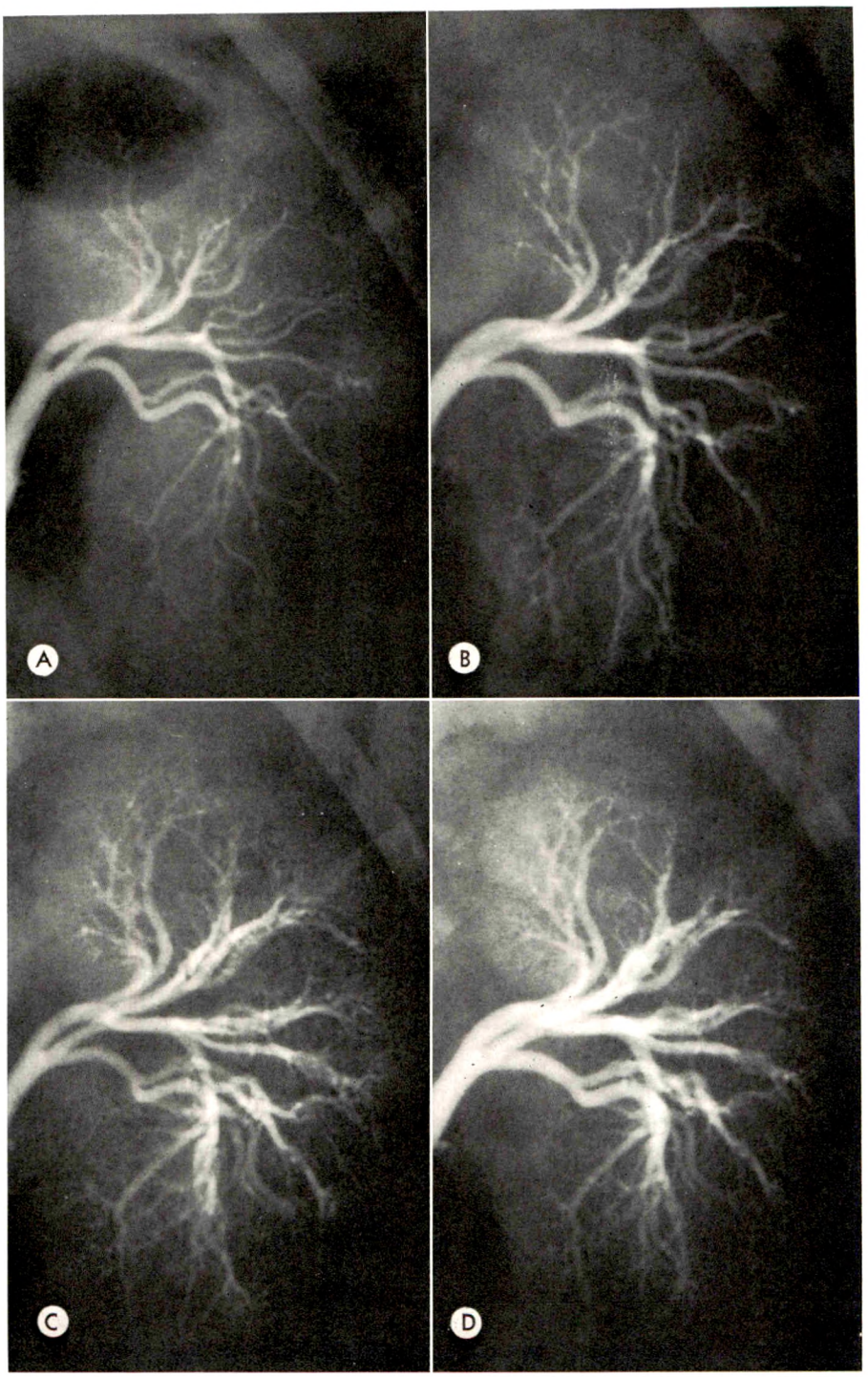


Fig. 1. (*A*) Control angiogram in 12 kg. dog; mean blood pressure 156 mm. Hg; ½ second film after injection of the Thorotrast. (*B*) 30 minutes after completion of Dibenzyline infusion with mean blood pressure 116 mm. Hg; ½ second film. The segmental and interlobar arteries are slightly increased in caliber. (*C*) Within a few minutes after bleeding with mean blood pressure 45 mm. Hg; ½ second film. As compared to *B*, the segmental arteries are slightly narrowed and the interlobar arteries are slightly dilated. (*D*) After epinephrine with mean blood pressure 30 mm. Hg; ½ second film. As compared to *C*, there is slight dilatation of the segmental arteries.

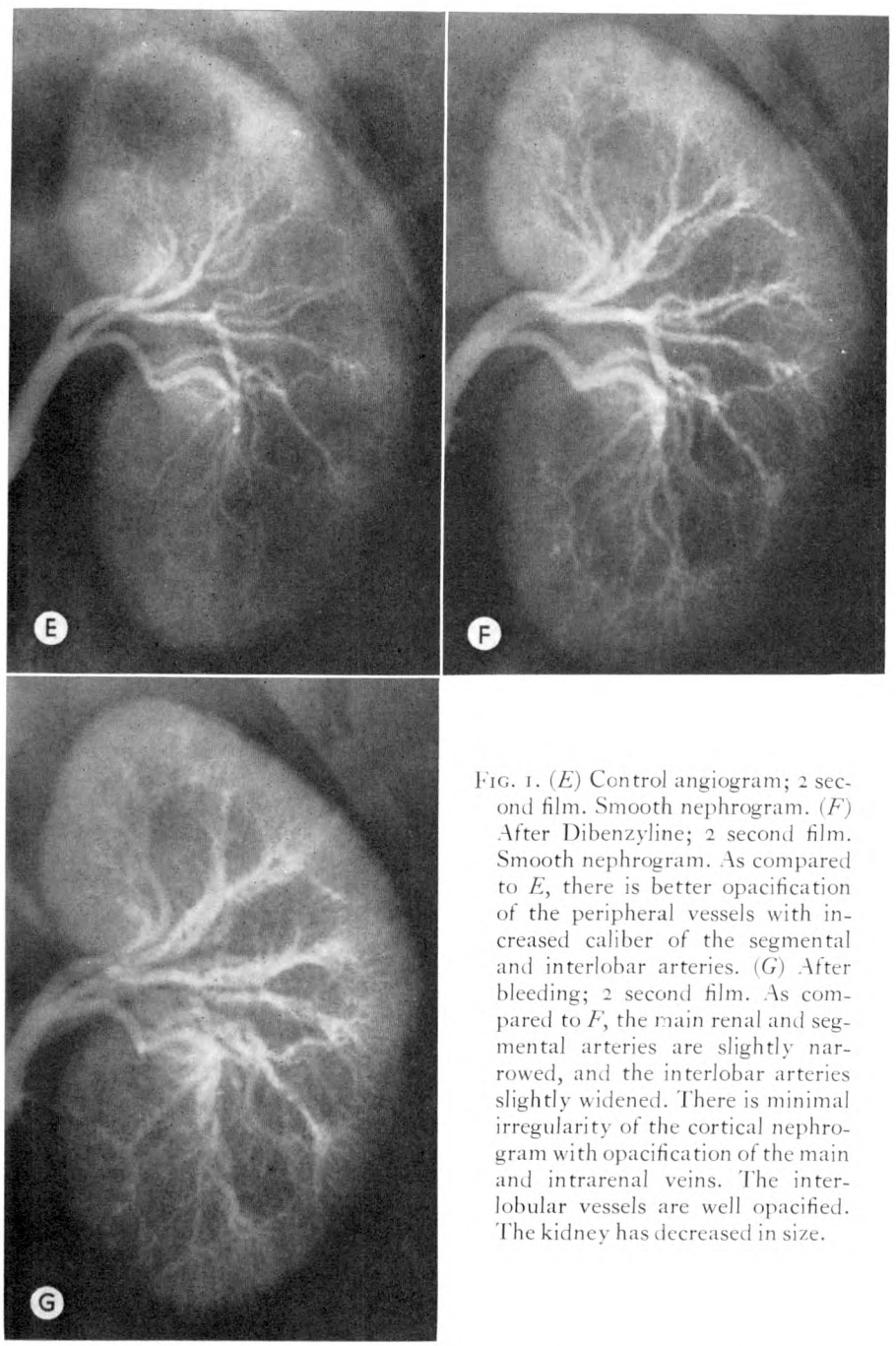


FIG. 1. (*E*) Control angiogram; 2 second film. Smooth nephrogram. (*F*) After Dibenzyline; 2 second film. Smooth nephrogram. As compared to *E*, there is better opacification of the peripheral vessels with increased caliber of the segmental and interlobar arteries. (*G*) After bleeding; 2 second film. As compared to *F*, the main renal and segmental arteries are slightly narrowed, and the interlobar arteries slightly widened. There is minimal irregularity of the cortical nephrogram with opacification of the main and intrarenal veins. The interlobular vessels are well opacified. The kidney has decreased in size.

epinephrine angiograms, the studies showed: no change or increase in caliber of the main renal, segmental, and interlobar arteries; no change or slight diminution in size of the main renal and intrarenal veins (Fig. 1, *D* and *F*; and 2*C*). In all 5 animals the kidney showed no change in size after the epinephrine. Angiographic criteria suggested no change or only slight diminution in renal blood flow. The 2 dogs who had shown minimal irregularity of the cortical nephrogram after hemorrhage continued to show the same findings to the same degree after the epinephrine (Fig. 1*H*; and 2*C*). None

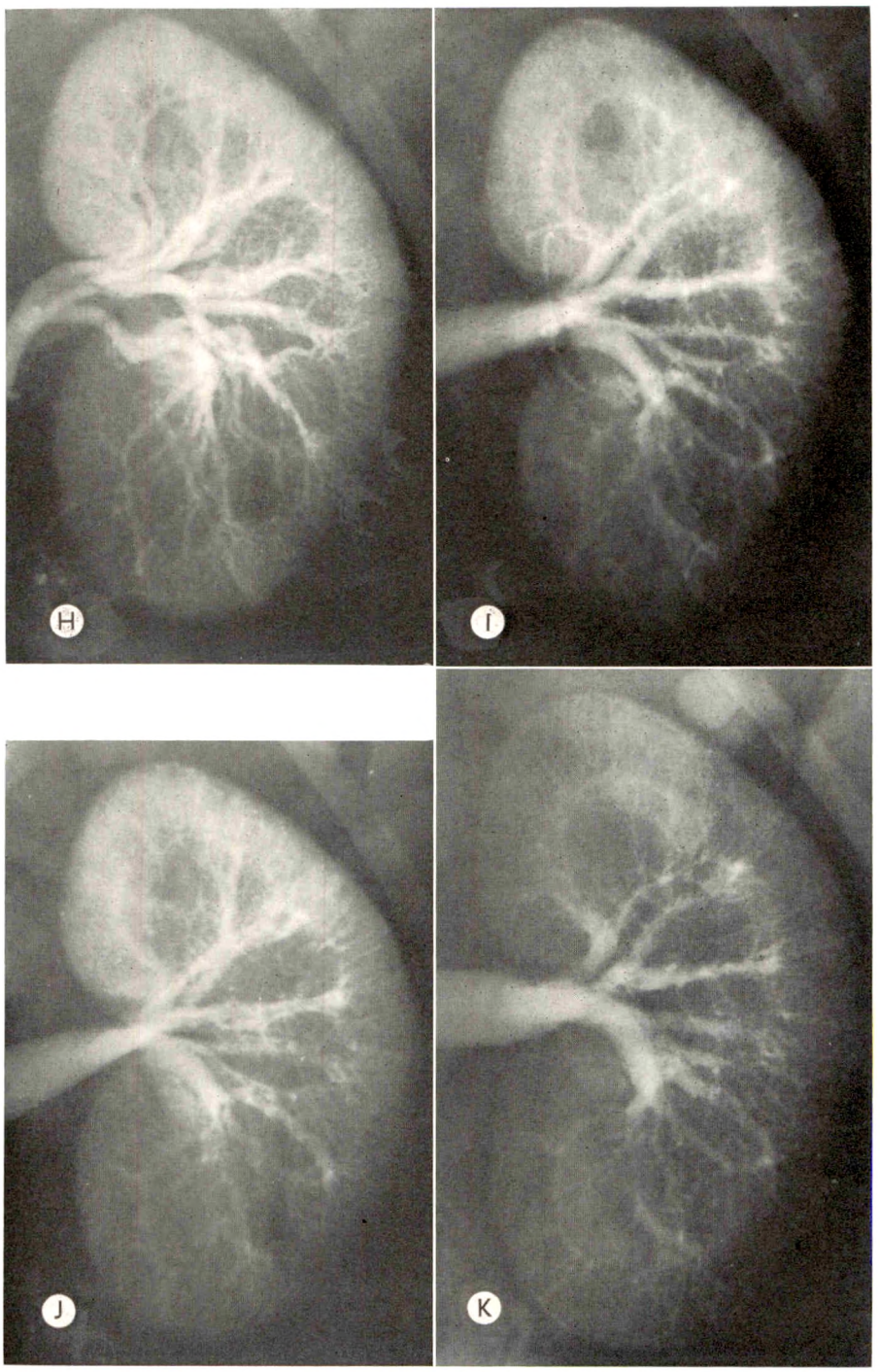


Fig. 1. (*H*) After epinephrine; 2 second film. There is still only minimal irregularity of the cortical nephrogram with good opacification of the interlobular vessels. (*I*) After bleeding; 4½ second film. Good vein opacification. (*J*) After epinephrine; 4½ second film. Good vein opacification, similar to *I*. (*K*) After reinfusion of the blood; 4½ second film. The kidney and main renal vein are increased in size as compared to *I*.

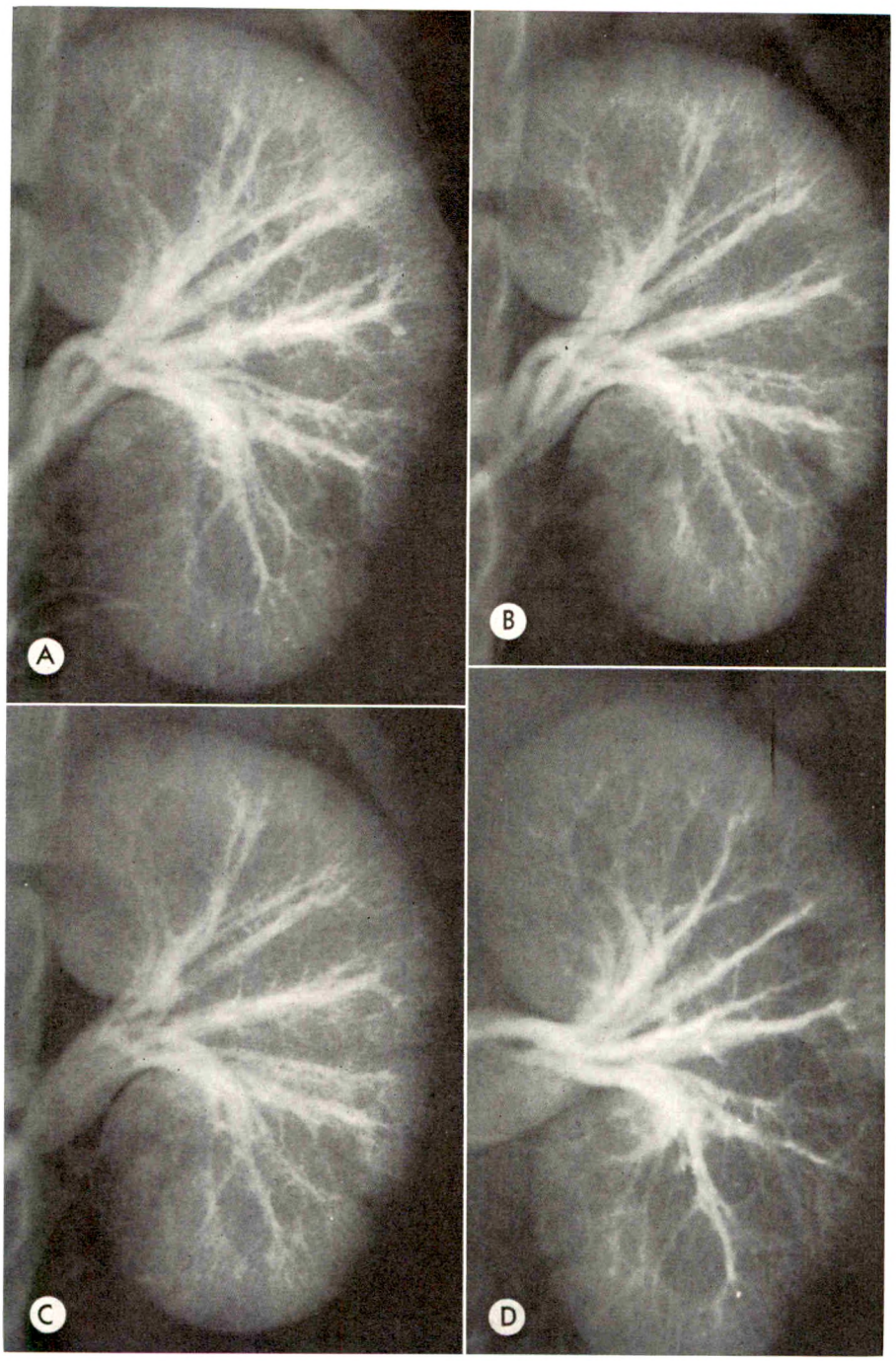


FIG. 2. (*A*) Angiogram in a 15 kg. dog after Dibenzyline; mean blood pressure now 91 mm. Hg, had been 141 mm. Hg in control state; 2 second film after injection of Thorotrast. There is opacification of the arteries and veins. The arcuate and interlobular vessels are seen quite well. (*B*) After bleeding with mean blood pressure 45 mm. Hg; 2 second film. The kidney is smaller than in *A*. The intrarenal and renal vessels are only slightly smaller than before bleeding. The interlobular vessels are still well opacified. There is only minimal irregularity of the cortical nephrogram. (*C*) After epinephrine, mean blood pressure 33 mm. Hg; 2 second film. No significant change from *B*. (*D*) After blood reinfusion mean blood pressure 107 mm. Hg; 2 second film. The kidney is larger than in *B*. Now it resembles angiogram of *A*.

Table III

EPINEPHRINE AFTER DIBENZYLINE AND HEMORRHAGE

	In-crease	De-crease	Un-changed	Indeter-minate
Main renal artery	2		3	
Segmental arteries	3		2	
Interlobar arteries	1		4	
Intrarenal veins		2	3	
Main renal vein		3	1	1
Flow		2	3	

Table IV

CHANGES AFTER REINFUSION OF BLOOD COMPARED WITH PRE-BLEEDING ANGIOGRAM

	Increase	Decrease	Unchanged
Main renal artery	3		1
Segmental arteries	3		1
Interlobar arteries	2		2
Intrarenal veins		2	2
Main renal vein		2	2
Flow			4

of the other animals showed these findings even with the extreme hypotension after the epinephrine.

REINFUSION OF BLOOD

The final angiogram was done within a few minutes of rapid reinfusion of the blood in 4 dogs. The mean systemic blood pressure was restored to the pre-bleeding level or slightly above that level. The average pre-bleeding (post-Dibenzyline) mean systemic blood pressure was 97 mm. Hg and at post-reinfusion of the blood 110 mm. Hg. As compared to the pre-bleeding angiograms the main renal and segmental arteries showed an increase in caliber, the interlobar arteries were the same in size or increased, the main renal and intrarenal veins were the same or decreased in caliber, the renal blood flow was the same as judged from the angiograms (Table IV). In all 4 animals the kidney size was the same as measured on the pre-bleeding angiogram (Fig. 1*K*; and 2*D*).

URINE FLOWS

In 4 of the experiments rough urine flows were determined by collecting 10 minute period bladder urine *via* a catheter in the bladder. These crude urine flow determinations are listed in Table V. After alpha-adrenergic block the urine flow tended to remain unchanged or increase. After hemorrhage the urine flow diminished and then after epinephrine remained at this low level or increased slightly. In 3 of the animals urine flow was monitored after reinfusion of the blood; in all 3 there was a marked diuresis.

SUMMARY

1. Alpha-adrenergic block effects changes in the appearance of the angiogram as compared to that of the control study in the anesthetized dog, manifested by an increase in size of the renal vessels. There is apparently a degree of constriction of the renal vessels in the anesthetized dog.

2. In hypovolemic hypotension after alpha-adrenergic block the angiogram shows only slight, if any, changes in the renal vessels. This differs markedly from the changes in hypovolemic hypotension without Dibenzyline pre-treatment where all renal vessels showed diminution in size.

3. After hemorrhage in the untreated dog there is evidence of redistribution of intrarenal blood flow with diversion of blood away from the cortex as shown by irregularity of the cortical nephrogram. In hypovolemic hypotension after Dibenzyline the diversion of blood away from the cortex is much less marked or not apparent.

Table V

URINE FLOW
(ml. per minute)

Dog No.	1	2	3	4
Control	0.3–0.5	0.2	1.5	0.5
After Dibenzyline	0.3–0.5	0.1	2–2.9	1.7–2
After hemorrhage	0.1	0.3	0.03	.03
After Epinephrine	0.2	0.9	0.1–0.7	.02
After blood reinfusion	1.5–2.7	3.0	5–10	—

4. Intravenous epinephrine in the hemorrhaged, Dibenzyline pre-treated dogs effected no change or increase in caliber of the renal arteries. This differs from the marked vasoconstriction resulting from epinephrine administered to the hemorrhaged dog without alpha-adrenergic block.

5. In hypovolemic hypotension the vasoconstriction is most marked in the cortex with diversion of blood away from the cortex very likely related to endogenous catecholamine action mediated by increased activity of the sympathetic nervous system. This effect is precluded or diminished by alpha-adrenergic blockade.

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ANGIOGRAPHIC EVALUATION OF THE NONVISUALIZING KIDNEY*

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NONVISUALIZATION of one or both kidneys during intravenous pyelography is a frequent roentgenographic finding and often presents a problem in diagnosis. Nephrotomography[1-12] and retrograde pyelography, although routinely utilized, often fail to provide sufficient diagnostic information and are inadequate for evaluating the functional capacity of the remaining renal parenchyma. Renal angiography has proved successful in our experience in providing this information and has a high diagnostic yield, particularly in the evaluation of the unilateral nonvisualizing kidney.

A group of 30 patients (Table 1) in whom renal arteriography and/or venography proved useful in evaluating the cause of unilateral nonvisualization has been reviewed and representative cases are reported.

REPORT OF CASES

CASE I. N.S. is a 66 year old female with known rheumatic heart disease complicated by atrial fibrillation and multiple valve disease. While hospitalized for evaluation of her heart disease, the patient awoke with a sudden onset of dull, constant nonradiating right flank pain. Examination of the abdomen revealed no abnormalities. Intravenous pyelography demonstrated a normal left kidney; however, the right kidney did not visualize. Over the next 24 hours the pain increased in severity and there was an associated decrease in urinary output. Abdominal and selective renal arteriography (Fig. 1, *A–C*) performed 24 hours after the onset of symptoms demonstrated an embolus in the right renal artery just distal to the bifurcation.

TABLE I

UNILATERAL NONVISUALIZING KIDNEYS EVALUATED BY ANGIOGRAPHY

Obstruction of renal artery	9
Agenesis	8
Renal tumor	5
Infection	3
Ureteral obstruction	3
Renal vein thrombosis	1
Arteriovenous fistula	1
Total Cases	30

CASE II. W.W. is a 43 year old male admitted for evaluation of acute right flank pain and hematuria. A diagnosis of membranous glomerulonephritis had been made 1 year prior to admission and the patient was being treated with steroids. An intravenous pyelogram obtained at the time the diagnosis was established was normal. Intravenous pyelography at the time of admission failed to visualize the right kidney. The left kidney was slightly enlarged and visualized normally. Retrograde pyelography (Fig. 2*A*) demonstrated slight enlargement of the right kidney and a normal collecting system. Selective right renal venography and inferior vena cavography (Fig. 2, *B* and *C*) demonstrated thrombosis of the right renal vein. The patient was anticoagulated and follow-up examinations over a period of 6 months demonstrated some return of function of the right kidney.

CASE III. D.W. is a 28 year old male admitted for evaluation of a nonvisualizing right kidney. An intravenous pyelogram (Fig. 3*A*) obtained during the course of a work-up for hypertension demonstrated that the left kidney was normal but enlarged. No right renal outline could be identified. Cystoscopy revealed a normal trigone and right ureteral orifice; however, on retrograde pyelography (Fig. 3*B*), an atretic blind right ureter was seen. Abdominal aortography (Fig. 3*C*) revealed an absent right renal artery and a diagnosis of renal agenesis was made. Surgical exploration revealed a small

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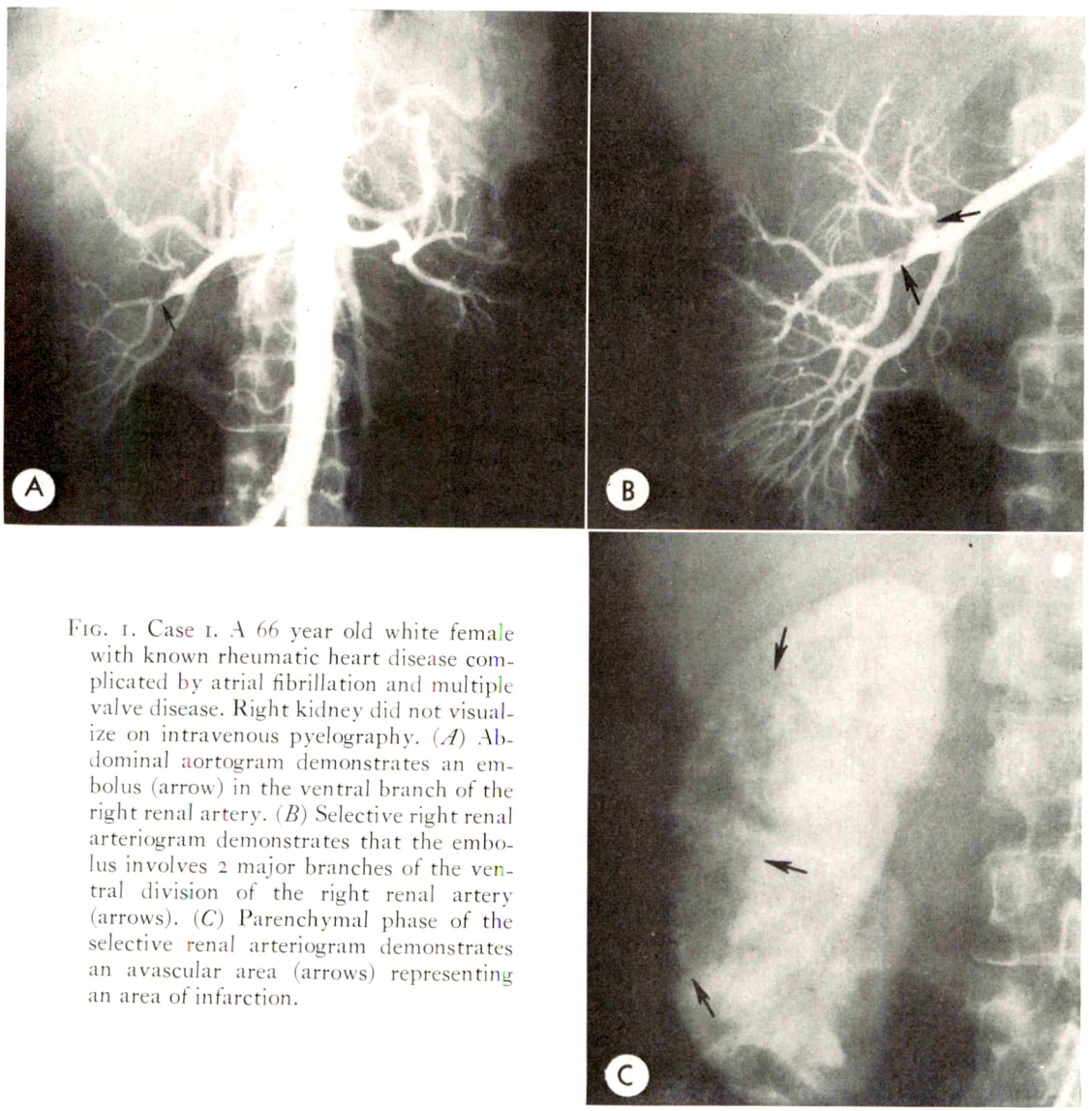


FIG. 1. Case 1. A 66 year old white female with known rheumatic heart disease complicated by atrial fibrillation and multiple valve disease. Right kidney did not visualize on intravenous pyelography. (*A*) Abdominal aortogram demonstrates an embolus (arrow) in the ventral branch of the right renal artery. (*B*) Selective right renal arteriogram demonstrates that the embolus involves 2 major branches of the ventral division of the right renal artery (arrows). (*C*) Parenchymal phase of the selective renal arteriogram demonstrates an avascular area (arrows) representing an area of infarction.

mass weighing approximately 5 gm. which contained small islands of residual renal tissue with several well formed glomeruli. The final pathologic diagnosis was renal dysgenesis.

CASE IV. L.M. is a 57 year old man with a 1 month history of dysuria and right flank pain associated with a 40 pound weight loss. Physical examination revealed a mass in the right flank region. Intravenous pyelography (Fig. 4*A*) using nephrotomography and total body opacification technique demonstrated enlargement of the right kidney and hydronephrosis of the right collecting system. Retrograde pyelography was attempted, but was unsuccessful because of inability to identify the right ureteral orifice. Abdominal aortography (Fig. 4, *B* and *C*) and selective renal arteriography (Fig. 4, *D* and *E*) demonstrated a large hydronephrotic collecting system with a thin rim of renal parenchyma. The abdominal aortogram demonstrated large lumbar vessels and a diagnosis of pyonephrosis was made. At surgery the patient was found to have a large tuberculous pyonephrosis with stenosis at the uretero-pelvic junction.

CASE V. O.G. is an 81 year old female admitted with a 6 month history of hematuria and weight loss. Intravenous pyelography

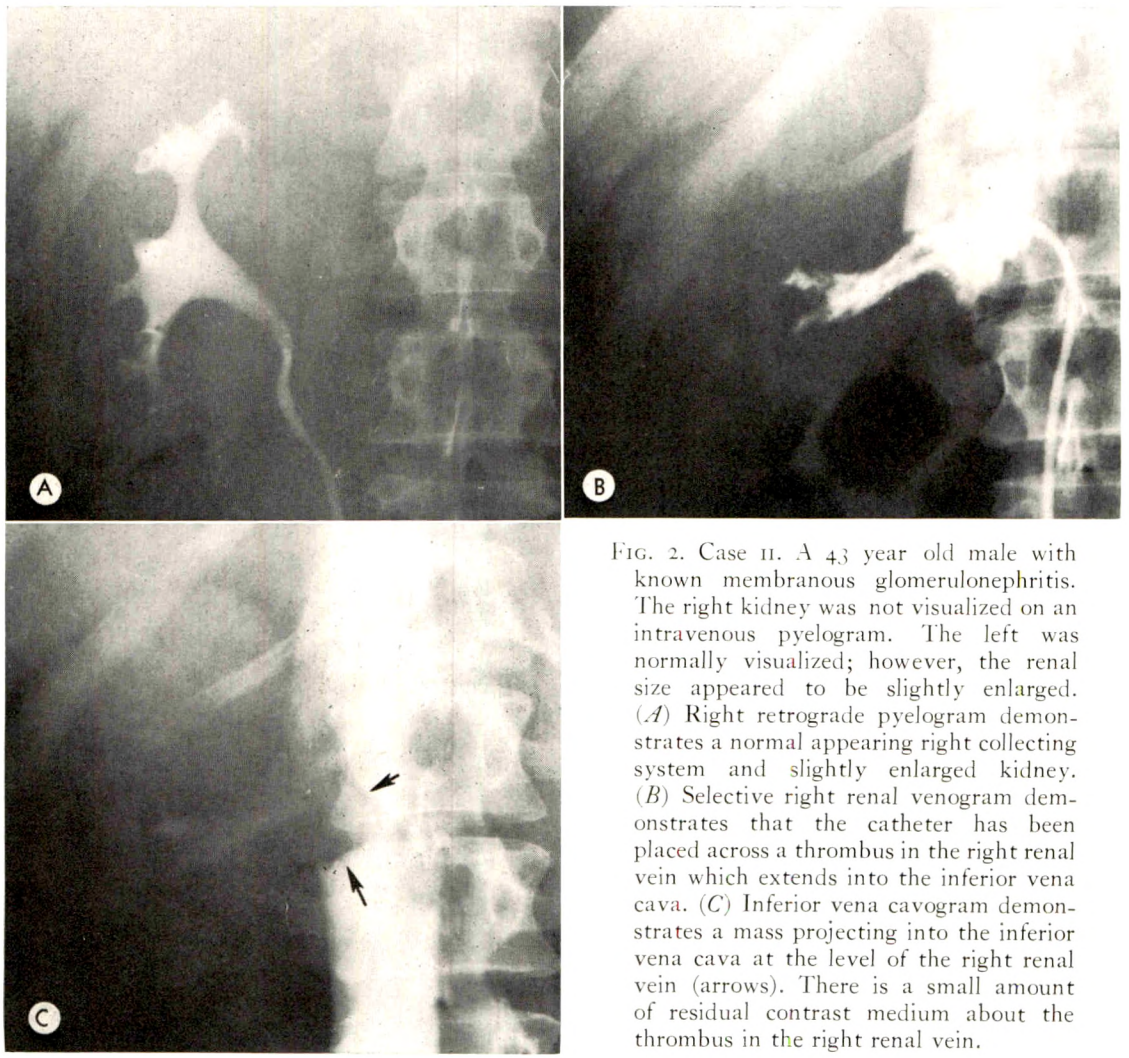


Fig. 2. Case II. A 43 year old male with known membranous glomerulonephritis. The right kidney was not visualized on an intravenous pyelogram. The left was normally visualized; however, the renal size appeared to be slightly enlarged. (*A*) Right retrograde pyelogram demonstrates a normal appearing right collecting system and slightly enlarged kidney. (*B*) Selective right renal venogram demonstrates that the catheter has been placed across a thrombus in the right renal vein which extends into the inferior vena cava. (*C*) Inferior vena cavogram demonstrates a mass projecting into the inferior vena cava at the level of the right renal vein (arrows). There is a small amount of residual contrast medium about the thrombus in the right renal vein.

demonstrated enlargement of the right kidney; however, the collecting system was not visualized. The left kidney was well visualized and appeared normal. Bilateral retrograde pyelography demonstrated a normal left collecting system. On the right there was obstruction of the ureter just distal to the uretero-pelvic junction with dilatation of the ureter at the site of obstruction by what appeared to be a mass within the ureter (Fig. 5*A*). Abdominal aortography and bilateral selective renal arteriography demonstrated a large hypernephroma occupying almost the entire right kidney (Fig. 5*B*). In addition, a small hypernephroma was noted in the upper pole of the left kidney. Because of the patient's age, surgery was deferred and there is no information as to whether the obstruction of the ureter was produced by tumor growing into the renal pelvis or by blood clots.

Case VI. G.H. is a 46 year old male admitted for evaluation of left flank pain of 2 months' duration. The patient denied urinary tract symptoms and no abnormalities were noted on physical examination. An intravenous pyelogram (Fig. 6*A*) demonstrated a normal right kidney; however, the left kidney was not visualized. A mass was noted in the region of the left kidney which displaced the body of the stomach anteriorly on an upper gastrointestinal examination. Retrograde pyelography (Fig. 6*B*) revealed that there was marked distortion of the left collecting system with dilatation of the calyces and narrowing of the renal pelvis. Renal arteriography (Fig. 6, *C* and *D*) demon-

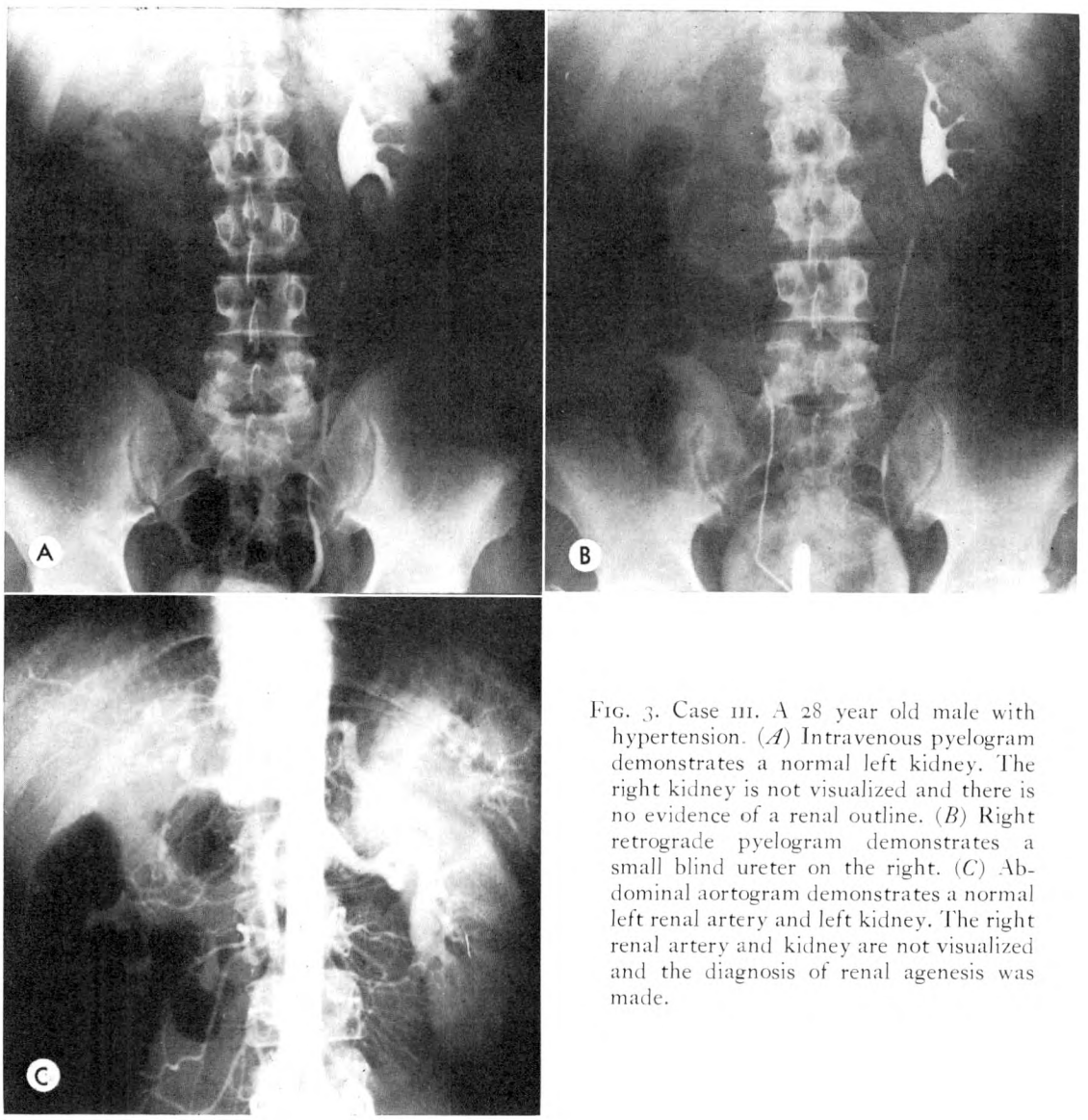


FIG. 3. Case III. A 28 year old male with hypertension. (*A*) Intravenous pyelogram demonstrates a normal left kidney. The right kidney is not visualized and there is no evidence of a renal outline. (*B*) Right retrograde pyelogram demonstrates a small blind ureter on the right. (*C*) Abdominal aortogram demonstrates a normal left renal artery and left kidney. The right renal artery and kidney are not visualized and the diagnosis of renal agenesis was made.

strated a relatively avascular mass in the left kidney with tumor vessels at its periphery. At surgery a large epidermoid carcinoma was found originating in the renal pelvis.

CASE VII. A.O. is a 41 year old female admitted for evaluation of intermittent painless gross hematuria of 2 weeks' duration. An intravenous pyelogram on admission demonstrated delayed visualization of the right kidney; however, a retrograde pyelogram was normal. The patient had intermittent episodes of hematuria during her hospitalization. A repeat intravenous pyelogram during one of these episodes demonstrated a normal left kidney, but failed to visualize the right kidney. A retrograde pyelogram demonstrated a mass in the renal pelvis which was felt to represent a blood clot (Fig. 7*A*). Selective arteriography performed the same day demonstrated an aneurysm of the proximal right renal artery with an arteriovenous fistula (Fig. 7, *B* and *C*).

DISCUSSION

Renal arteriography and/or venography is of greatest value when retrograde pyelography fails to establish the etiology of renal

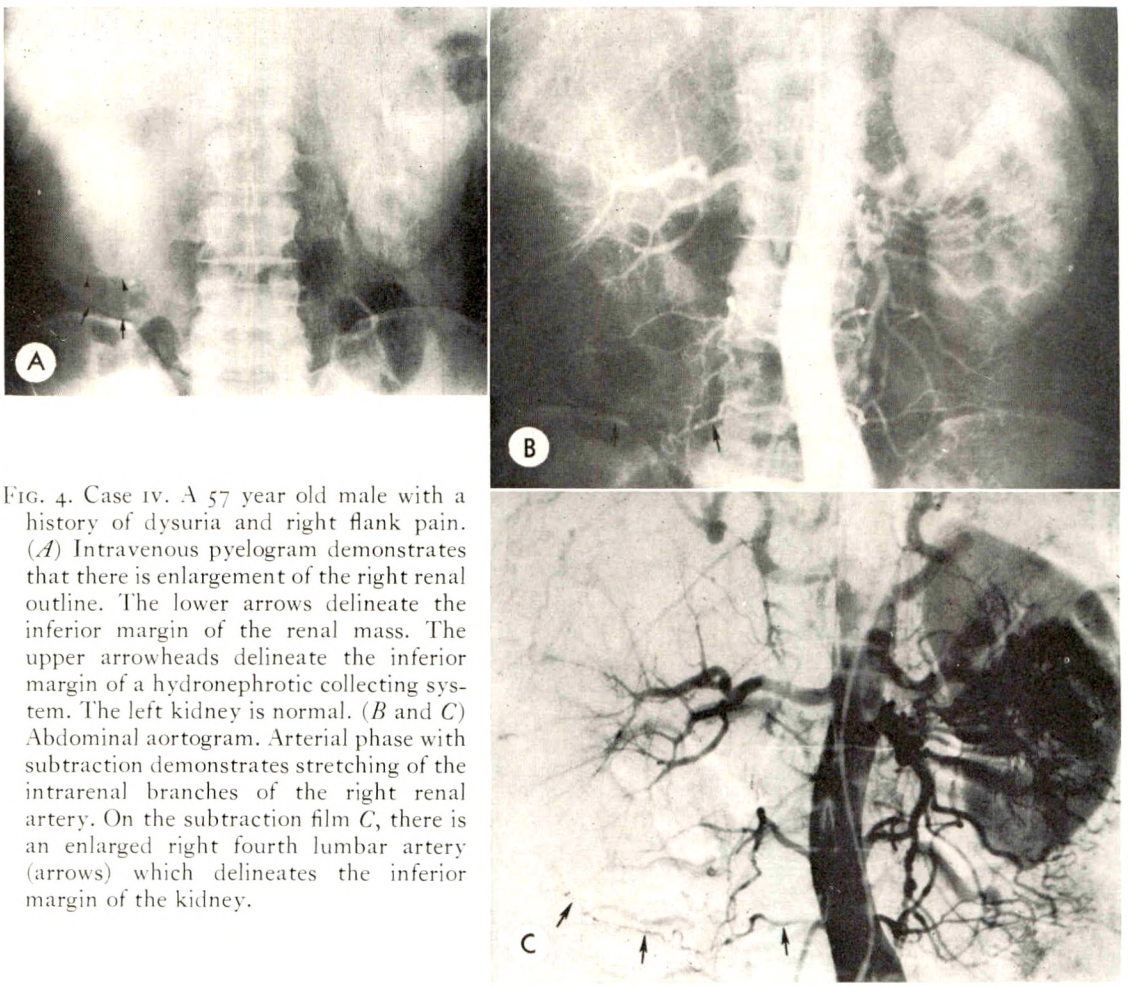


Fig. 4. Case IV. A 57 year old male with a history of dysuria and right flank pain. (*A*) Intravenous pyelogram demonstrates that there is enlargement of the right renal outline. The lower arrows delineate the inferior margin of the renal mass. The upper arrowheads delineate the inferior margin of a hydronephrotic collecting system. The left kidney is normal. (*B* and *C*) Abdominal aortogram. Arterial phase with subtraction demonstrates stretching of the intrarenal branches of the right renal artery. On the subtraction film *C*, there is an enlarged right fourth lumbar artery (arrows) which delineates the inferior margin of the kidney.

nonvisualization.[5,9] Definitive diagnosis by retrograde pyelography is limited to those entities which produce a characteristic derangement of the renal collecting system or ureter and the differential diagnosis of renal mass lesions and parenchymal diseases is beyond the capabilities of the method. The examination may be normal, incomplete or unsuccessful. Acute occlusion of the renal artery or vein usually results in nonvisualization during intravenous pyelography; however, the collecting system remains normal until renal atrophy occurs as demonstrated by Case II. Technical difficulties may interfere with ureteral catheterization or there may be incomplete filling of the ureter and/or calyceal system. When the renal pelvis is markedly dilated, it may obstruct the ureter at the uretero-pelvic junction and prevent catheterization.

Hydronephrosis and renal insufficiency are relative contraindications to retrograde pyelography because of the danger of secondary infection or renal shutdown.[3] Nephrotomography[1,12] has been widely used in the diagnosis of hydronephrosis because of the ability to identify the dilated collecting system by the technique of total body opacification[8] or by identification of the crescent sign.[2,7] Becker[1] has demonstrated that nephrotomography is often useful in evaluating the size of a nonvisualizing kidney or in establishing that a kidney is present. Although the examination is relatively simple to perform and requires little in the way of specialized equipment, in most cases the

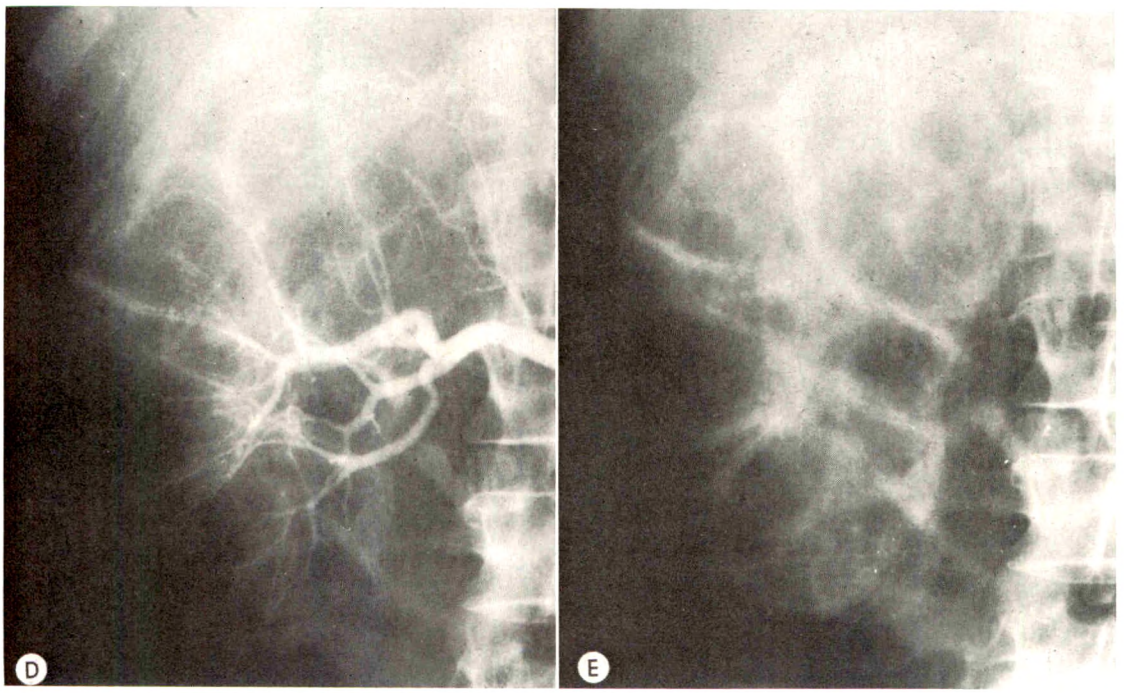


FIG. 4. (*D* and *E*) Representative roentgenograms from a selective right renal arteriography, demonstrate a large hydronephrotic collecting system with thinning of the cortex and stretching of the renal arteries. Because of the prominent lumbar artery, a diagnosis of infection was made. Pathologic diagnosis was tuberculous pyonephrosis.

information obtained is not specific enough to establish the cause of nonvisualization. Absence of the renal outline does not necessarily mean absence of a kidney. There may be unsatisfactory opacification if the renal parenchyma is involved by tumor or infection or if the kidney is nonfunctional because of ureteral or vascular obstruction.

Renal arteriography and venography can provide considerable assistance in establishing the etiology of renal nonvisualization. Idbohrn[4,5] has demonstrated the value of renal arteriography in patients with delayed excretion during intravenous pyelography. In addition to the diagnostic information obtained, he felt that renal arteriography was useful in predicting the functional capacity of the remaining renal parenchyma. Renal arteriography may also be of value in preoperative planning. Idbohrn[6] has shown that obstructive uropathy results in progressive reduction of the main renal artery, reaching a critical 50 per cent level in 30 to 40 days. At this point, renal damage is irreversible and the relief of obstruction frequently fails to restore renal function. Siegelman and Bosniak[9] have discussed the value of renal arteriography in the diagnosis and management of hydronephrosis. An objective assessment of the potential renal function in a hydronephrotic kidney can be made from the size of the renal artery and the amount of remaining parenchyma. This often assists in deciding whether to attempt plastic repair or perform a nephrectomy. Furthermore, the number, course and origin of the renal arteries can be assessed.

The indications for renal arteriography and/or venography in the evaluation of a nonvisualizing kidney include:

1. Incomplete, unsuccessful or normal retrograde pyelogram.
2. Absence of a renal outline on nephrotomography.
3. Hydronephrosis of unknown etiology.
4. Renal mass.
5. Trauma.

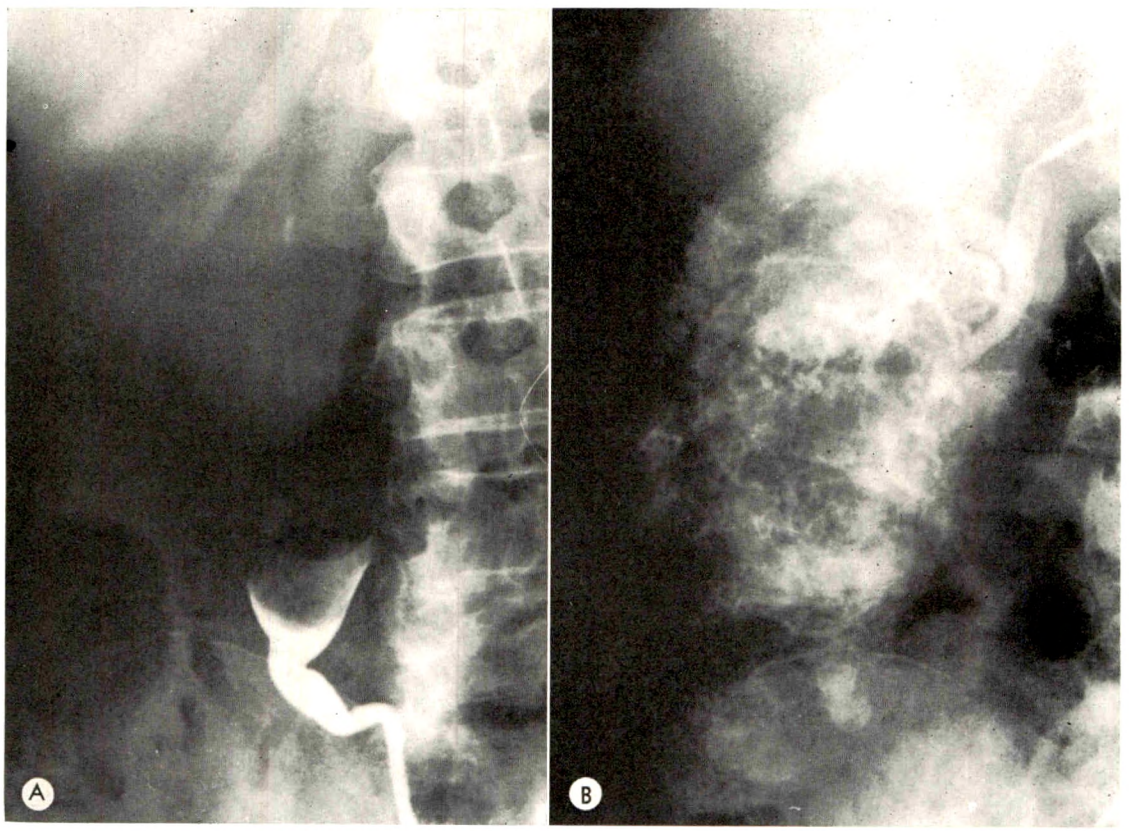


Fig. 5. Case v. An 81 year old female with bilateral hypernephroma. Intravenous pyelogram demonstrated enlargement of the right kidney; however, the collecting system was not visualized. The left kidney was visualized and appeared to be normal. (*A*) Right retrograde pyelogram demonstrates that there is a mass dilating and obstructing the proximal right ureter. (*B*) Selective right renal arteriogram demonstrates involvement of the right kidney by a large hypernephroma. At the time of arteriography, a small hypernephroma was also noted incidentally in the upper pole of the left kidney.

6. Evaluation of the renal vasculature and the functional capacity of the remaining renal parenchyma.

The patients examined in our series do not represent a true spectrum of the problem of nonvisualization but rather reflect the types of patients referred for angiographic studies. Urinary tract outflow obstruction is the most common cause of nonvisualization[10,11] and the roentgenographic changes are those of hydronephrosis. Unless the obstruction is complete, the diagnosis is usually established by delayed roentgenograms; nephrotomography or retrograde pyelography and arteriography are rarely necessary for diagnosis. Only 3 patients with hydronephrosis secondary to nontumorous ureteral obstruction were studied in our series, attesting to the fact that these patients rarely are referred for arteriographic examinations. Because of the value of arteriography in assessing the functional capacity of a hydronephrotic kidney, it is advisable that these patients be studied prior to any surgical procedure to assess the status of the renal vasculature.

In our series the most common cause of nonvisualization was obstruction of the renal artery followed by agenesis of the kidney (Table 1). Renal artery obstruction occurred as the result of severe arteriosclerotic disease, renal artery thrombosis, renal artery embolus and trauma. The patients reported illustrate the problems in differential diagnosis remaining after nephrotomography and retrograde

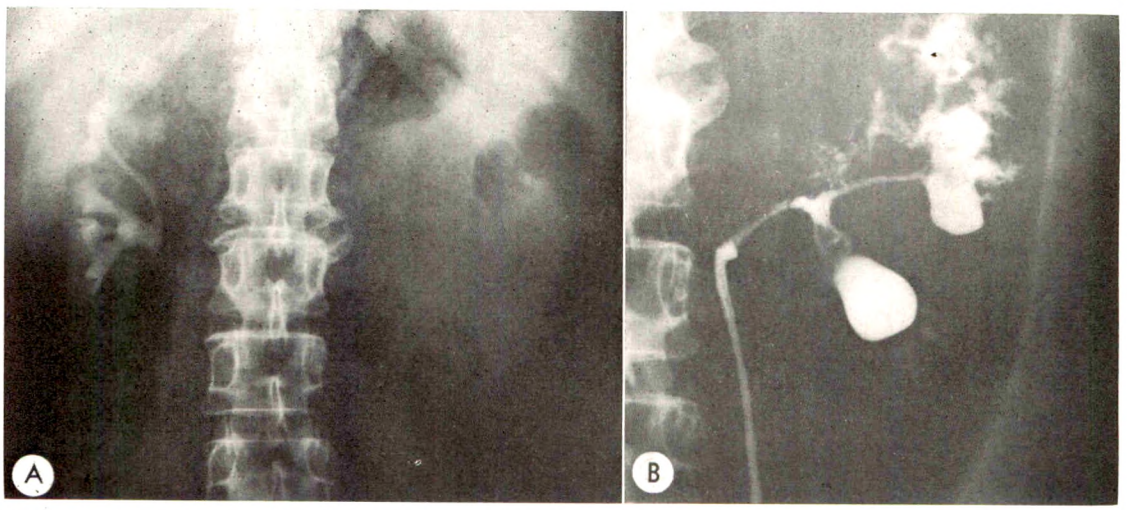


FIG. 6. Case VI. A 46 year old male with epidermoid carcinoma of the left kidney. (*A*) Intravenous pyelogram demonstrates a large mass in the region of the left kidney. The left collecting system was not visualized. The right collecting system is normal. (*B*) Retrograde pyelogram demonstrates that there is marked distortion of the left collecting system with dilatation of those few calyces identified.

pyelography have been obtained. The patients with renal vein thrombosis and arteriovenous fistula had normal appearing collecting systems on retrograde pyelography, while the patient with epidermoid carcinoma of the renal pelvis was mistakenly diagnosed and initially treated as tuberculosis because of the bizarre appearing calyceal system. The patient with bilateral hypernephromas was initially

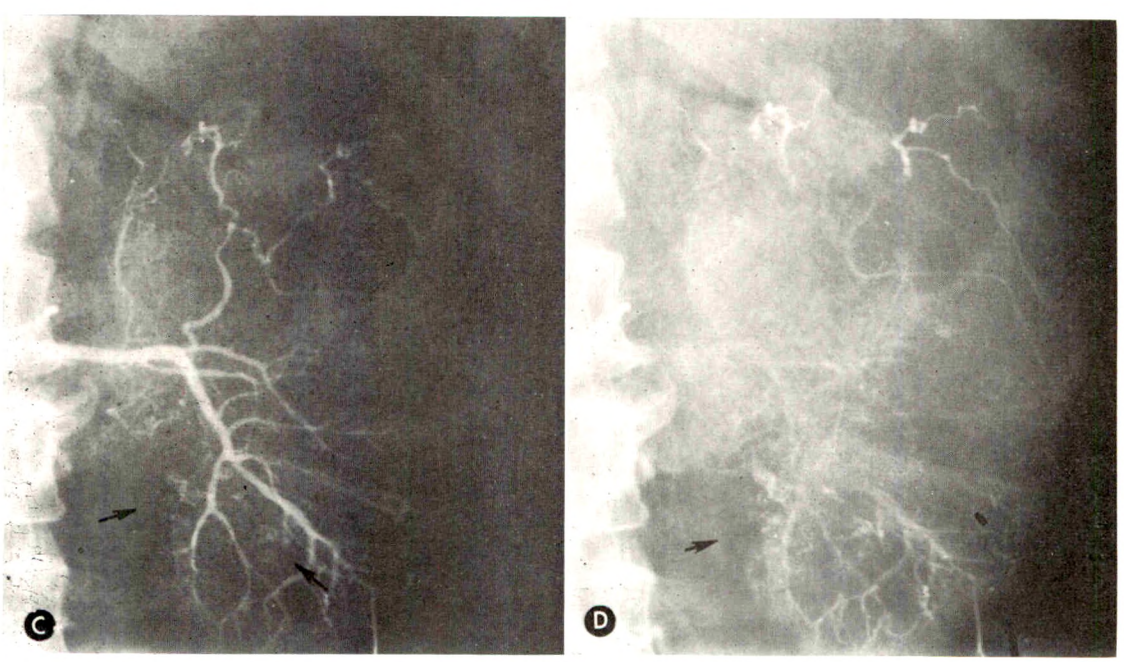


FIG. 6. (*C* and *D*) Representative roentgenograms from a selective left renal arteriography demonstrate stretching of the intrarenal branches with multiple small collections of tumor vessels (arrows) and destruction of the parenchyma.

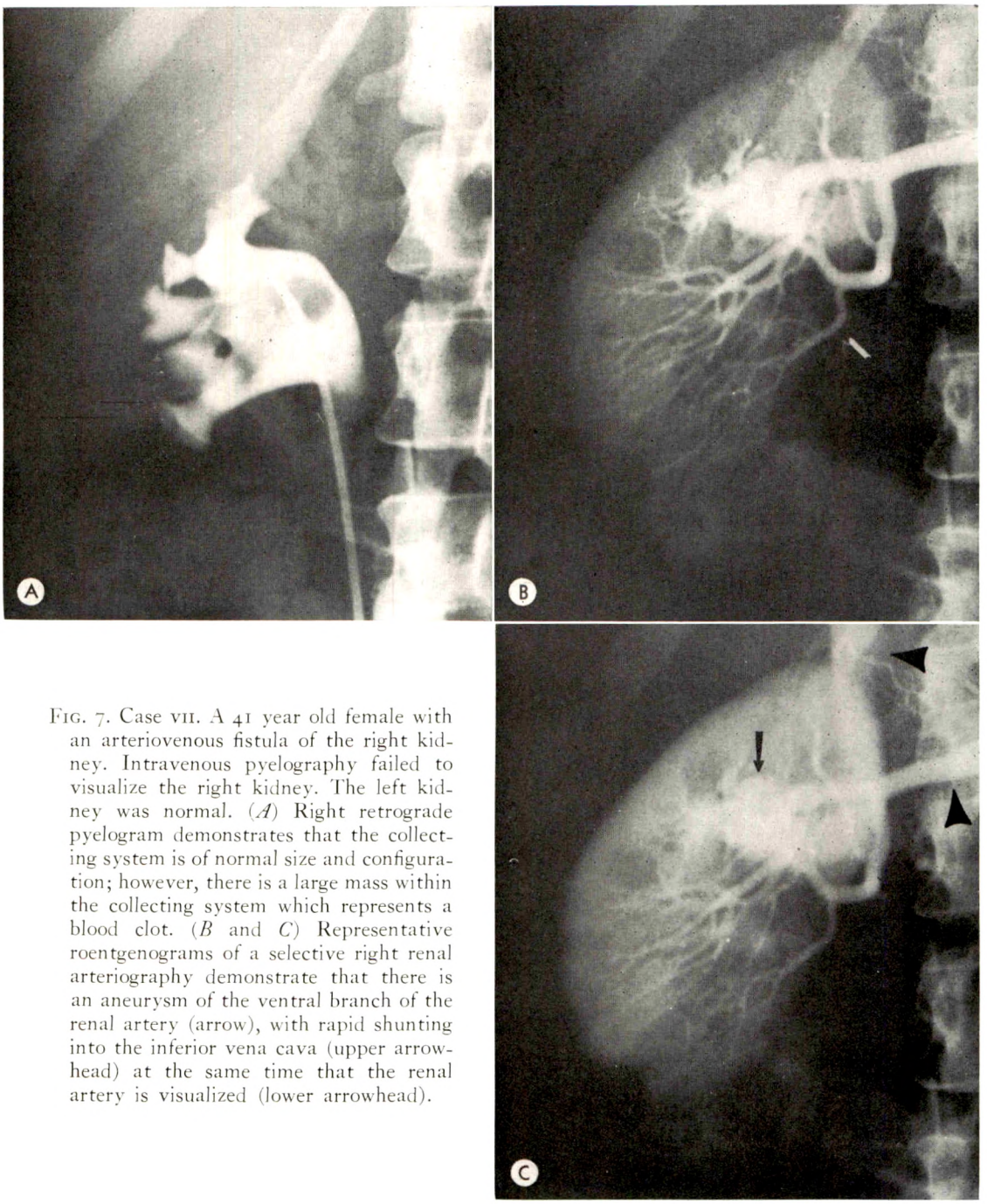


Fig. 7. Case VII. A 41 year old female with an arteriovenous fistula of the right kidney. Intravenous pyelography failed to visualize the right kidney. The left kidney was normal. (*A*) Right retrograde pyelogram demonstrates that the collecting system is of normal size and configuration; however, there is a large mass within the collecting system which represents a blood clot. (*B* and *C*) Representative roentgenograms of a selective right renal arteriography demonstrate that there is an aneurysm of the ventral branch of the renal artery (arrow), with rapid shunting into the inferior vena cava (upper arrowhead) at the same time that the renal artery is visualized (lower arrowhead).

thought to have hydronephrosis on the right as a result of a ureteral tumor. Retrograde pyelography was technically unsuccessful in the patient with tuberculous pyonephrosis, and a blind ureter was identified in the patient with renal agenesis. In all cases arteriography and/or venography was diagnostic.

CONCLUSION

Intravenous and retrograde pyelography may be unsuccessful in establishing the

cause of unilateral renal nonvisualization. A group of patients in whom renal arteriography and/or venography proved useful is reported.

Obstruction of the renal artery by thrombosis, embolus or trauma was the most common cause of unilateral nonvisualization evaluated in our series.

Other causes in order of decreasing frequency included renal agenesis, renal tumor, infection, ureteral obstruction, renal vein thrombosis and arteriovenous fistula.

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VOL. 119, NO. 4

SEMISELECTIVE RENAL ANGIOGRAPHY*

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IT IS the purpose of this communication to describe a simple technique for renal artery visualization without disturbing superimposition of other abdominal vessels. This technique has been used in 50 patients and was found to be superior to previously used end occluded catheter or open catheter methods.

METHOD

A ring-shaped catheter similar to the one previously described by Williams *et al.*[3] and Paulin[2] for nonselective coronary arteriography is used. The catheter consists of a loop with numerous lateral side holes (Fig. 1). After percutaneous introduction of the guidewire, the catheter is advanced over the guide into the upper abdominal or lower thoracic aorta. Following withdrawal of the guidewire, the catheter resumes its original ring shape. The diameter of the catheter ring is slightly smaller than the diameter of the abdominal aorta. It is gently withdrawn and rotated under fluoroscopic control until it reaches the level of the renal arteries. The renal arteries are identified by small test injections or by a sudden slight expansion of the catheter loop when it enters the renal artery orifices. (This applies only if both renal arteries arise at the same level.) If one renal orifice is higher, the catheter loop is positioned at the proximal renal artery level (Fig. 2). Ideally, the catheter loop should lie in a horizontal position, which is best accomplished by advancing it into the thoracic aorta then gentle withdrawal and simultaneous rotation, thus keeping the catheter coil tight.

Arteriography is performed in the usual fashion using approximately 40–50 cc. of contrast medium at a slightly slower injection speed than commonly used for renal arteriography with end open or end occluded catheters. Delivery rates and amounts of contrast medium are adjusted to estimated renal flow rates, size of patient, size of aorta, etc. An approximate flow rate of 20 cc./second is suitable for most patients.

For the introduction of the catheter, a curved, preferably teflonized guidewire is used since straight guidewires may tend to exit through one of the lateral side holes. For exchange, the catheter is withdrawn into the common iliac artery which straightens the loop, and a guidewire is re-introduced under fluoroscopic observation. Exit of the guidewire through a side hole is thus readily visible.

By positioning the catheter loop at the mouth of the celiac axis or superior mesenteric artery, contrast visualization of these arteries is significantly enhanced; however, visualization remains inferior to selective catheterization techniques.

RESULTS

The renal arteriograms of 35 patients undergoing renal arteriography with the described catheter were analyzed. Special attention was given to the degree of renal artery visualization, the nephrographic effect and the degree of disturbing opacification of the superior mesenteric and celiac branches. There was uniformly excellent renal artery visualization. Only 1 case showed suboptimal demonstration of 1 kidney which was due to inadequate positioning of the catheter. In this case the right renal artery arose approximately 1 cm. cephalad to the left renal artery, but the catheter was positioned at the level of the left renal artery. Consequently, visualization of the right renal artery was only fair, but still sufficient for diagnostic purposes. Most cases showed a minor degree of supe-

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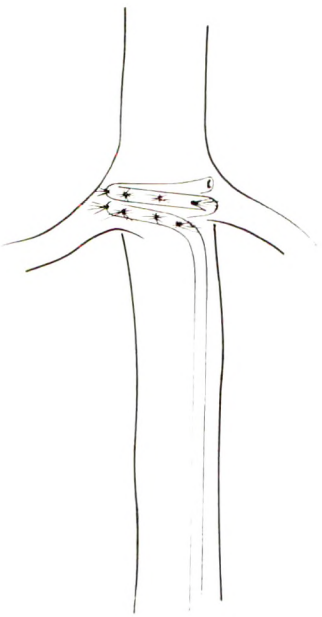


FIG. 1. Diagrammatic representation of the ring catheter positioned at the level of the renal arteries. Note that the catheter bulges slightly into the renal artery orifices.

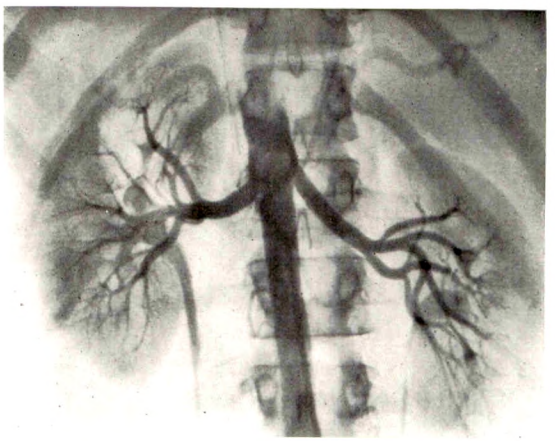


FIG. 2. Renal arteriogram in a patient with the renal arteries arising at different levels. The loop is positioned correctly at left renal artery level. Note only the faint opacification of disturbing abdominal branches.

rior mesenteric artery opacification which was, however, so dilute that it did not interfere with the interpretation of the renal arteriogram. Transitory faint filling of the celiac axis was rare.

In cases with suspected hypernephromas, where bilateral renal arteriography is desirable in addition to selective renal arteriography, the selective procedure could be eliminated by this technique.

DISCUSSION

Ideally, in renal arteriography, the renal arteries should be densely opacified without disturbing superimposition of other abdominal vessels which make interpretation difficult. This is particularly true in the evaluation of renal donors where small segmental renal branches can be easily overlooked.

In order to achieve this goal, an open end catheter with additional side holes can be positioned below the level of the renal arteries; and the injection can be made with such a force that the contrast medium reaches only the level of the renal arteries during diastole. This titration of blood flow can be carried out under fluoroscopy, and, depending on speed of blood flow, the catheter can be withdrawn more or less below the level of the renal arteries and the injection force adjusted. This technique, although successful in experienced hands, results in considerable dilution of contrast medium during systole where unopacified blood enters the renal arteries, thus causing a "weak" renal arteriogram.

If end open catheters with side holes are positioned exactly at the level of the renal arteries as recommended by some angiographers, opacification of the celiac axis and superior mesenteric artery is usually excessive. A renal arteriogram of inferior diagnostic quality is the result.

Superior visualization and technically easier catheter positioning is accomplished by the use of an end occluded or pigtail type catheter. Under these circumstances, the catheter can be positioned at the level of the renal arteries; and the forward jet through the end hole is eliminated. The catheter, however, remains separated from the renal artery orifice; and, therefore, the contrast medium is again diluted, particularly during systole.

A ring-shaped catheter loop, on the other hand, allows delivery of contrast medium

through lateral side holes directly into the renal arteries. Systolic dilution is, therefore, eliminated, and the concentration of the contrast medium in the renal arteries is higher as compared with the other catheterization techniques (Fig. 3, *A* and *B*). Only 10 per cent of the contrast medium is ejected from the end hole of the ring catheter, and even this small amount of contrast medium is not propelled retrograde against the orifice of the superior mesenteric artery or celiac axis.

The use of this catheter for semiselective visualization of the abdominal branches was suggested by Williams and associates,[3] but it never became popular. It was used by us successfully in 1962[1] but was later abandoned because of the inconvenience of making this type of catheter. With the availability of commercially made disposable catheters, its use is again suggested.

No complications have occurred in our patients; but most of the performed studies were in the younger age group, primarily renal donors. With any type of catheter with numerous side holes, blood may enter to the most proximal side hole, and thrombus formation with embolization may occur unless the catheter is intermittently, forcefully flushed with heparinized Ringer's solution. A continuous catheter drip system is inadequate to prevent thrombus formation in these catheters.

The second possible source of complication may be dislodgement of atherosclerotic plaques upon withdrawal of the catheter from the iliac artery. In older persons, the ring catheter should be straightened by the guidewire prior to its removal. In the older age groups and particularly in abdominal aneurysms its use is contraindicated.

CONCLUSION

The use of a ring-shaped catheter similar to the "Littmann loop" or "Paulin catheter" allows superior visualization of the renal arteries. The technique is simple and has been found to be safe in 35 examined patients. Contrast visualization of the renal arteries was excellent with minimal or no interference from opacified abdominal branches. No complications have occurred.

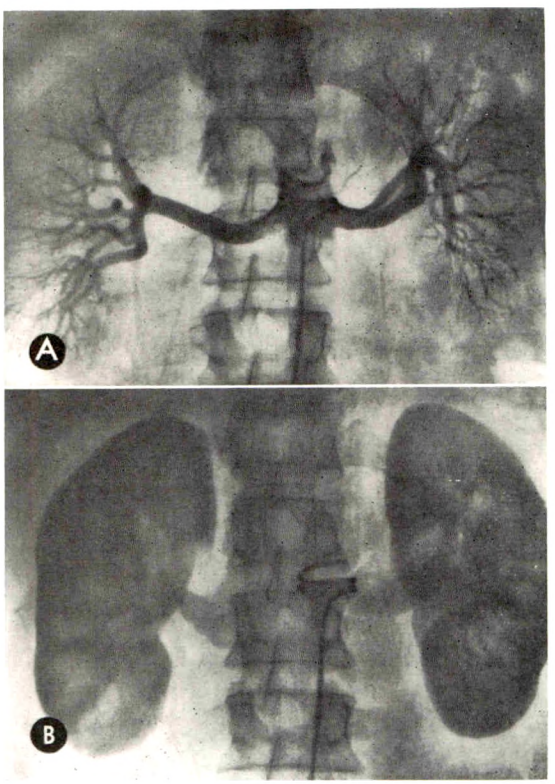


Fig. 3. (*A*) Well positioned loop at renal artery level opacifying both kidneys during systole. (*B*) Because of high contrast level, both renal veins are clearly visualized (arrows).

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LESIONS CAUSING RADIOLUCENT DEFECTS IN THE RENAL PELVIS*

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URO-RADIOGRAPHIC procedures have improved vastly since their meager beginnings in the early 1900s. Improvements in excretory and retrograde urography have provided better visualization of the renal contours, calyces, pelves, ureters and the bladder. Angiography has made possible visualization of the renal vasculature and, along with nephrotomography, differentiation of renal cysts and parenchymal tumors has become possible in 95 per cent of the cases.

Despite these advances, urologists and radiologists alike have difficulty interpreting lesions which present as "filling defects" on excretory urograms and retrograde pyelograms. Stones, tumors, inflammatory lesions, cysts, congenital anomalies and normal variants all enter into the differential diagnosis. Attempts to improve the diagnostic accuracy of these lesions through the use of cytology and bacteriology have been made. However, these studies by themselves are often misleading. It is our feeling that careful study of the radiographic findings will help in the accurate identification of lesions producing filling defects of the renal pelvis.

MATERIAL

Negative renal pelvic defects secondary to technical deviations in obtaining the pyelograms are sometimes confusing. Incomplete filling of the renal pelvis and calyces may produce the appearance of a pathologic filling defect (Fig. 1, *A* and *B*). Such confusion can be alleviated by ensuring more complete filling of the transport system through the use of adequate amounts of contrast media, applying compression devices over the lower abdomen, and occasionally by placing the patient in the prone or Trendelenburg position.[5]

Iatrogenic pyelographic defects occur following the intentional or inadvertent introduction of air during retrograde pyelography. These defects are usually small, centrally located and change position during subsequent filming sequences. They can be avoided by filling the ureteral catheter with a nonopaque, sterile fluid prior to its introduction into the upper urinary tract. When retrograde air pyelography is desired for diagnostic evaluation of renal pelvic lesions, it should be performed following the opaque pyelography since retention of small remnants of the air will produce additional confusing negative defects on subsequent opaque pyelographic films.

Occasionally the entire ureter, pelvis and calyceal system is filled with air as the result of a vesicovaginal fistula or a ureterointestinal anastomosis (Fig. 2, *A* and *B*). The collecting system outlined in this manner rarely offers a problem in diagnosis due to the large amount of gas which is present and the patient's clinical history.

Normal variation in renal pelvic morphology may simulate pathologic lesions roentgenographically. Bifid and trifid renal pelves are among the most common of these variants that produce shadows misinterpreted as pelvic filling defects. Overlap of the major or minor calyces will appear as a radiolucent filling defect in the anteroposterior projection. Calyces viewed on end and overlying intestinal gas can also appear as a pelvic filling defect. Roentgenograms obtained in the oblique

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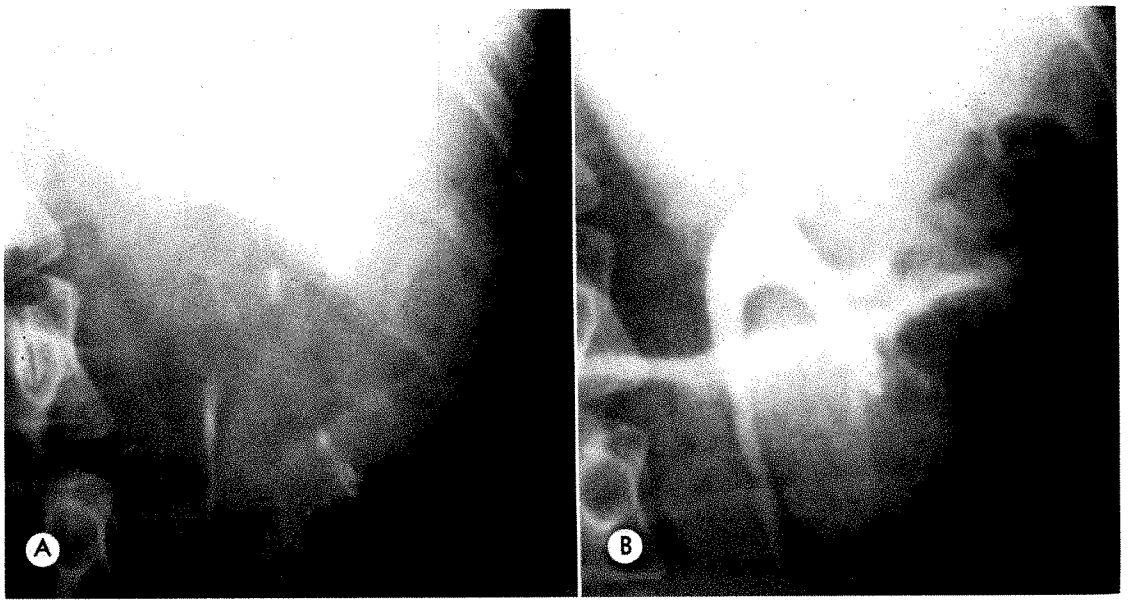


FIG. 1. (*A*) Incomplete filling of the renal pelvis on the 10 minute roentgenogram. (*B*) External abdominal compression device was applied and a normal renal pelvis is demonstrated.



FIG. 2. (*A*) Plain film excretory urogram shows gas over the right renal area. (*B*) Following injection of contrast medium, this air is noted within the collecting system. The patient has a ureterosigmoidostomy because of extrophy of the bladder.

projection will differentiate the simulated filling defect from an intrinsic calyceal or pelvic lesion.

Intrahilar branching renal vessels sometimes compress portions of the transport system simulating an intrinsic filling defect. These are most obvious when they cross the superior infundibulum. In this area they produce transverse linear compression lines which with adequate filling and oblique or lateral projections will be localized extrinsic to the collecting system. Fraley reported flank pain due to this anomaly.[9]

Extrinsic compression from excessive accumulation of fatty tissue around the collecting system within the renal hilum may produce negative defects on pyelography (Fig. 3). In our experience this has been seen most commonly in older patients who also have loss of renal parenchyma from arteriosclerotic ischemia, infection, or infarction. The major calyces are elongated and narrowed by the extrinsic pressure from increased fat in the renal sinus. Parenchymal opacification with nephrotomography accentuates the presence of fatty tissue in the renal sinus and helps to establish the diagnosis of renal sinus lipomatosis.[8]

Aside from normal variants and artifacts, the principal causes of lucent defects in the renal pelvis and calyces are stones, infection, blood clot, neoplasms and cysts. However, combinations of these causes are the major source of confusion which necessitate as accurate a differentiation as is possible.

Opaque stones are visible on the plain film roentgenogram prior to administration of contrast material and will not be discussed here. Nonopaque stones which produce filling defects are composed of uric acid or the urates of ammonium, sodium, magnesium or potassium (Fig. 4). They account for 4 per cent of all renal and



FIG. 3. Nephrotomographic demonstration of fibrolipomatosis. Note increased radiolucency in renal sinus.

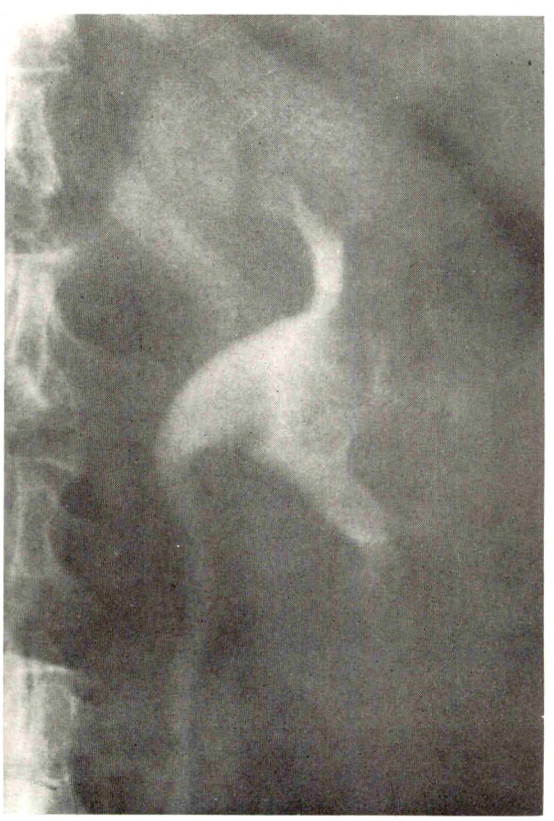


FIG. 4. Uric acid stone in renal pelvis.

ureteral calculi.[6] Hyperuricemia associated with gout or antimetabolic management of leukemia predisposes to calculi of this composition (Fig. 5). Cystine stones vary in radiodensity and daily urinary excretion of cystine should be performed in all recurrent stone formers. Likewise, although less common, xanthine stones and matrix stones composed of mucoproteinacious material sometimes contain insufficient quantities of calcium salts to permit visualization on plain roentgenograms (Fig. 6). Roentgenographically, the lucent filling defects of nonopaque calculi produced on excretory urography will not change in size or shape over a period of several days, while those of blood clot or air bubbles will change. All can vary in position within the urinary tract from one examination to the next. While uric acid stones are usually smooth and regular in outline, matrix stones may produce an irregular filling defect not dissimilar from blood clot, tumor or sloughed tissue. Very small lucent stones may be obscured by opaque contrast material and go undetected. The classic clinical stone symptoms of abdominal and flank pain, renal colic and gross or microscopic hematuria are not helpful in differentiating between these lesions, because any of these pathologic processes may produce urinary obstruction with this symptom complex.

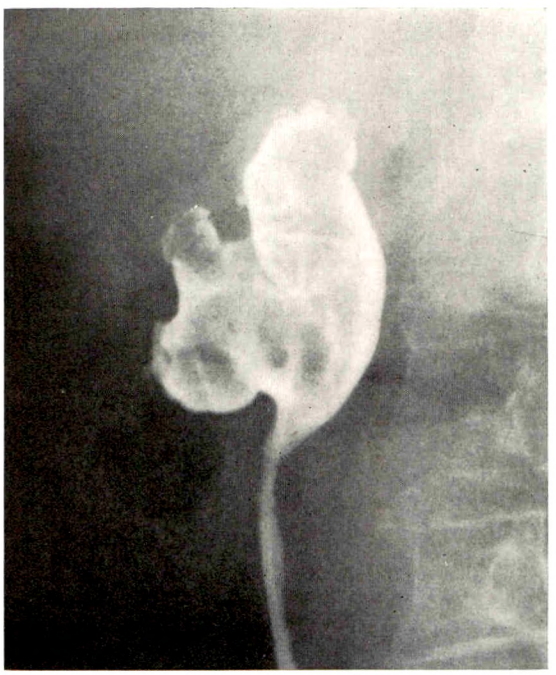


Fig. 6. Matrix stone.



Fig. 5. Uric acid crystals in a patient receiving vincristine therapy for leukemia.

Chronic long standing infection or severe acute inflammation can produce renal pelvic filling defects. Ureteritis cystica and pyelitis cystica result from chronic inflammation located beneath the mucosa of the renal pelvis or ureter (Fig. 7). Multiple small cysts project into the lumen causing a roentgenographic appearance characterized by sharp, smoothly outlined filling defects that project into the pelvic or ureteral lumen when visualized in profile.

Papillary necrosis is a condition in which the renal papillae become necrotic and slough into the pelvis producing a negative pyelographic defect (Fig. 8). The patho-

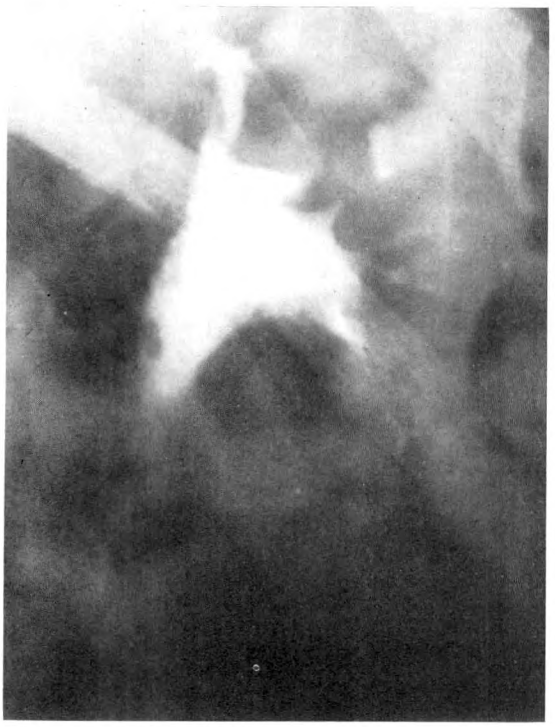


FIG. 7. Pyelitis cystica.

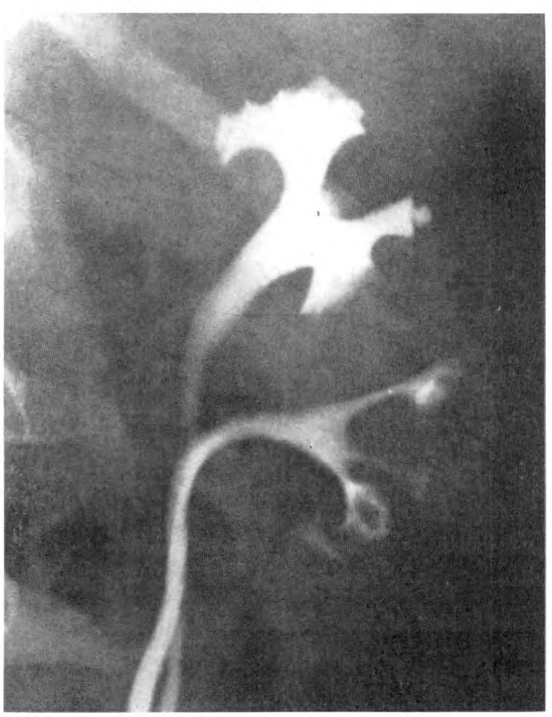


FIG. 8. Retrograde pyelogram showing reduplicated collecting system with papillary necrosis affecting upper collecting system in the medullary form and lower collecting system in the papillary form.

genesis of this disease is a vascular thrombosis secondary to multiple factors including chronic infection, diabetes mellitus, long-term phenacetin ingestion, chronic urinary tract obstruction, and sickle cell disease.[7] In addition to the negative filling defects within the collecting system, the pyelographic picture includes erosion of the papilla, cavitation, pyelointerstitial blackflow and a signet ring shadow in the papillary region. Two of our patients with papillary necrosis required pyelostomy to remove sloughed papillae from the renal pelvis. The residual debris subsequently become impregnated with

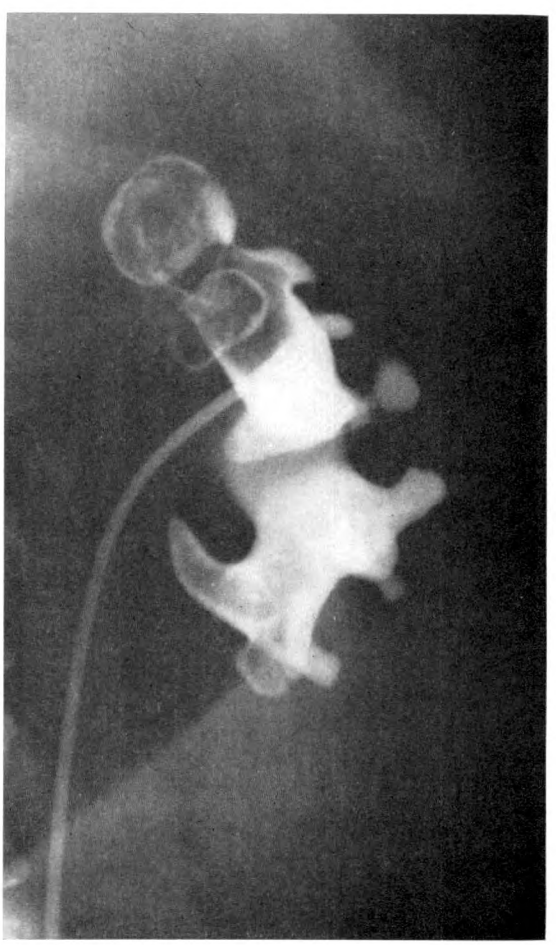


FIG. 9. Retrograde pyelogram with filling defect in the upper pole of the left kidney. Diagnosis was papillary necrosis with *Candida albicans* impregnating the debris forming a fungus ball. (Reproduced with permission of *Radiology*.[1])

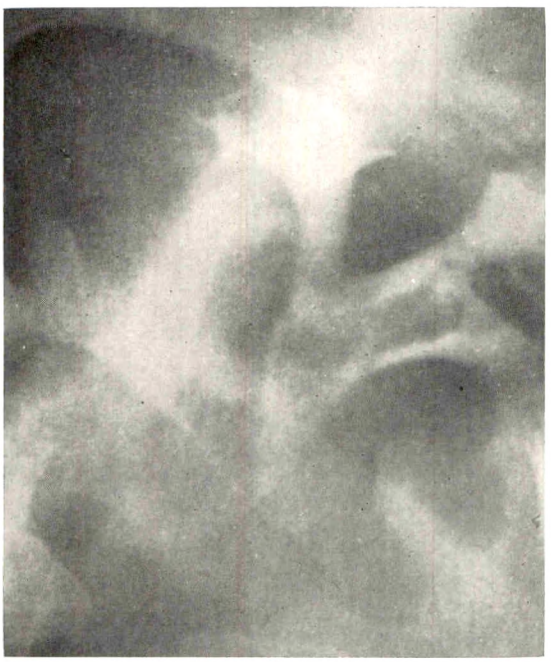


Fig. 10. Blood clot in renal pelvis secondary to trauma.

Candida albicans producing fungus balls which presented roentgenographically as radiolucent filling defects[1] (Fig. 9).

Squamous metaplasis of the uroepithelium is an uncommon cause of filling defects in the renal pelvis.[11] This is associated with urinary tract infection and renal calculi. The diagnosis is made by finding keratinized squamous epithelium in the urine.

Blood clots can be responsible for irregular lucent filling defects of the renal pelvis (Fig. 10). These occur secondary to trauma, infection, anticoagulant overdosage, urolithiasis or neoplasm. In the latter 2 cases, the clot may alter the roentgenographic appearance of the more significant underlying lesion, or completely obscure it. The major significant difference of the renal pelvic filling defect secondary to blood clots is the change that takes place in the size, shape and location of the defect on subsequent examinations.

Benign renal masses such as peripelvic (Fig. 11, *A* and *B*) or renal parenchymal (Fig. 12) cysts may impinge upon the renal pelvis and present as a filling defect. In some instances this may occur on an intermittent basis only. Benign hamartomas (angiomyolipoma) may impinge upon the renal pelvis producing a lucent pressure defect.[10]

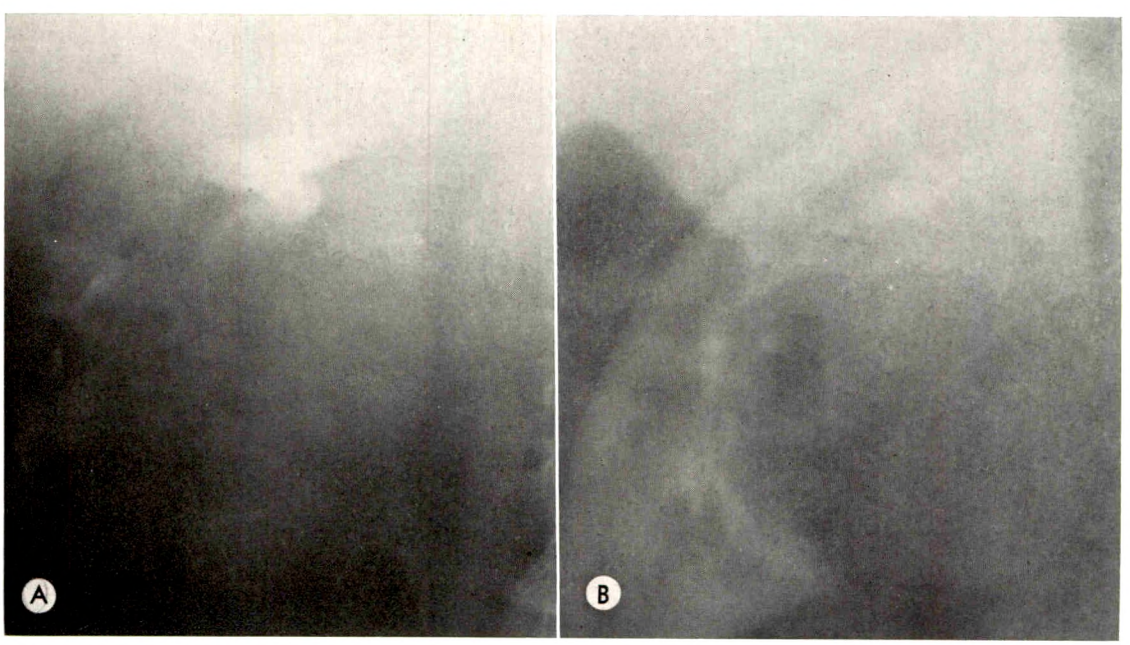


Fig. 11. Peripelvic cyst. (*A*) Excretory urogram. (*B*) Nephrogram phase of selective arteriography.

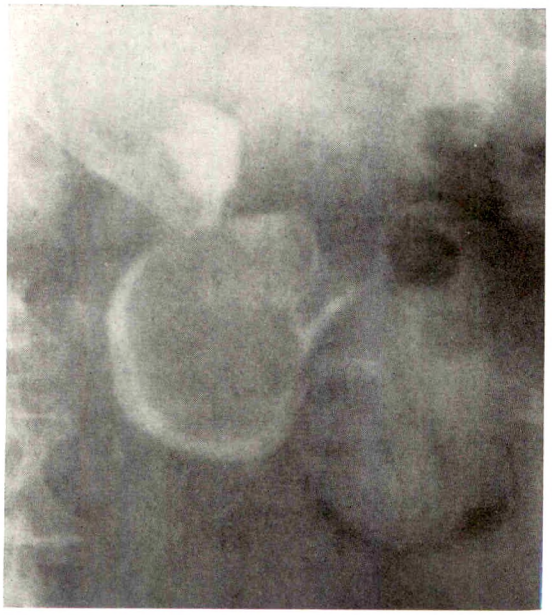


FIG. 12. Cystic hamartoma distorting the renal pelvis. (Reproduced with permission from: The Urinary Tract: Roentgen Diagnosis by Thornbury and Culp. Copyright 1967 by Year Book Medical Publishers.)

Of the 5 disease categories producing renal pelvic filling defects, neoplasms are the most lethal, with an average 5 year survival rate in most series of about 50 per cent. Transitional cell papillomas and transitional cell carcinomas account for 80–85 per cent of the primary malignant tumors of the renal pelvis.[2] They occur most frequently between the fourth and seventh decade and are more common in men by a ratio of 4 to 1. While the most common symptom is painless, intermittent, total hematuria, pain occurs when obstruction develops secondary to the lesion or clot formation. Spread of renal pelvic transitional cell carcinoma to the ureter and bladder in 40 per cent of the cases necessitates careful endoscopic and roentgenographic visualization of these structures when such a lesion is suspected. Roentgenographically, these neoplasms produce irregular, mottled filling defects which occasionally contain calcification. When the tumor involves one group of major or minor calyces, it will produce lo-

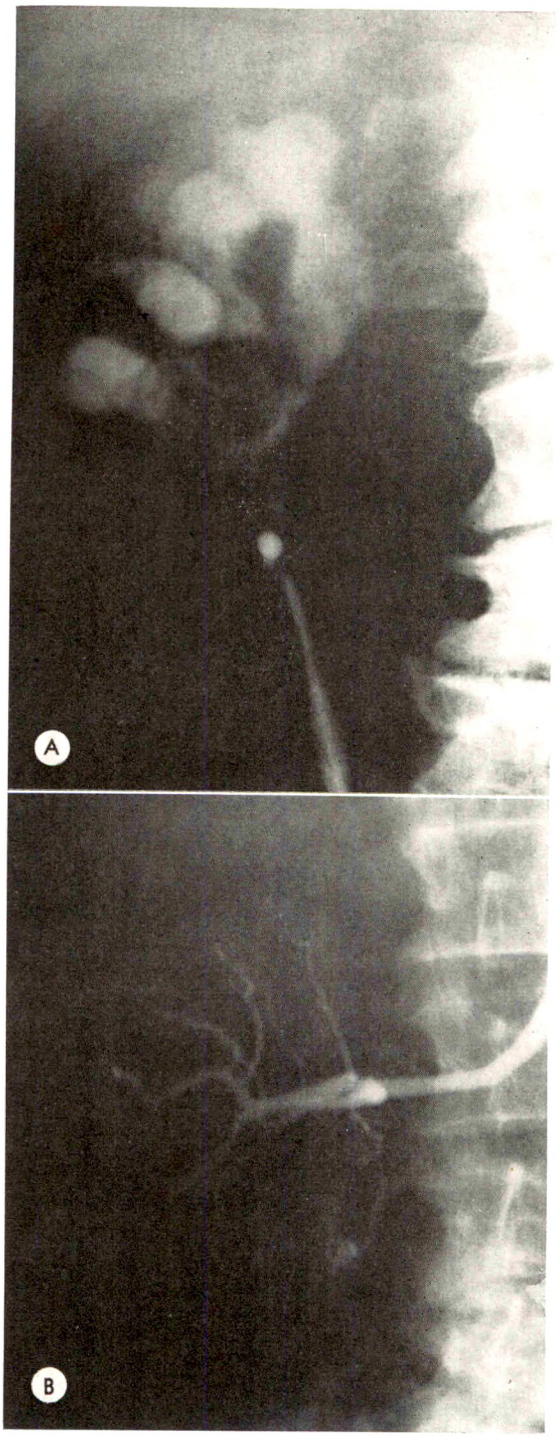


FIG. 13. (*A*) Retrograde pyelogram showing stenotic area of ureteropelvic junction and irregular defects in renal pelvis. (*B*) Selective renal arteriogram which shows "tumor vessels" in region of ureteropelvic junction. Pathologic diagnosis was transitional cell carcinoma.

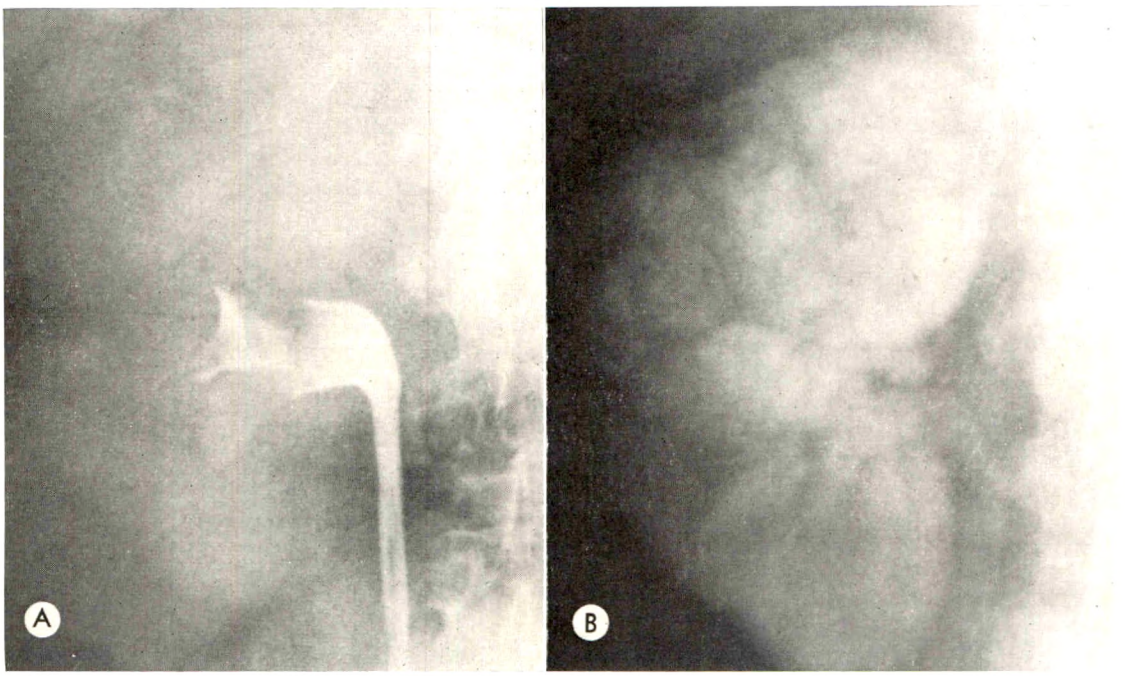


Fig. 14. Renal cell carcinoma. (*A*) Radiolucent defect in the renal pelvis on retrograde pyelogram. This was probably a combination of tumor and blood clot. (*B*) Nephrogram phase of selective renal arteriography demonstrates the extent of the tumor.

calized caliectasis with the remainder of the collecting system appearing normal (Fig. 13, *A* and *B*).

Approximately 15 per cent of malignant renal pelvic neoplasms are squamous cell carcinomas. These lesions are flat, more invasive, affect men and women equally, and are more frequently associated with stones or infection. The renal pelvic defects associated with this type of neoplasm are most

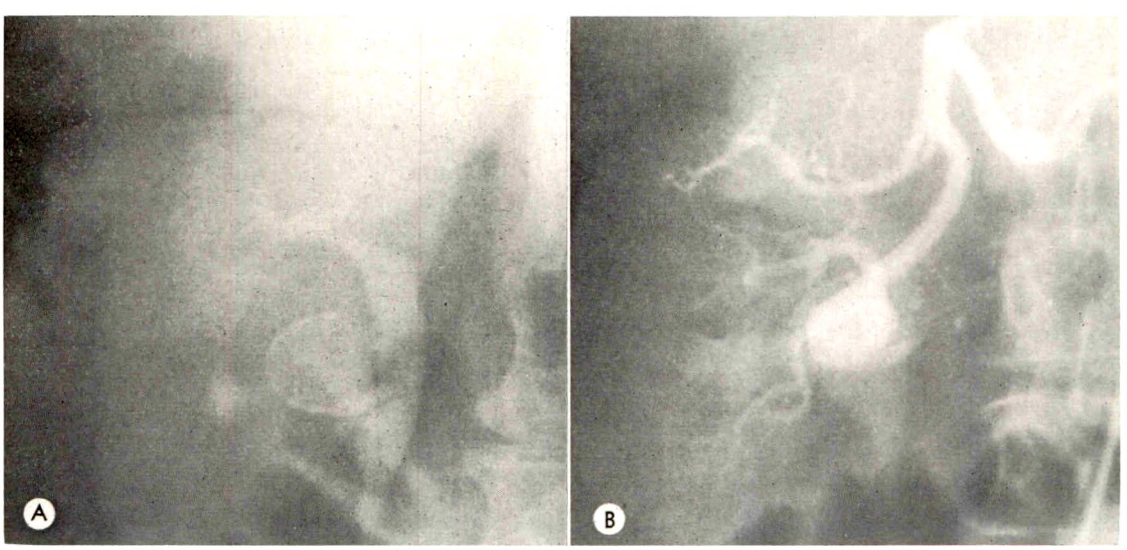


Fig. 15. (*A*) Calcified renal artery aneurysm causing defect in renal pelvis. (*B*) Selective renal arteriogram demonstrating the aneurysm.

frequently secondary to accompanying inflammatory changes and urolithiasis, since the lesion seldom projects far enough into the lumen to create intrapelvic defects.[3]

It is sufficient to say that adenocarcinoma of the renal pelvis is extremely rare and is associated with staghorn calculi. Therefore, it is not frequently involved in the differential diagnosis of pelvic filling defects on pyelography.[4]

Not all lucent neoplastic filling defects arise primarily in the pelvis. Renal cell carcinoma may invade the collecting system, producing clinical symptoms and urographic findings indistinguishable from a primary pelvic tumor, especially when there is no roentgenographically visible parenchymal mass (Fig. 14, *A* and *B*).

Neoplasms metastatic to the kidney from other organs are not uncommon due to the rich blood supply of the kidneys. The primary neoplasm most frequently resides in the lung, breast or stomach. The renal lesions are discovered primarily at autopsy and are seldom of clinical importance. However, they may produce a lucent filling defect and must be considered in a patient with symptoms of renal lesions when a known primary neoplasm exists elsewhere.

Renal artery aneurysms are rare but may also present as an extrinsic filling defect (Fig. 15, *A* and *B*).

SUMMARY

Radiolucent filling defects of the renal pelvis may occur because of either intrinsic or extrinsic causes.

A detailed history and complete physical examination should be the first steps in evaluating the patient. Obtaining the proper laboratory studies, especially examination of the urinary sediment, bacterial cultures of the urine, serum and urinary uric acid determinations will also be of assistance.

Technical and morphologic causes can be differentiated by close attention to urologic and radiologic techniques. Proper bowel preparation, adequate dosage of contrast media, application of abdominal compression bands over the lower abdomen, oblique projections of the kidney and nephrotomography will help solve technical and morphologic causes of radiolucent defects.

Selective renal arteriography and possibly ultrasonic scans are of assistance in differentiating mass lesions of the kidney.

By careful attention to these details most lesions can properly be diagnosed.

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Vol. 119, No. 4

BRUSH BIOPSY OF THE RENAL PELVIS*

A PRELIMINARY REPORT

By ROBERT C. BROWN, M.D.,† CHARLES E. HAWTREY, M.D.,‡
and EDNA E. PIXLEY, J.D., M.S.§
IOWA CITY, IOWA

BRUSH biopsy techniques have been perfected and are in general use in obtaining specimens from the bronchi.[3,4,9] This technique has also been used in the esophagus.[5] We have recently demonstrated this technique for use in the kidney.

MATERIAL AND METHOD

The kidney would appear to be an excellent organ for brush biopsy, in that the ureter is usually easy to catheterize. The problems of placing a catheter into the middle and lower pole calyces and the diameter of the ureter are the limiting factors in using brush biopsy techniques in the urinary system. The ureter will easily accept a No. 6 French diameter catheter; however, the standard ureteral catheter cannot be maneuvered in the renal pelvis. A small size, No. 5 French, Selector Catheter* and a small nylon brush (Fig. 1, *A* and *B*) have been developed which can be used to enter the middle and lower pole calyces. This catheter was originally developed for vascular catheterization. It has 4 small wires in the wall which attach the catheter tip to the proximal end. The catheter is joined to the control handle by a Luer Lok and the wires are attached to a flat metal plate by the small coil springs with nylon fittings. A "Joy stick" is attached to the flat plate. This allows the catheter tip to be easily controlled by movement of the operator's thumb. There are 2 adaptors in the handle; one which transmits the brush to the catheter, and the other which allows the injection of fluid into the catheter. The kidney may be flushed with saline for cell sampling or contrast media may be instilled through the catheter. Aspiration of urine from the renal pelvis prior to and after saline flushing affords another method of obtaining cells for cytology.

To date, we have used a catheter on 9 sets of urinary tracts which were obtained at autopsy. The kidneys, ureter and blad-

* Medi-Tech Inc., 372 Main Street, Watertown, Mass. 02172.

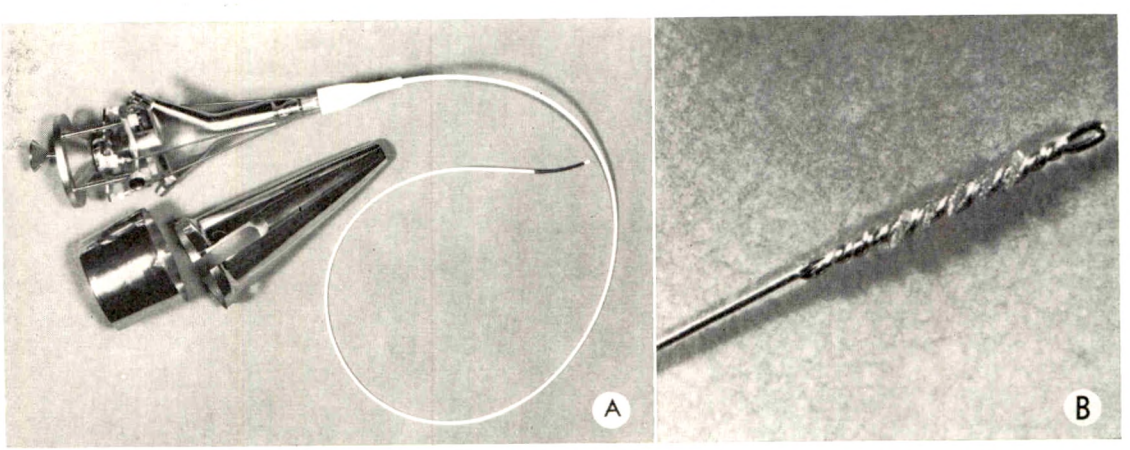


FIG. 1. (*A*) Selector catheter control handle. Four wires run longitudinally through the wall of the catheter connecting the tip to the "Joy stick." The covering to the apparatus is seen below the control handle. (*B*) Magnified view of the nylon brush. Note the blunt end of the brush.

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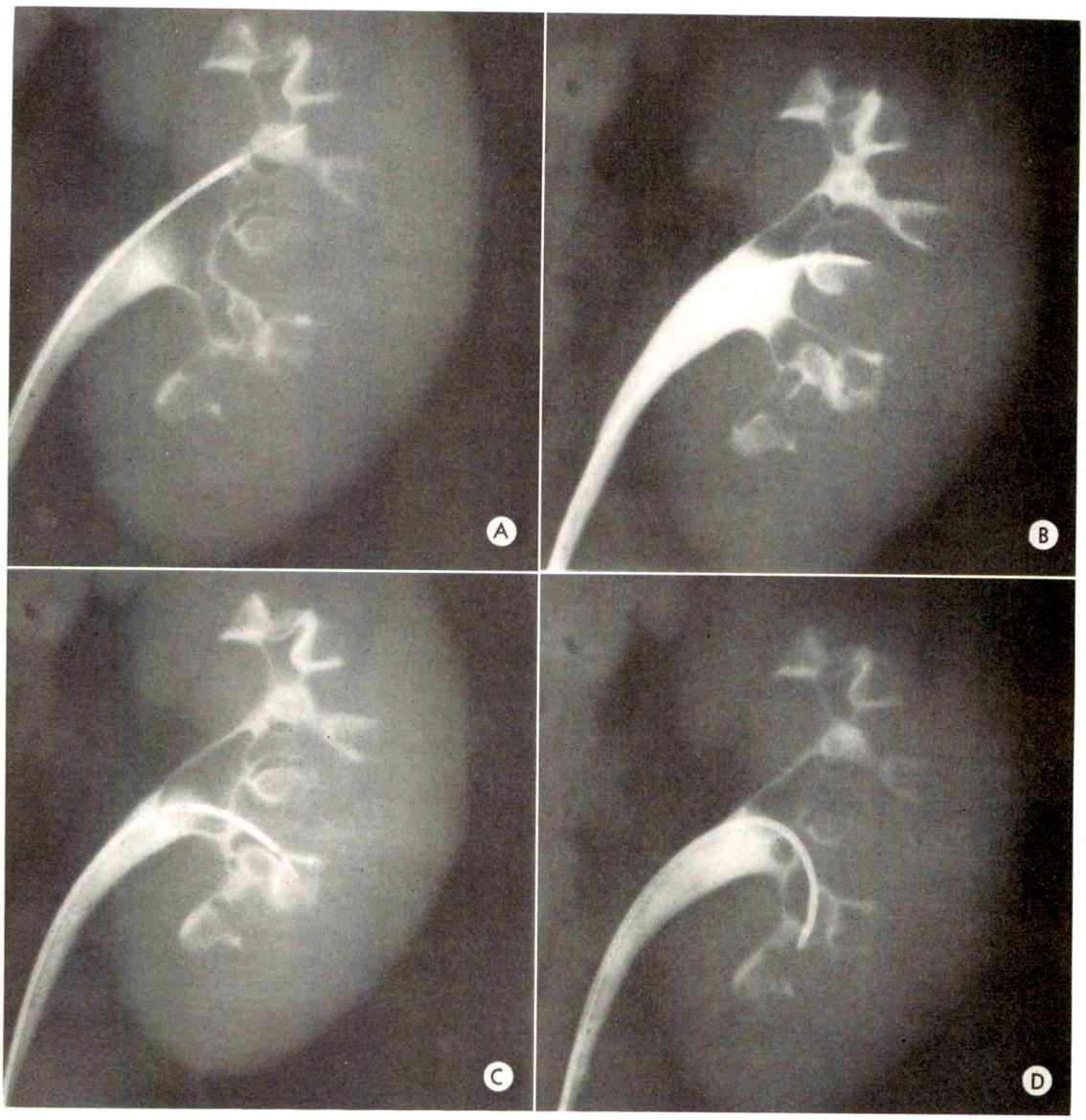


FIG. 2. (*A*) Catheter with brush extended into an upper pole calyx. (*B*) Catheter with brush extended into a middle pole calyx. (*C*) Catheter with brush extended into a lower pole calyx. (*D*) Catheter without brush extended in a lower pole calyx.

der were removed intact and after opening the bladder, the catheter was directed into the urethral orifice and up to the renal pelvis. The brush was introduced and both ureters and renal pelvis were brushed. Vigorous brushing was performed in an attempt to injure the ureter; however, the wire is of such fine caliber that sufficient pressure cannot be exerted to damage either the ureter or pelvis.

In 1 specimen we performed the procedure using fluoroscopic control (Fig. 2, *A–D*). The brush can be noted in the various calyces. In order to place the brush into the lowermost calyx, the tip of the catheter is introduced into the renal pelvis and the brush is extended beyond it. After directing the catheter into the lower pole, the brush is further advanced into the lower pole calyx. The brush must be pushed through the catheter tip prior to bending the catheter, because sufficient force cannot

be applied to the wire to advance the brush through the catheter tip once maximum angulation of the tip has occurred.

As soon as the specimen was obtained, the brush was pulled back into the tip and the catheter then removed. With the catheter removed from the bladder, the brush was again extended and washed in a 4 per cent formalin buffered with 2 per cent sodium acetate fixative solution. Specimens were then sent to the cytology section of the pathology department for further study.

The specimens were processed using mil lipore membrane filters (5 to 8 μ) with negative (vacuum) or positive (syringe) pressure.[8] For the vacuum method, the recommended negative pressure is 20 mm. of mercury or its equivalent, 11 inches of water. Conventional millipore apparatus (a collection funnel, ground glass base, filter flask and vacuum source) is used. Millipore filter monitors (0.45 μ) are used with a syringe instead of the above equipment for the positive pressure method.

Discernible pieces of tissue and/or blood clot can be removed *via* forceps and placed on a piece of lens paper. The lens paper is folded about the fragments, the encased specimen is placed in a previously numbered cassette, and the lid snapped in place. The cassette is then placed in 4 per cent formalin or Zenker's fixative for 20–30 minutes. This further fixes and hardens the fragments. The specimen is then processed as a routine cell button or tissue: embedded in paraffin, sectioned with microtome (3–4 μ thickness), slides dried and stained. Hematoxylin and eosin are routinely used as stains for cell block preparations but extra sections can be cut for special staining.

The filter preparations are stained according to conventional Papanicolaou staining techniques, but the time required in hematoxylin is decreased and 1-propanol is substituted for absolute alcohol before xylol. The cell types vary with their origin. Transitional cells are found in the bladder, ureter, renal pelvis and calyx. Simple columnar cells are found in the papillary ducts and lower portion of the collecting tubules. Simple cuboidal cells without brush borders are found in the distal convoluted tubules and the upper portion of the collecting tubules. Pyramidal cells with the brush borders are found in the proximal convoluted tubules. Transitional epithelial cells, stratified and pseudostratified columnar cells, and stratified squamous cells can be found in the urethra. Urinary casts of various compositions are seen in specimens from some of the patients.[1,2]

RESULTS

The urinary brush specimens contain many individual and cell aggregates of excellent morphologic detail.[1] In one brushing of the renal pelvis a variety of cell types were obtained (Fig. 3): epithelial cells of ureter and pelvic origin which normally show a marked variety of cell shapes; polyhedral cells with protoplasmic processes; elongated spindle cells; and cells of angular shaped processes. The shapes of these cells correspond to the actual shapes of their *in situ* growth pattern in the tissue. The protoplasmic processes represent intercellular bridges. One cell in the smear suggests the presence of a

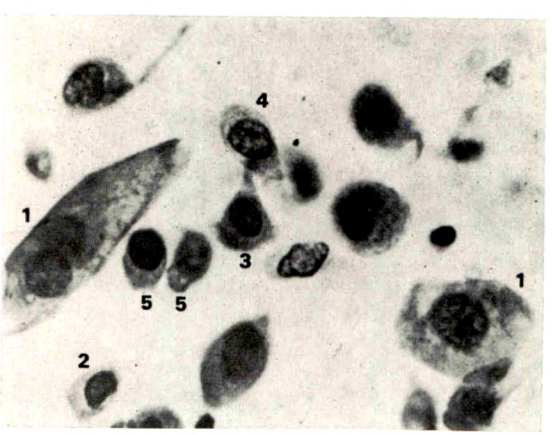


Fig. 3. Specimen taken from renal pelvis: (1) transitional cell probably from pseudostratified epithelium; (2) transitional cell from renal pelvis and ureter; (3) epithelial cell from ureter or renal pelvis; (4) columnar cell with brush border; and (5) small columnar cells probably from papillary ducts or lower collecting tubules.

brush border of a columnar epithelial cell, that is, a terminal bar with cilia. Small columnar cells are present which suggest an origin from papillary ducts or more likely from the lower portion of the collecting tubules.

The specimens are distinguished by the lack of cellular changes usually seen with cellular degeneration. The nuclei do not appear unduly large nor does the chromatin material appear as a homogeneous mass. Marked vacuolization of the nuclei or cytoplasm is not noted. Normal nuclear cytoplasmic ratios are apparent and the cytoplasmic borders of the cells are well defined. The nuclear membranes are clearly delineated and the nucleoli are identifiable. The red blood cells are intact, without evidence of crenation or distortion.

DISCUSSION

We have attempted to use this technique on 2 patients and found no difficulty in passing the Selector Catheter into the renal pelvis through the cystoscope. We also had no difficulty in maneuvering the catheter within the renal pelvis. Unfortunately, we do not have the capability of performing these maneuvers under fluoroscopic control and had to depend on taking overhead roentgenograms for catheter positioning. It is essential to have fluoroscopic guidance of the catheter tip and for visualization of the brush in order to place the brush within the proper calyx.

When performing this examination in a patient, maximum fluid intake with collection of urine for 12–18 hours after the brushing is recommended. Small fragments of tissue loosened but not removed by the brush are thus flushed into the urine sample by the usual physiologic process. Specimens should be transported immediately to the laboratory for processing. If delay is unavoidable, add an amount of 4 per cent buffered formalin equal to the amount of collected specimen. Intermix the formalin and specimen thoroughly. *Avoid contamination.*

SUMMARY

A new use of brush biopsy techniques is described for the renal pelvis.

A special French No. 5 catheter with matching nylon brush and the ability to perform retrograde ureteral catheterization under fluoroscopic control are essential to the successful completion of this technique.

Excellent cell preparations were obtained which should permit accurate cytologic interpretation.

No damage to the uroepithelium was found.

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GAS PRODUCING PERINEPHRIC ABSCESS*

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DURING the past 10 years we have had the unusual opportunity of observing 9 cases of gas producing perinephric abscess. Previously only isolated case reports have appeared in the literature.[2–8,11–15]

We therefore feel that a presentation of these cases with their roentgenographic findings, a consideration of the differential diagnosis, and a review of the literature is of value.

Perinephric abscess is a difficult diagnosis both clinically and roentgenographically. However, the appearance of extraluminal gas surrounding the kidney is highly suggestive of the diagnosis.

ETIOLOGY

The etiology of perinephric infection may be either antecedent intrarenal disease or disease of extrarenal origin.

A. Antecedent urinary tract disease
   1. Urinary tract infection
   2. Obstructive uropathy
   3. Trauma to kidney, ureter or bladder
   4. Instrumentation, particularly in removal of ureteral calculi

B. Extraurinary infection with direct or hematogenous spread
   1. Superficial infection such as furuncle, carbuncle, paronychia or wound infection
   2. Deep infection such as osteomyelitis, nasopharyngitis, tonsillitis, otitis media, endocarditis, perforated ulcer, diverticulitis of the colon and pancreatitis.

Six of the 9 cases were diabetic, 8 were females. The most usual cause of gas producing perinephric abscess is infection with *E. coli*, *Aerobacter aerogenes*, or *Pseudomonas aeruginosa*.

PATHOLOGY

The pathogenesis of perinephric abscess is either a breaking through of the renal fascia or extension through an open cortical defect in it. However, it does not usually spread to the contralateral side because the medial fascia surrounding the kidney is closed and the spine and great vessels act as a natural deterrent. This inflammatory process may extend around the entire kidney, but it is usually most pronounced on the dorsal and inferior aspect, where the renal fascia is open and the surrounding tissues offer less resistance.

CLINICAL FINDINGS

The patient usually presents a history suggestive of a septic course, with dysuria, chills, fever and flank or abdominal pain the predominant symptoms.

Physical findings include flank tenderness, abdominal muscle guarding, abdominal mass, crepitation in the region of the affected kidney and retraction of the lumbar spine from the affected side. Less common and less specific findings are jaundice, ascites, unilateral leg edema, limitation of hip motion, pleurisy and dehydration. Nevertheless, the course of illness may be insidious and chronic.

Laboratory studies generally reveal leukocytosis, elevated sedimentation rate and pyuria. However, it must be emphasized that sterile urine cultures do not exclude the diagnosis, and blood cultures may be positive.

ROENTGENOGRAPHIC FINDINGS

The roentgenographic findings in this disease are most helpful. The presence of

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extraluminal gas around or inside the kidney and in the retroperitoneal space is nearly pathognomonic. As pointed out, above the renal fascia is a collagenous, thin, elastic connective tissue sheath of medium or dense consistency that invests the kidneys and adrenals fairly closely. Perirenal infection may be either diffuse and poorly defined or in the form of a localized mass. The exudate usually localizes in the dorsal lateral perirenal fat near the lower pole because the infection follows the path of least resistance abetted by gravity in the supine position. The kidney is therefore usually rotated about its vertical axis. The kidney in anteroposterior projection may appear larger because of magnification. A lateral roentgenogram will document its anterior displacement. As exudate increases, it tends to seek the infrahilar area extending inferolaterally along the ureter. Lateral displacement of the inferior renal pole will result.

According to Evans *et al.*[3] the classical appearance of perinephric abscess depends upon the anatomic relationship of the perinephric space (Fig. 1). Based on this the characteristic roentgenographic appearance includes:

A. Obliteration of the perirenal space
B. The fluid density axis is usually vertical
C. The kidney is usually displaced anteriorly, medially and superiorly in the acute case, and in the chronic case laterally
D. Obliteration of the upper half of the psoas muscle shadow
E. Displacement of descending duodenum or duodenal-jejunal junction anteriorly
F. Ascending and descending colon is displaced anteriorly and laterally
G. Flank stripe is preserved
H. Loss of visualization of hepatic and splenic angles because of obliteration of posterior retroperitoneal fat on which the signs are dependent.

These findings according to Evans *et al.*[3]

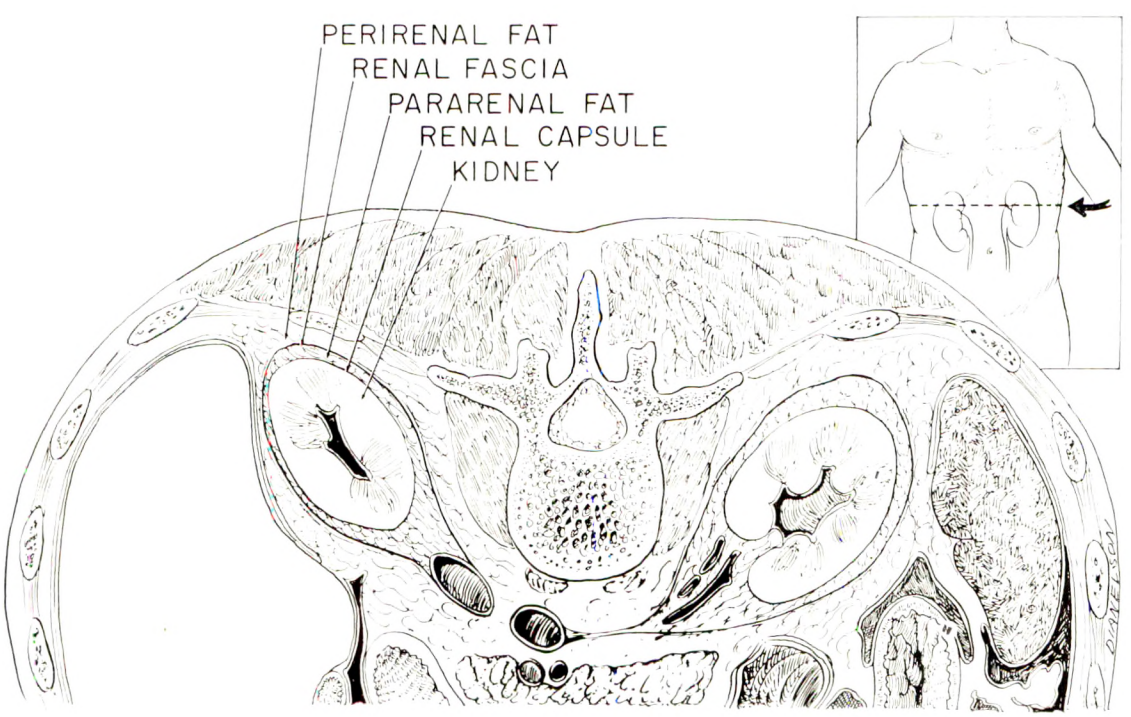


FIG. 1. Anatomic cross section demonstrating closure of the fascia around the great vessels.

are limited to the anatomic perinephric space. However, our case material demonstrates that infection can frequently extend through the perinephric space and into the anterior and/or posterior pararenal spaces surrounding the kidney.

Other findings are delayed excretion on intravenous pyelography, fixation and decrease in the mobility of the kidney, haziness and enlargement or deformity of the renal outline, retraction of the lumbar spine from the involved side, elevation of the diaphragm with or without pleural effusion.

REPORT OF CASES

Case I. C.J., a 61 year old Caucasian female, known diabetic taking 40 units of NPH insulin daily, was admitted in coma with a 3 day history of increasing nausea, fever and lethargy.

Physical examination revealed a dehydrated comatose patient, with Kussmaul respirations. Control of ketacidosis was obtained and the patient began to complain of right flank pain and moderate right costovertebral angle tenderness was present. No abdominal findings were noted. White blood cell count was 17,000 per mm.^{3}, with a left shift. Urine culture and sensitivity revealed *Pseudomonas aeruginosa* and *Aerobacter aerogenes*.

Intravenous pyelography, nephrotomography and right renal arteriography (Fig. 2; 3; and 4) were performed. These studies revealed right renal enlargement, extraluminal gas, obliteration of the psoas muscle shadow and displacement of the pericapsular artery. The patient was subsequently taken to surgery and 250 cc. of purulent material was drained from a perinephric abscess with a positive culture of *Pseudomonas* being obtained. The patient's recovery was uneventful.

Case II. S.T., a 57 year old Negro female, known diabetic, was admitted with left flank pain and dysuria of 5 weeks' duration. For the immediate 3 days prior to admission the pain had increased and was accompanied with nausea and emesis. Physical examination revealed a temperature of 101°F., diffuse abdominal tenderness without a palpable mass and bilateral costovertebral angle tenderness, greatest on the left.

Intravenous pyelography was consistent with a left perinephric abscess (Fig. 5). The colon was displaced by a retroperitoneal mass (Fig. 6). At surgery 300 cc. of green pus was drained. Culture of the material revealed *Klebsiella* and *Aerobacter*. Recovery was uneventful.

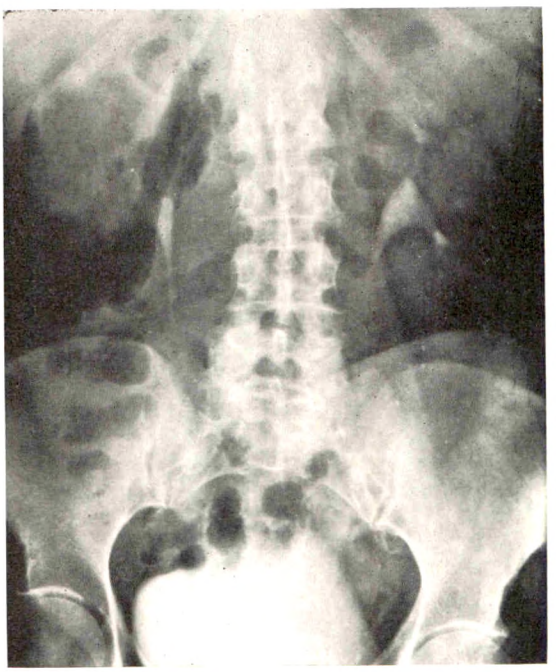


Fig. 2. Case I (C.J.). Intravenous pyelogram shows blurred right renal enlargement, extraluminal gas with displacement of the inferior pole of the kidney laterally and obliteration of the psoas muscle shadow.

Case III. P.G., a 45 year old white female, known diabetic, was admitted with abdominal pain and right flank pain. Physical examina-

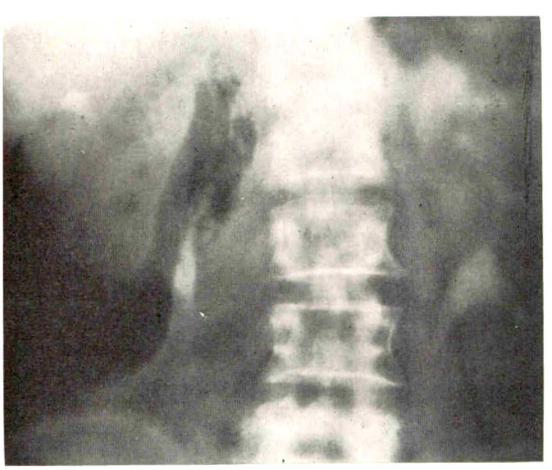


Fig. 3. Case I (C.J.). Nephrotomogram demonstrates the right kidney displaced medially.

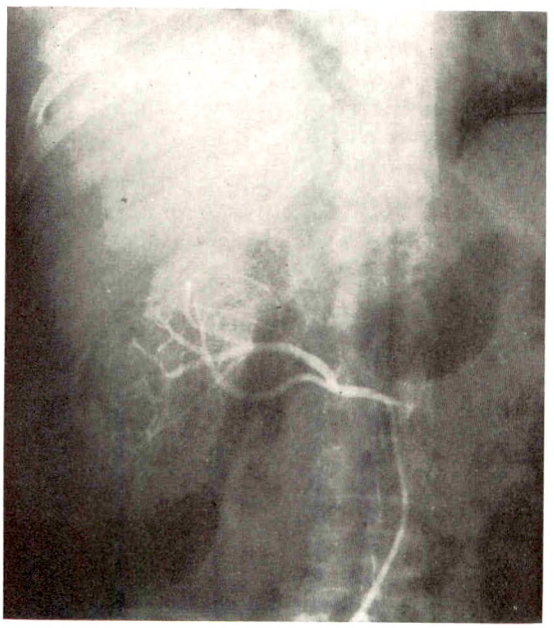


Fig. 4. Case I (C.J.). Selective renal arteriogram reveals lateral displacement of the pericapsular artery; the loss of the hepatic angle is well demonstrated.

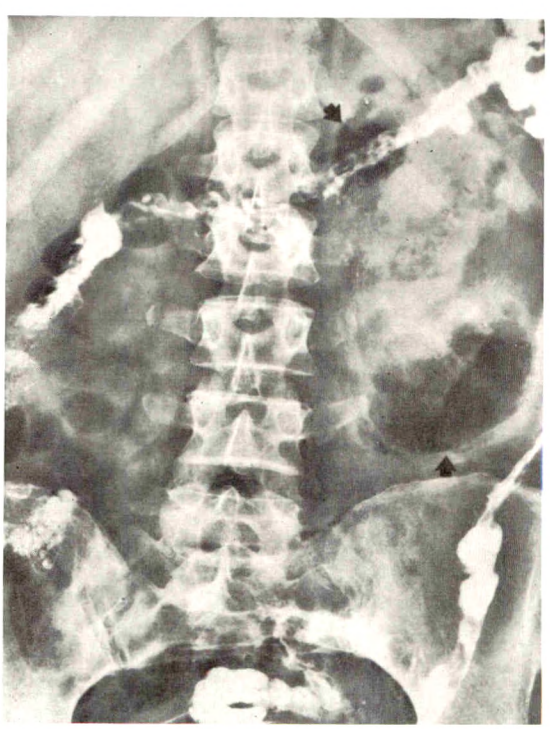


Fig. 5. Case II (S.T.). Barium enema examination shows the colon displaced laterally by extraluminal gas collection. The renal pelvis is outlined by gas and there is loss of splenic angle.

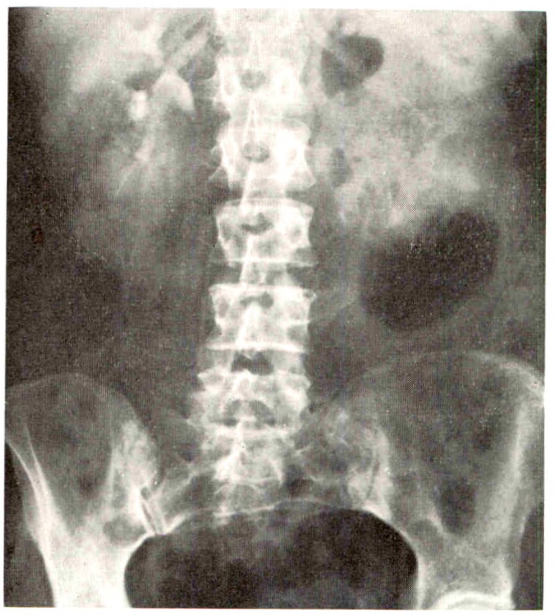


Fig. 6. Case II (S.T.). Intravenous pyelogram shows large extraluminal gas collection in the left retroperitoneal space with inferior convexity. There is gas present in the pelvis, the kidney is displaced medially and the psoas muscle shadow is obliterated.

tion showed a temperature of 101°F., diffuse abdominal tenderness and right flank tenderness. Following admission the fever increased to 104°F. and her diabetes became more difficult to control.

Intravenous pyelography revealed bilateral nephrogram only, with extraluminal gas about the right kidney (Fig. 7). At emergency surgery 200 cc. of purulent material was evacuated from the perinephric space with the culture revealing *Aerobacter aerogenes*. The patient expired 2 days later. Autopsy was not obtained.

Case IV. E.P., a 63 year old Negro female, known diabetic with a history of recurrent right-sided pyelonephritis and left renal calculi, was admitted with left flank pain, nausea, emesis and fever of 15 hours' duration. Recent urinary tract symptoms were denied.

Physical examination revealed a fever of 103°F., left flank tenderness and minimal left abdominal tenderness to deep palpation. Urine studies demonstrated microscopic pyuria, and the white blood cell count was 24,000 per mm.^{3}, with 90 per cent polymorphonuclears. Because of the calculi seen on the abdominal roentgenogram (Fig. 8), a cystoscopy and left ureteros-

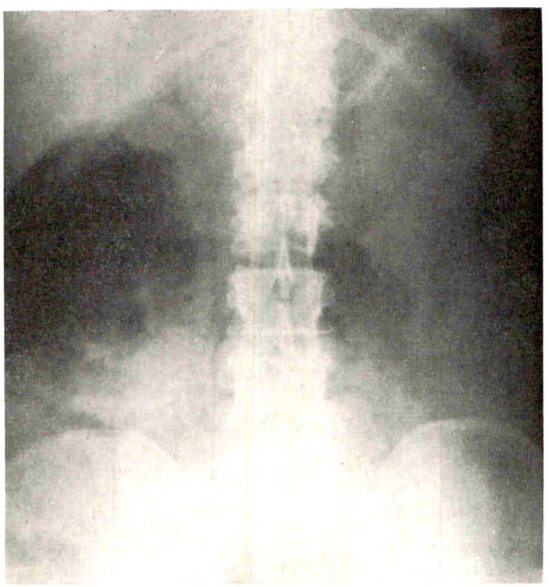


Fig. 7. Case III (P.G.). Intravenous pyelogram shows faint nephrogram with large right renal shadow and extraluminal collection of gas with inferior convexity. The psoas muscle shadow and the hepatic angle are lost.

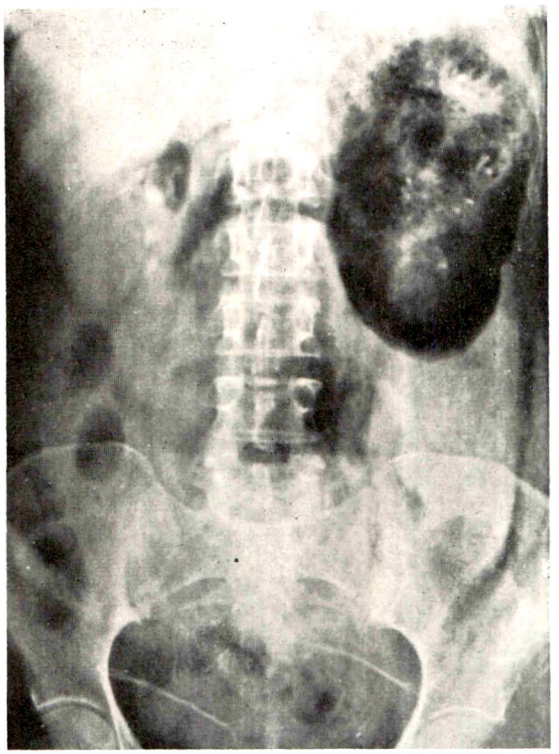


Fig. 8. Case IV (E.P.). A flat roentgenogram shows nephrocalcinosis and large left perinephric abscess surrounding the kidney. The properitoneal fat line is outlined by gas inside the posterior pararenal space. A calculus in the distal ureter had been removed. The diagnosis at autopsy was perinephric abscess and necrotizing papillitis (calcification in area of sloughed papillae).

tomy were performed with the removal of 2 calculi.

Postoperatively the patient experienced a stroke.

Subsequent autopsy revealed a left perinephric abscess with 400 cc. of purulent material. Culture grew *Aerobacter aerogenes*. There was necrotizing papillitis accounting for the calcification of the left kidney.

Case V. E.B., an 85 year old Negro female, entered the hospital with a right flank mass and tenderness of uncertain duration. A past history of right hydronephrosis and nephrolithiasis was elicited. Recently she had noted fever and anorexia, but no more definite history was obtainable. Flat roentgenogram of the abdomen demonstrated right extraluminal gas, and staghorn calculus (Fig. 9).

Physical examination revealed temperature of 100°F. and the presence of a rounded rather fluctuant erythematous mass projecting from the right flank. Aspiration of the mass gave purulent material with culture for *Proteus mirabilis* obtained. The patient later expired and no postmortem examination was obtained.

Case VI. O.J., a 65 year old Negro female with a 12 year history of Orinase treated diabetes mellitus, was admitted with a 3 day history of increasing weakness, abdominal pain, nausea and emesis.

Physical examination showed an obese elderly female, mildly diaphoretic with minimal epigastric and left upper quadrant abdominal tenderness.

Admitting serum glucose was 774 mg. per cent with urinalysis revealing 4+ glucose, 2+ acetone and 20–30 white blood cells per high power field.

Following control of the patient's diabetes her temperature increased to 38°C. and she reported left flank tenderness to palpation.

Intravenous pyelography revealed a gas pattern outlining the medial and inferior border of the left kidney (Fig. 10). Left renal exploration and drainage of the abscess were accomplished. The culture grew *Aerobacter*. Recovery was uneventful.

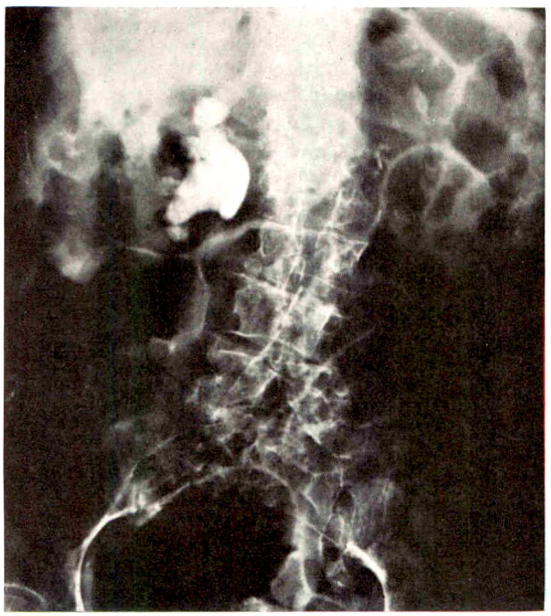


FIG. 9. Case V (E.B.). Flat roentgenogram demonstrates a right staghorn calculus displaced medially by extraluminal gas, and loss of hepatic angle.

CASE VII. H.J., a 49 year old white male, was admitted with a 36 hour history of lower abdominal pain and inability to urinate. Physical examination showed a temperature of 101°F., a right lower quadrant mass and penile edema. Laboratory studies revealed a blood urea nitrogen of 67 mg. per cent, creatinine of 5.4

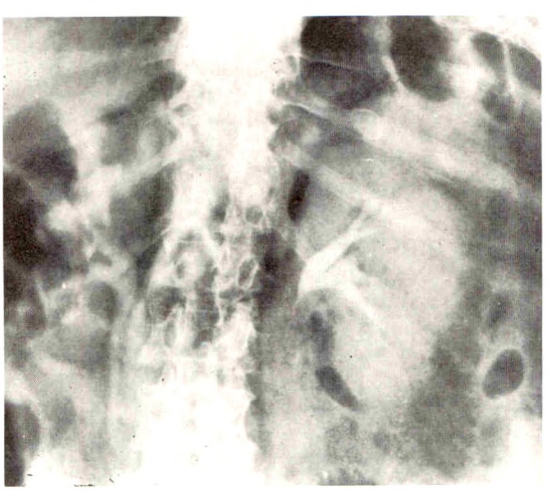


FIG. 10. Case VI (O.J.). Intravenous pyelogram shows left medial renal displacement, extraluminal gas and obliteration of the psoas muscle shadow and the splenic angle.

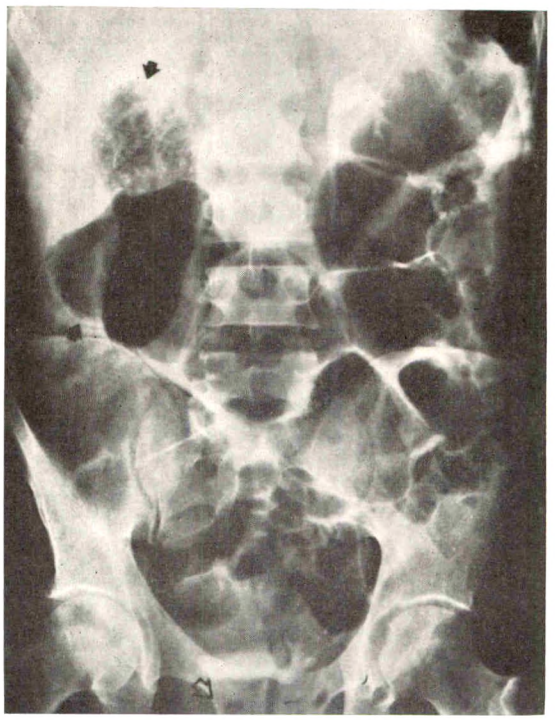


FIG. 11. Case VII (H.J.). Intravenous pyelogram shows nonfunctioning right kidney, extraluminal gas inferiorly, medially and in the right inguinal area.

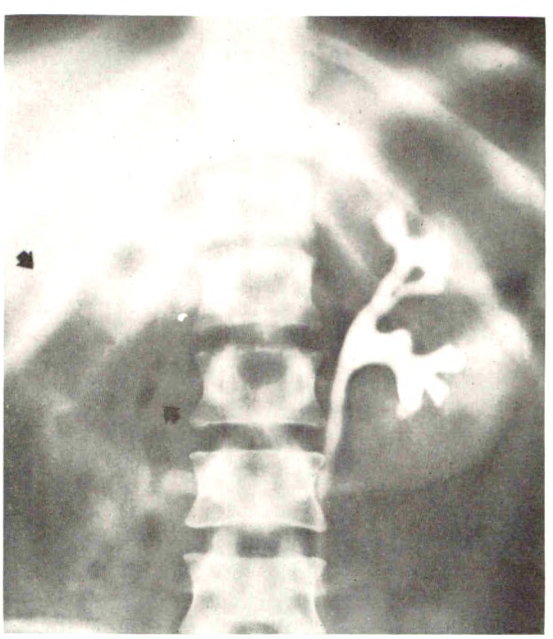


FIG. 12. Case VIII (R.O.). Intravenous pyelography shows nephrotomogram with nonfunctioning right kidney and extraluminal gas surrounding it. The psoas muscle shadow and hepatic angles are lost.

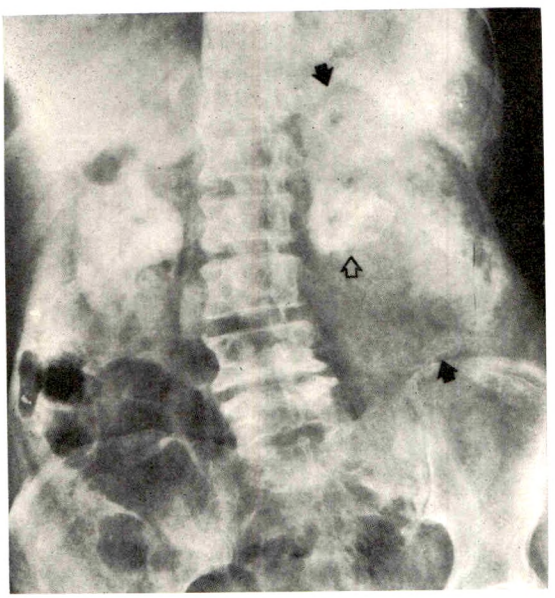


Fig. 13. Case IX (G.F.). Intravenous pyelogram shows left pelvic calculus displaced medially. There are several small parenchymal calcifications and extraluminal gas with loss of the psoas muscle shadow.

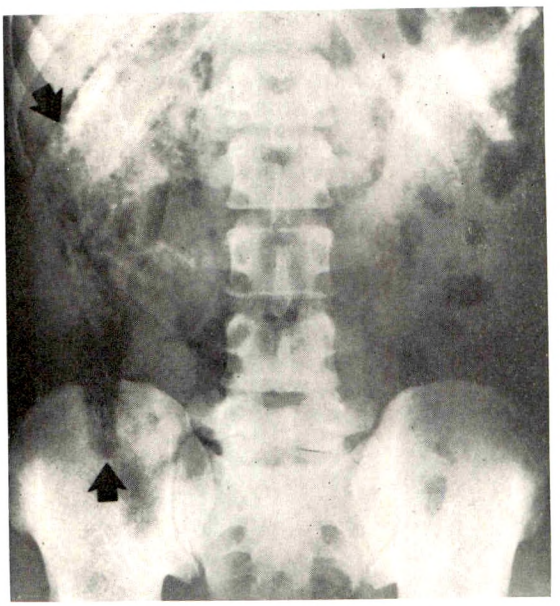


Fig. 14. Traumatic perforation of the second portion of the duodenum. Extraluminal gas outlines the psoas muscle and the right kidney following blunt abdominal trauma with complete transection of the second portion of the duodenum.

mg. per cent, and a white blood cell count of 26,700 per mm.^{3}. Urine culture and sensitivity gave *Paracolon* and *Pseudomonas*. Cystotomy was performed and an incision and drainage of an abscess of the penis and scrotum was done. The patient improved temporarily. However, 10 days later intravenous pyelography revealed a right retroperitoneal abscess with nonfunction of the right kidney and extraluminal gas, along with gas in the right inguinal area, suggesting ascending infection (Fig. 11).

Case VIII. R.O., a 44 year old Negro female, with juvenile onset of diabetes maintained on 40 units NPH insulin daily, was admitted with 1 week history of chills, fever, nausea, emesis and right flank and abdominal pain.

Examination revealed fever of 102°F., right flank and right deep abdominal tenderness. White blood cell count was 18,300 per mm.^{3}, and marked pyuria was present. Intravenous pyelography, and nephrotomography demonstrated nonfunctioning right kidney with extraluminal gas and obliteration of the psoas muscle shadow (Fig. 12).

At surgery 300 cc. of purulent material was drained through a right flank incision from the perinephric space. Culture was positive for *E. coli*. The patient recovered.

Case IX. G.F., a 72 year old white female, admitted with left flank pain of several days' duration, temperature of 102° F. and left costovertebral angle tenderness. Past history revealed recurrent urinary tract infections and left-sided hemiplegia secondary to a previous stroke. No further information was available.

Laboratory studies revealed a white blood cell count of 26,400 per mm.^{3}, with left shift, marked pyuria and urine culture of *E. coli*.

Intravenous pyelography demonstrated a large left staghorn pelvic calculus with multiple small calcifications and faint collection of extraluminal gas laterally and inferiorly (Fig. 13).

At surgery a left nephrectomy and evacuation of 300 cc. of purulent material from the perinephric space was carried out. Recovery was uneventful.

DIFFERENTIAL DIAGNOSIS

In consideration of the diagnosis of gas producing perinephric abscess, the presence of extraluminal gas in the upper quadrants has been demonstrated in the following conditions: 1. traumatic duodenal perforation (Fig. 14); 2. rectal perforation (Fig.

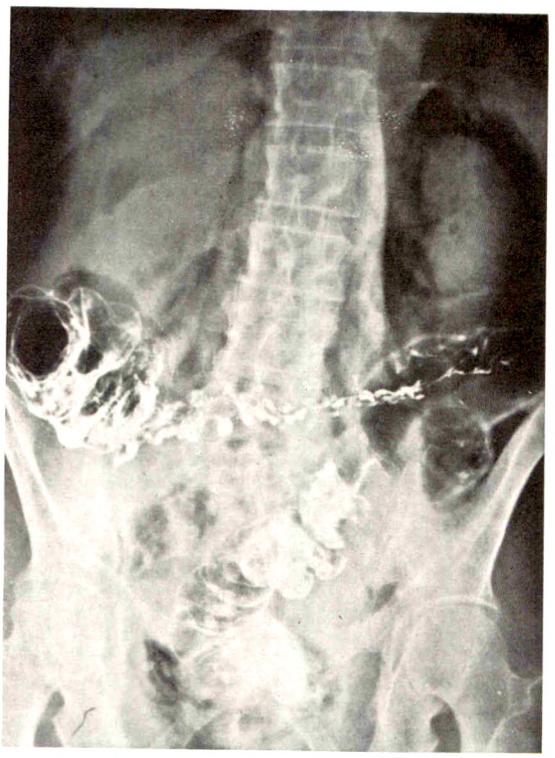


FIG. 15. Traumatic perforation of the rectum following a barium enema examination reveals the extraluminal gas completely outlining the left kidney, and some streaks of air outlining the right kidney and extending up along the crus of the right diaphragm. There is intramuscular perforation with extravasation of the barium at the level of the rectum.

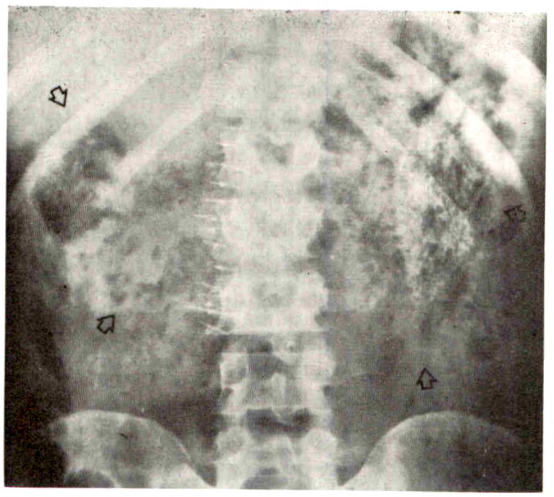


FIG. 16. Emphysematous pancreatitis. Diffuse collection of extraluminal gas traversing the entire upper abdomen in the projection of the pancreas. The bilaterality as well as the configuration conforming to the pancreas is highly suggestive.

15); 3. pancreatic abscess; 4. emphysematous pancreatitis (Fig. 16); 5. lesser sac abscess (Fig. 17, *A* and *B*); 6. intraperitoneal abscess; 7. postoperative perinephric hematoma (Fig. 18, *A* and *B*); 8. interposition of colon; 9. flank abscess; 10. portal vein gas; 11. intrahepatic abscess; and 12. abdominal wall abscess.

The only conditions which as a practical

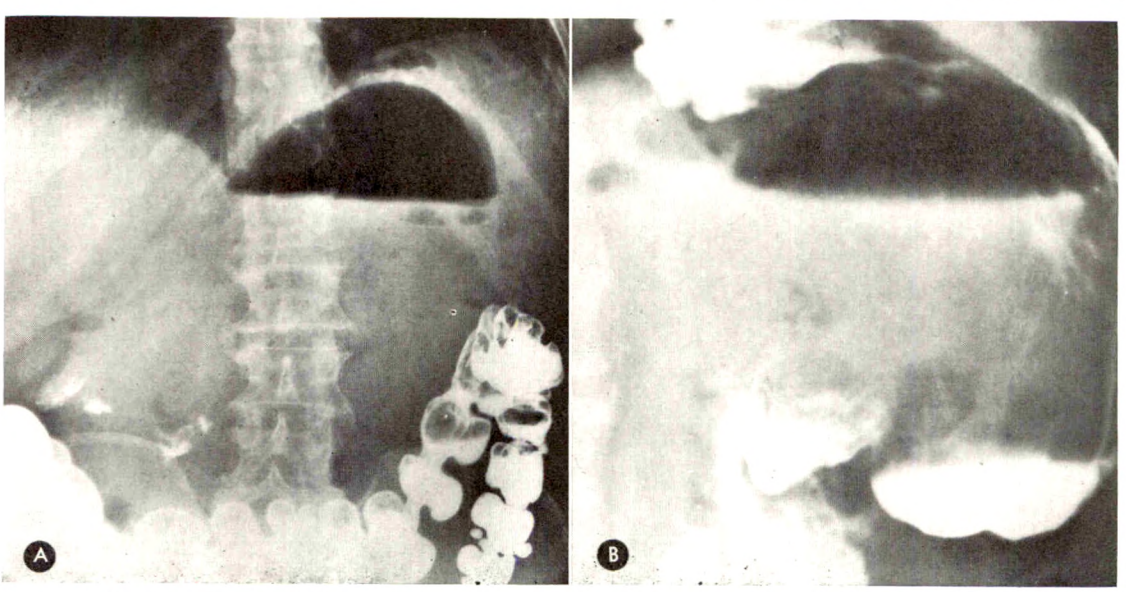


FIG. 17. (*A* and *B*) Lesser sac abscess, following perforation of a gastric ulcer with huge air fluid level extending from the left side slightly across the midline. The diaphragm is not reached. The stomach is compressed anteriorly by the air fluid collection. The colon is displaced downward.

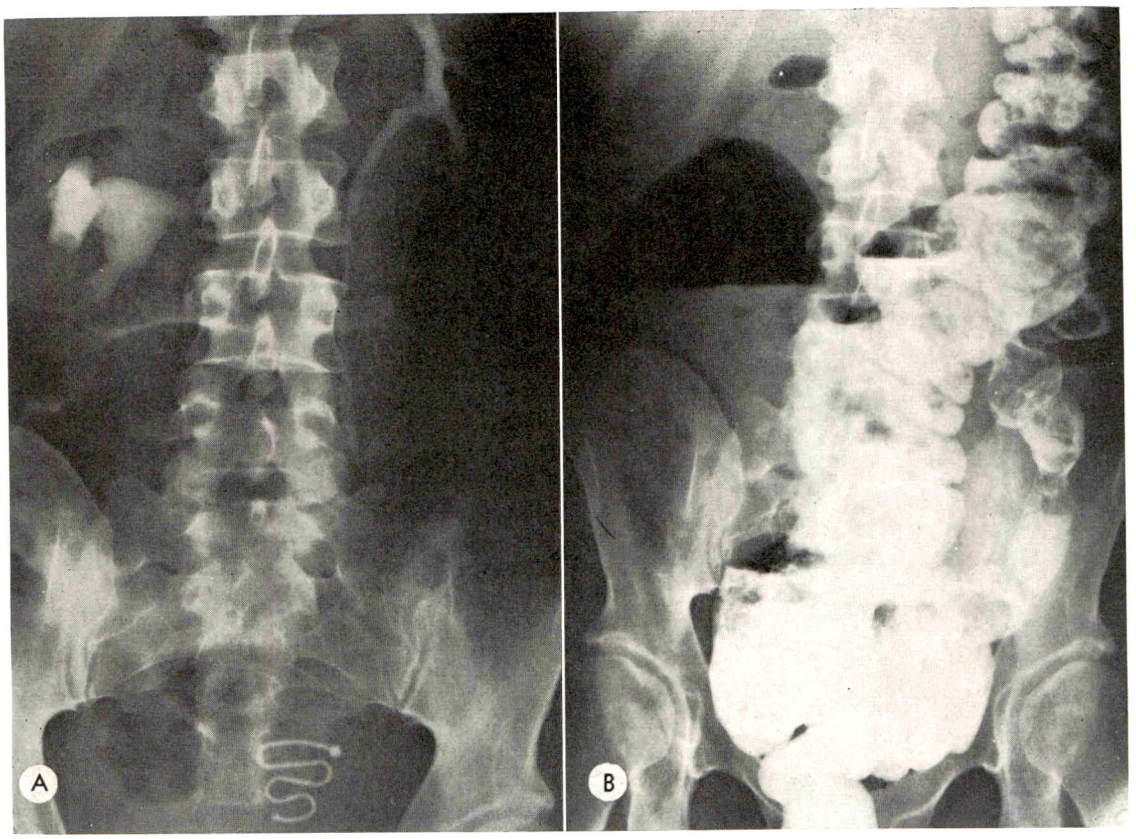


Fig. 18. Liquefying perirenal hematoma following a heminephrectomy for an angiomyolipoma of the kidney. The patient developed pain in the right upper quadrant and a falling hematocrit.

(*A*) Intravenous pyelogram reveals a collection of extraluminal gas around the right kidney, with extension inferiorly compressing an intrauterine conception device to the left. (*B*) Upright roentgenogram following barium enema examination reveals the large air fluid level displacing the cecum to the midline. The presumptive diagnosis was a gas containing perinephric abscess; however, at surgery a liquefied hematoma of about 1,000 cc. was found in the retroperitoneal space which had apparently resulted from hemorrhage, with air being sucked in through the flank drains. Courtesy of Dr. Eugene Borchart, Lutheran General Hospital, Park Ridge, Illinois.

consideration can cause diagnostic difficulty are those in the retroperitoneal space.

Both traumatic duodenal[16] (Fig. 14) and rectal (Fig. 15) perforation result in dissection of air into the retroperitoneal space. Both may dissect for considerable distance; however, the duodenal findings are usually on the right side, and the rectal air may be around both or either kidney.

In acute pancreatitis with abscess or saponification there is a mottled pattern of radiolucencies which may extend beyond the pancreas (Fig. 16). This "abdominal fat necrosis sign"[1] makes the diagnosis more readily ascertained.

With lesser sac abscess an upright roentgenogram reveals an air fluid level in the left upper abdomen extending slightly over the midline, but not reaching the diaphragm[8] (Fig. 17, *A* and *B*). The stomach is displaced anteriorly and the colon inferiorly. An intraperitoneal abscess in an upright roentgenogram will appear similar to a lesser sac abscess, but will be in the midline beneath the central tendon of the diaphragm. This should not be confused with retroperitoneal gas.

Postoperative perinephric hematoma is seen to correspond closely to that of an abscess. Liquefaction of the hematoma and air entering from the drain site is the apparent etiology (Fig. 18, *A* and *B*).

SUMMARY

Nine cases of gas producing perinephric abscess are reported, with an anatomic and pathologic correlation of the material.

A differential diagnosis of extraluminal gas is presented.

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THE ANGIOGRAPHIC DEMONSTRATION OF PARTIALLY INFARCTED PHEOCHROMOCYTOMAS OF THE ADRENAL GLAND*

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and JULIANNE L. IMPERATO, M.D.‡
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THE diagnosis of pheochromocytoma is not made by angiography, but rather by biochemical analysis of catecholamines and catecholamine metabolites in the urine. Nonetheless, angiography plays a major role in the management of patients with this disease. The main purpose for performing angiography, therefore, is not diagnostic but rather to localize the tumor. It is well known that 10 per cent of these tumors can be located outside of the adrenal glands and at times may be multiple.[7] Inasmuch as pheochromocytomas are rather vascular, the angiographic procedure of choice is arteriography.[1,2,4] In most instances, a simple flush aortogram reveals the tumor readily; however, at times, selective arteriography must be performed. Some of these tumors cannot be shown by arteriography: Rossi and coworkers,[7] in reviewing 99 cases of pheochromocytomas, found 11 cases which were not localized by arteriography. Inadequate technique may be responsible for such failure; however, partial infarction of the tumor might also be incriminated.

The purpose of this paper is to make the angiographer aware that infarction of pheochromocytomas does occur and can result in a false negative arteriogram, that in spite of necrosis these tumors still secrete high levels of catecholamines, and that they can be localized by adrenal venography.

REPORT OF A CASE

A 47 year old female presented at another hospital with a 3 month history of profuse sweating, nervousness and fatigue. Upon admission, hypertension in the range of 170/110 to 200/140 mm. Hg was noted. The patient was the mother of 11 children and had no previous history of hypertension. During her hospitalization, she developed a left hemisphere cerebrovascular accident. The physical examination was not contributory. The laboratory findings revealed 24 hour urinary VMA of 36 mg./24 hr. (normal 1–5 mg./24 hr.). The 24 hour total catecholamine measured 2,600 μg. (normal up to 120 μg.). A Regitine test was performed using 5 mg. of phentolamine HCl which resulted in a drop of the blood pressure from 190/125 to 80/30 mm. Hg within 1 minute. Eight minutes after injection of the medication, the blood pressure returned to 180/115 mm. Hg. On the basis of these tests, pheochromocytoma was diagnosed. The patient was placed on dibenzyline 15 mg. p.o., q. 12 h. and responded typically by becoming normotensive. The chest roentgenogram was normal and the electrocardiogram revealed left ventricular hypertrophy with strain.

The patient was transferred to the New York Hospital where the diagnosis of pheochromocytoma was again confirmed. On November 1, 1972, angiographic evaluation was performed with the patient receiving the usual dibenzyline medication. Aortography, bilateral selective renal arteriography, celiac, superior mesenteric and selective inferior phrenic arteriographies were all negative. The patient was returned the following day to the Department of Radiology, where bilateral selective adrenal venograms were obtained. At the time of adrenal venography, blood was sampled high and low in the inferior vena cava as well as from the left and right adrenal veins. Epinephrine and norepinephrine levels were obtained according to a method to be reported elsewhere, and demonstrated a step up of norepinephrine at the level of the right adrenal gland. The left adrenal

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venogram was interpreted as being normal (Fig. 1), whereas the right adrenal venogram revealed the presence of an avascular mass within the core of the right adrenal gland (Fig. 2).

On the basis of the above results, the patient was subjected to surgery and the right adrenal gland was removed with a tumor measuring 3 cm. in diameter and weighing 10 gm. The tumor appeared encapsulated and had a necrotic center (Fig. 3). Histologic examination confirmed the diagnosis of pheochromocytoma.

DISCUSSION

The importance of arteriography in the localization of pheochromocytoma does not have to be reiterated. On the other hand, one must realize that approximately 10 per cent of these tumors cannot be demonstrated by this technique. Previous authors felt that the majority of tumors which were not angiographically demonstrable were missed because of technical factors.

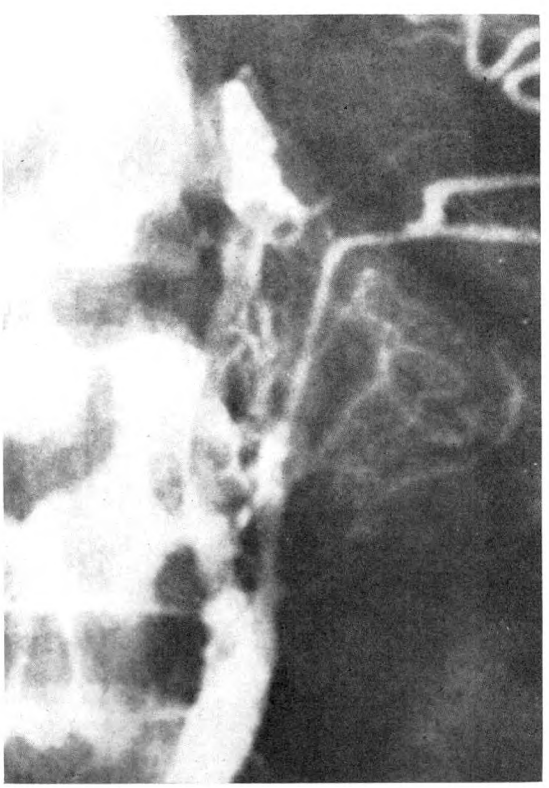


FIG. 1. Left adrenal venogram showing a normal gland.

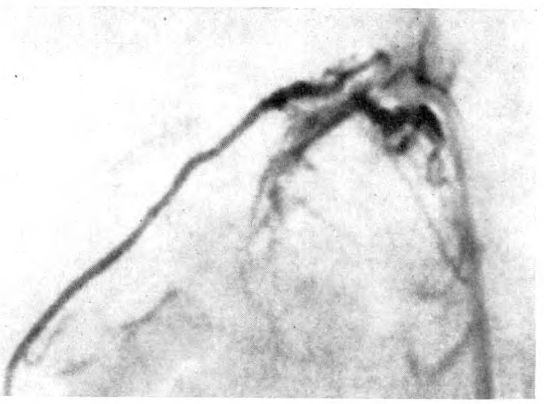


FIG. 2. Right adrenal venogram, subtraction technique. The adrenal veins are draped over a mass which cannot be opacified by the retrograde injection.

Rossi *et al.*[7] stated that if the amount of contrast medium injected is sufficient, if the tip of the catheter is in the proper place, and if selective catheterization is performed, most of these tumors should be visualized. Fry *et al.*,[2] who failed to opacify 4 out of 8 tumors, believe that layering of contrast medium and insufficient flow through the tumor may be the reason for failure. Our case shows that pheochromocytomas might be partially infarcted and still remain biochemically active. When this happens, arteriography may fail to identify the mass unless it is large enough



FIG. 3. Adrenal gland and tumor cut through the frontal plane and positioned as seen in the body. White solid arrowheads point to normal adrenal gland. White punched out arrowheads outline the tumor. Black arrow points to an area of fibrosis.

to displace neighboring vessels. Adrenal venography has been used for the diagnosis of pheochromocytomas by others because they felt that it was safer than intra-arterial injections. Thus, Mitty[5] demonstrated a pheochromocytoma located in the right adrenal gland using this technique. In his case, however, a reticular venous network was opacified during the retrograde injection, indicating that this was not an avascular tumor. Our example, on the other hand, shows a completely avascular tumor which partially replaces the adrenal gland. The specimen obtained at surgery confirms that impression.

In view of the above discussion, it becomes obvious that if a pheochromocytoma is suspected, but cannot be visualized by arteriography, one must proceed to adrenal venography. This procedure can localize even small tumors of the adrenal gland.[3–6] In the presence of a negative venogram and arteriogram, one should entertain the possibility of an infarcted extra-adrenal tumor (10 per cent of all pheochromocytomas are extra-adrenal).

SUMMARY

Approximately 10 per cent of pheochromocytomas cannot be visualized by arteriography. Past reports have incriminated a number of causes for this occurrence.

Partial necrosis of these tumors may occur and may be one more reason for non-visualization of pheochromocytoma by arteriography. Thus, it is recommended that adrenal venography be performed when a pheochromocytoma is suspected clinically, but cannot be demonstrated by arteriography.

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We thank Dr. George Gray from the Department of Pathology for the photography of the specimen.

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ADRENAL ADENOMA AND HYPERPLASIA*

THE IMPORTANCE OF ARTERIOGRAPHIC DIFFERENTIAL DIAGNOSIS

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BENIGN or malignant tumors, or hyperplasia of the adrenal cortex can produce hyperadrenocorticism (Table I). It is important to accurately differentiate between hyperplasia and tumor in the surgical management of these patients. The precise presurgical lateralization of tumors to avoid a blind exploratory laparotomy cannot be overemphasized. When a small autonomous adrenocortical tumor is causing the patient's symptoms, the surgeon need only remove the tumor. If the lesion is one of adenomatous hyperplasia, he must be prepared to do a bilateral total adrenalectomy or an unilateral adrenalectomy with 90 per cent resection of the contralateral hyperplastic gland.[6,17]

The majority of adrenal tumors do not grow to a large size. They are not readily detected by routine intravenous pyelography,[12,13] and nephrotomography will not demonstrate a lesion until it has reached a minimum diameter of about 2.5 cm.[4] The popularity of retroperitoneal gas insufflation has gradually faded because of its relative diagnostic inaccuracy and the high incidence of complications.[4,19] Since the adrenal glands are well vascularized organs and the cortical tumors are often very vascular, arteriography is considered to be the roentgenologic examination of choice for their diagnosis.[8,18,20]

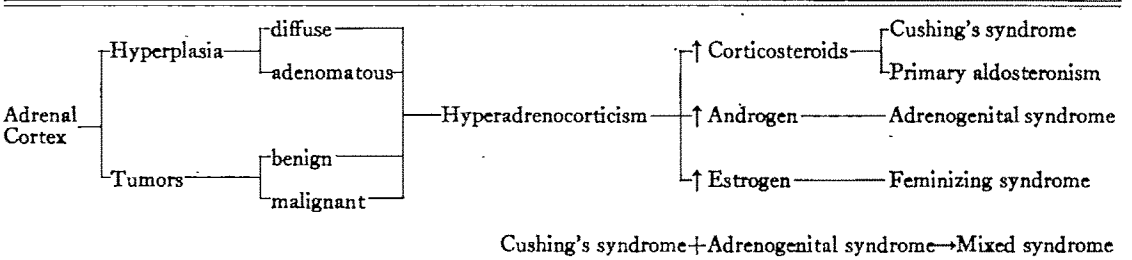
This communication will document the differential arteriographic findings in 3 cases of hyperadrenocorticism due to benign adenoma or hyperplasia and 1 case of non-functioning cortical adenoma. Each case was correctly diagnosed by arteriography and subsequently confirmed by surgery. The importance of aortography and selective arteriography prior to surgery for hyperadrenocorticism is discussed. Correlated pathologic findings are presented.

REPORT OF CASES

CASE 1. A 28 year old white female was admitted to the University of Kansas Medical Center because of a gradual increase in body hair for the last year, and irregular menstruation since discontinuation of birth control pills. She stated that her voice had always been husky. There was no history of hypertension, facial flushing or palpitation. Physical examination revealed a moderately built, healthy white female with an excessive growth of hair on the face, upper lip, chin, abdomen and lower extremities. The abdomen was soft without pal-

TABLE I

CLASSIFICATION OF HYPERADRENOCORTICAL SYNDROMES PRODUCED BY HYPERPLASIA AND TUMORS OF THE ADRENAL CORTEX



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pable masses or organs. Pelvic examination revealed a markedly enlarged clitoris.

Routine blood cell count and urinalysis as well as liver function tests and the VDRL were normal. Fasting blood sugar was 92 mg./100 ml., and blood urea nitrogen (BUN) was 14 mg./100 ml. Serum electrolytes were within normal limits. The urinary 17 ketosteroids were 32.4 mg./24 hr. (normal in female 10 ± 5 mg./24 hr.) and 17 ketogenic steroids were 32.4 mg./24 hr. (normal in female 13 ± 6 mg./24 hr.). Suppression test with a high dose of dexamethasone (Liddle test) was negative. T_4 was 3.6 mg./100 ml. and true thyroxin was 6.0 mg./100 ml. The electrocardiogram showed incomplete right bundle branch block.

Chest roentgenography and intravenous pyelography with nephrotomography failed to demonstrate any abnormalities. A flush abdominal aortogram (Fig. 1*A*) was normal except for a slight vascular blush detected in the left adrenal area. Selective left renal arteriography following injection of epinephrine failed to demonstrate the left inferior adrenal artery or the gland. Selective injection into a common inferior phrenic artery trunk arising directly from the aorta demonstrated a large vascular tumor measuring 4×4.2 cm. The tumor was located in the lateral portion of the adrenal and compressed the remaining normal tissue medially. A coarse vascular network was supplied by a large feeding artery arising from the left inferior phrenic artery. Early filling of a vein draining the tumor was seen in the arterial phase (Fig. 1*B*). In the capillary phase, a diffuse tumor staining was sharply demarcated from the adjacent normal tissue. Selective right renal arteriography followed by superselective inferior adrenal arteriography showed excellent opacification of a concave, triangular shaped adrenal which was smaller than normal in size (Fig. 1*C*). A cortical adenoma of the left adrenal with contralateral glandular atrophy was diagnosed arteriographically.

At surgery, a well circumscribed, encapsulated adrenal cortical tumor was found and removed with the left adrenal gland. A 5 month postsurgical follow-up revealed that the patient was doing well on supportive hormone therapy.

Pathologic examination revealed a well encapsulated pale gray mass measuring 4 cm. in diameter and weighing 38 grams. The cut surface was uniformly tan with flecks of golden yellow. No gross hemorrhage or necrosis was identified (Fig. 1*D*). The cells comprising the tumor were predominantly oval in shape with moderately distinct cell borders and variable amounts of granular eosinophilic to vacuolated cytoplasm. The nuclei were uniformly round to oval and the chromatin was moderately granular. Mitosis or necrosis was not identified. Numerous sections of the specimen stained by Humberstone's method for elastic tissue in vascular structures showed a distinct serial alveolar arrangement of cells separated by thin walled, blood-filled sinusoids lined by flattened endothelium. In many areas there were dilated capillary channels (Fig. 1*E*). The final diagnosis was benign adenoma.

Case II. A 14 year old girl was admitted to KUMC because of amenorrhea and a 30 pound weight gain during the past year. Physical examination revealed a well-developed, well-nourished Caucasian female with marked truncal obesity. The blood pressure was 140–170/100–110 mm. Hg. She had a moon facies, a buffalo hump, was hirsute and had large purple striae over her trunk and thighs.

Routine blood studies and serum electrolytes were normal. The urinary 17-hydroxycorticosteroids were elevated and increased with ACTH stimulation, but were not suppressed with a triple dose of dexamethasone. She had a diabetic glucose tolerance curve.

The chest roentgenogram and excretory pyelogram with tomograms were normal. An abdominal aortogram demonstrated tumor vessels in the left adrenal area (Fig. 2*A*). A homogeneous blush was seen in the capillary phase (Fig. 2*B*). The right adrenal gland appeared to be atrophic and opacified poorly. The arteriographic diagnosis was left adrenal tumor.

At surgery, a 2.5 cm., round, rather firm mass was found in the region of the left adrenal. During manipulation of this mass, the blood pressure rose from 100 to 200. Left adrenalectomy including the tumor and biopsy of the right adrenal were performed. The postoperative course was uneventful. Six months follow-up revealed that the patient was doing well on supportive cortisone therapy.

Pathologic study showed the left adrenal gland to weigh 9.5 grams and to contain a centrally located ovoid tumor measuring $2.8 \times 2.2 \times 2.3$ cm. Cut sections showed it to be composed of dark brown, finely granular, soft tis-

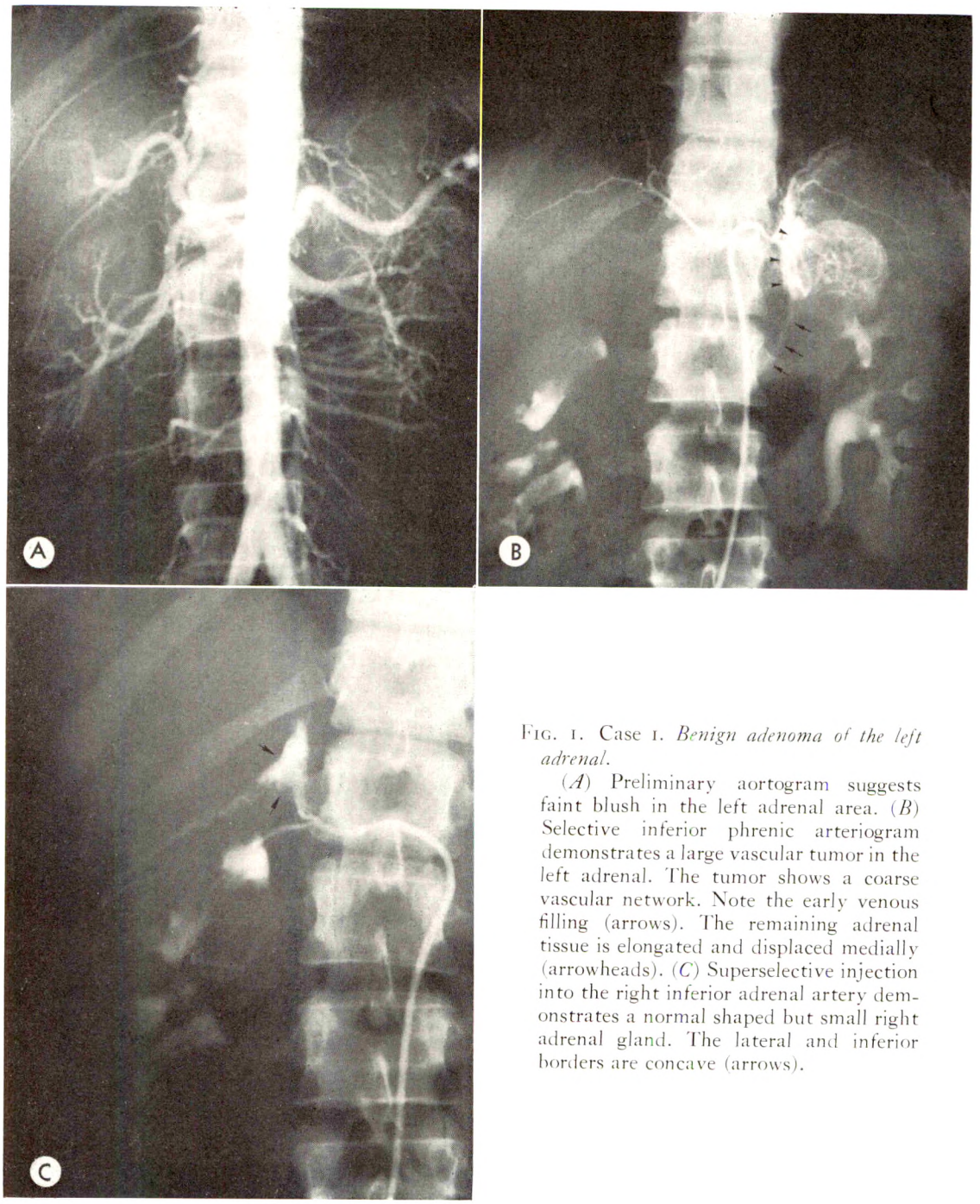


FIG. 1. Case 1. *Benign adenoma of the left adrenal.*

(*A*) Preliminary aortogram suggests faint blush in the left adrenal area. (*B*) Selective inferior phrenic arteriogram demonstrates a large vascular tumor in the left adrenal. The tumor shows a coarse vascular network. Note the early venous filling (arrows). The remaining adrenal tissue is elongated and displaced medially (arrowheads). (*C*) Superselective injection into the right inferior adrenal artery demonstrates a normal shaped but small right adrenal gland. The lateral and inferior borders are concave (arrows).

sue. Histologically, the tumor was composed of closely packed lobules of cells with numerous intervening thin-walled blood vessels. The tumor cells were round to polygonal in shape with only minimal pleomorphism. They had finely granular, eosinophilic cytoplasm. No mitoses were seen. The histomorphology was that of a benign cortical adenoma. The biopsy specimen of the right adrenal showed cortical atrophy.

CASE III. A 43 year old Negro male had been in good health until 2 months prior to admission to KUMC for evaluation of pedal edema.

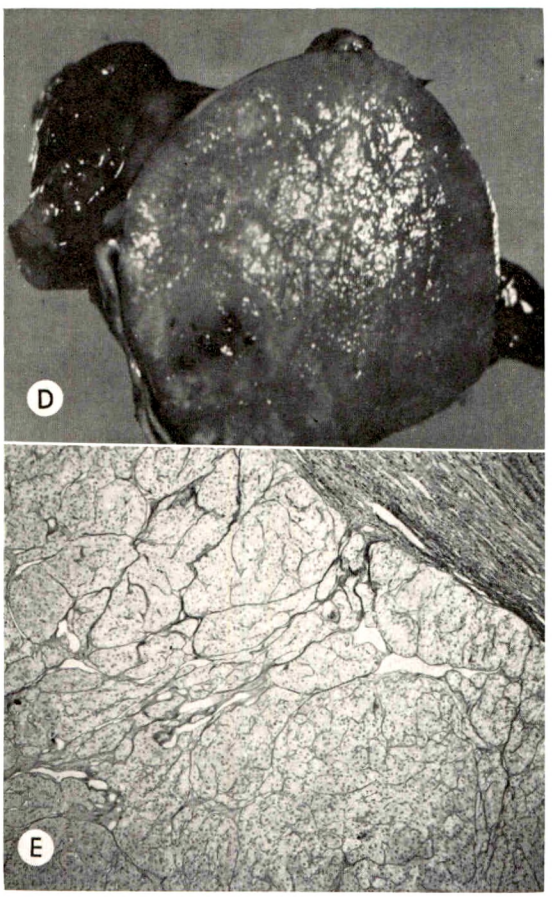


Fig. 1. (*D*) The pathologic specimen of the tumor reveals a rim of compressed gland at the upper left. (*E*) The adenoma is well encapsulated. Note the prominent vascularity (Humberstone stain counterstained with H. & E. ×125).

He also complained of dyspnea on exertion, orthopnea and headache. He was found to have hypertension. His mother and one brother were also hypertensive.

Physical examination revealed an obese Negro male in no acute distress. The blood pressure was 142/100 mm. Hg. The fundi showed a Grade 1 Kimmelstiel-Wilson disease with no evidence of hemorrhages or exudates. There was an irregular cardiac rhythm with splitting of the second sounds. No murmurs or thrills were noted. The abdomen was obese, but no masses or organs were palpable.

Laboratory studies showed a normal blood cell count, urinalysis, and serum electrolytes. Fasting blood sugar was 117 gm./100 ml., with a normal GTT. Liver function tests, PBI, T_4 and metanephrines were all normal. The 24 hour urinary catecholamine was elevated to 155 micrograms. The VMA was elevated on one occasion, but normal on two occasions.

The chest roentgenogram revealed cardiomegaly with left ventricular prominence. The intravenous pyelogram was normal. The abdominal aortogram failed to demonstrate any tumor vessels or abnormal blushes in the renal or suprarenal areas. Selective injection into the left inferior phrenic artery demonstrated a dense homogeneous opacification of the left adrenal gland, which was moderately enlarged but of normal semilunar configuration (Fig. 3). No abnormal vessels suggesting the presence of tumor was demonstrated. The arteriographic findings indicated adrenal hyperplasia.

At surgery, the left adrenal was markedly enlarged and the right adrenal was slightly enlarged. Frozen sections confirmed the diagnosis of adrenal hyperplasia. The surgeon noted that each adrenal was drained by an exceptionally big vein.

The left adrenal was crescent in shape, weighed 8.5 grams and measured 6.5×2.5 cm. The cut surface presented a yellow cortex and grayish-brown medulla. The right adrenal was roughly oval in shape and was enmeshed in a thick pad of adipose tissue. It weighed 6.5 grams. Histologically, the left adrenal showed a few cortical nodules consisting of columns of

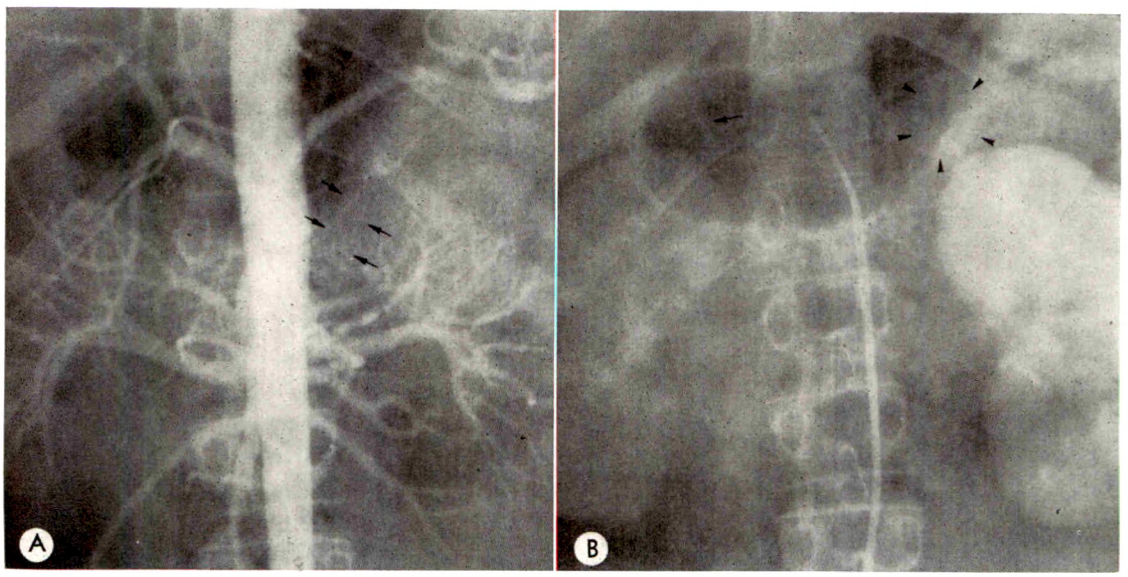


FIG. 2. Case II. *Benign adenoma of the left adrenal.*

(*A*) Abdominal aortogram demonstrates tumor vessels (arrows) in the left suprarenal region. (*B*) A round tumor blush (arrowheads) is well visualized in the capillary phase. The right adrenal gland is small and poorly opacified (arrow).

adrenal cells resembling those of the fascicular zone circumscribed by collagenous tissue. The medulla was not remarkable. The diagnosis was nodular cortical hyperplasia.

CASE IV. A 61 year old female was admitted to the University Hospital because of persistent epigastric pain, vomiting, diarrhea and 40 pound weight loss. She had a 6 year history of hiatus hernia and gastric ulcer. Five months prior to admission, she had the sudden onset of left upper quadrant and left lower chest pain. Physical examination revealed an obese white female in mild distress. The blood pressure was 158/96 mm. Hg. A large firm mass was palpable in the left upper quadrant.

Laboratory studies including routine blood studies, serum electrolytes, liver function studies and urinalysis were normal. The urinary 17-ketosteroids and 17-ketogenic steroids were within normal limits. The electrocardiogram during hospitalization varied from a normal sinus rhythm to a supraventricular tachycardia.

The chest roentgenograms were normal. Intravenous pyelography and nephrotomography demonstrated a large mass in the left suprarenal area. Preliminary abdominal aortography disclosed a large suprarenal mass (Fig. 4*A*). Selective left renal arteriography depicted irregular tumor vessels arising from the inferior adrenal arteries and patchy collections of contrast material in the tumor mass (Fig. 4*B*). The upper pole of the left kidney was sharply demarcated. A non-functioning malignant tumor of the left adrenal was diagnosed arteriographically.

At surgery, a large well encapsulated tumor was found in the left suprarenal region. The tumor compressed the tail of the pancreas and the splenic vessels upward, but was not adherent to any surrounding tissues. Complete excision of the tumor with left nephrectomy was performed.

The tumor weighed 515 grams and measured 17×17×4.5 cm. When it was opened, necrotic grayish-pink material with black stagnant blood was found. There were multiple firm nodules throughout the mass. The capsule was smooth, glistening and adherent to the mass superiorly. Microscopically, capsular invasion by the tumor was not identified. The tumor cells were large and had abundant granular and occasionally vacuolated cytoplasm. The nuclei were markedly pleomorphic. Occasional macronucleoli were present. Blood vessel invasion was not noted. The benignancy or malignancy of this non-functioning cortical tumor could not be determined with certainty.

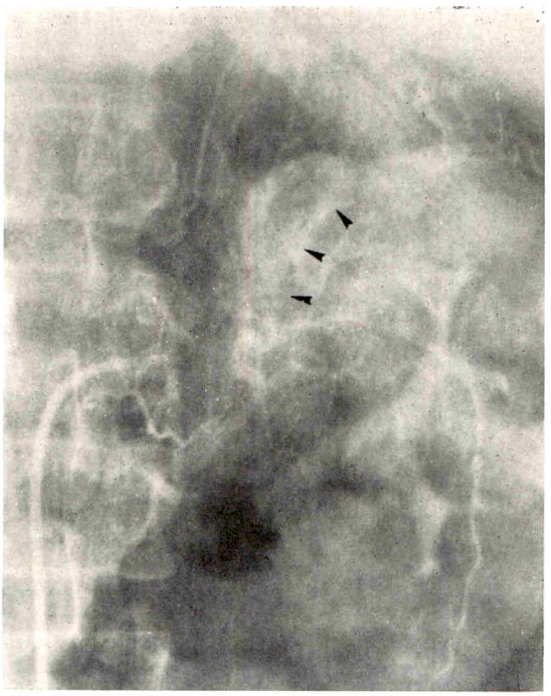


Fig. 3. Case III. *Adrenal hyperplasia.*
Selective left inferior phrenic artery injection demonstrates a densely opacified diffusely enlarged, adrenal gland (arrowheads).

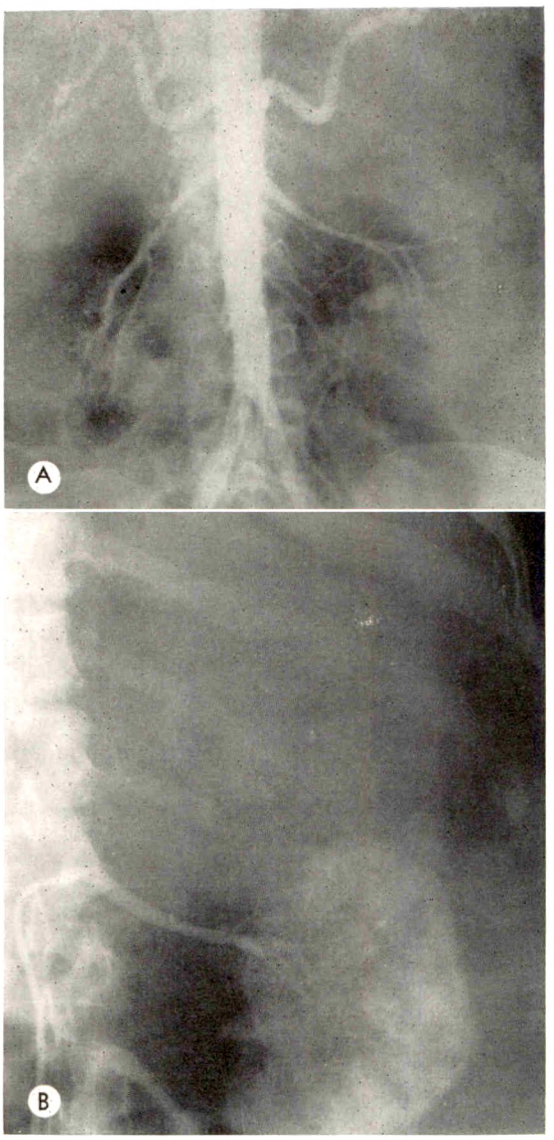


Fig. 4. Case IV. *Non-functioning cortical adenoma of the left adrenal.*
(*A*) Preliminary aortogram discloses a large mass in the left suprarenal area. The left kidney is displaced downward and laterally. (*B*) Selective left renal arteriogram depicts irregular tumor vessels and abnormal collections of contrast material.

DISCUSSION

The adrenal glands are 2 small organs located immediately above and in front of the upper pole of each kidney. The right adrenal is triangular in shape and attaches loosely to the right kidney. The left is more semilunar and attaches more firmly to the left kidney. The average size of the adrenal glands is 3–5 cm. in length, 3–5 cm. in width, and 0.4–0.6 cm. in thickness. The average weight is 3.5–5 grams each.[5]

The adrenal glands have a rich arterial supply consisting of 10 to 60 branches from adjacent larger arteries with great individual variation and asymmetry.[16] There are 3 main arterial sources. The superior adrenal arteries almost always originate from the inferior phrenic artery and supply the superomedial portion of the gland (Fig. 5). The middle adrenal artery arises directly from the aorta. It is usually a single branch and supplies the anteromedial portion of the gland. The inferior adrenal arteries arise from the ipsilateral renal artery in the majority of cases. They supply the remaining largest portion of the gland.

The results of aortography without selective arteriography for visualization of the adrenal gland have been disappointing.[9,20] Only 50 per cent of cases in a series using a sophisticated aortographic technique ade-

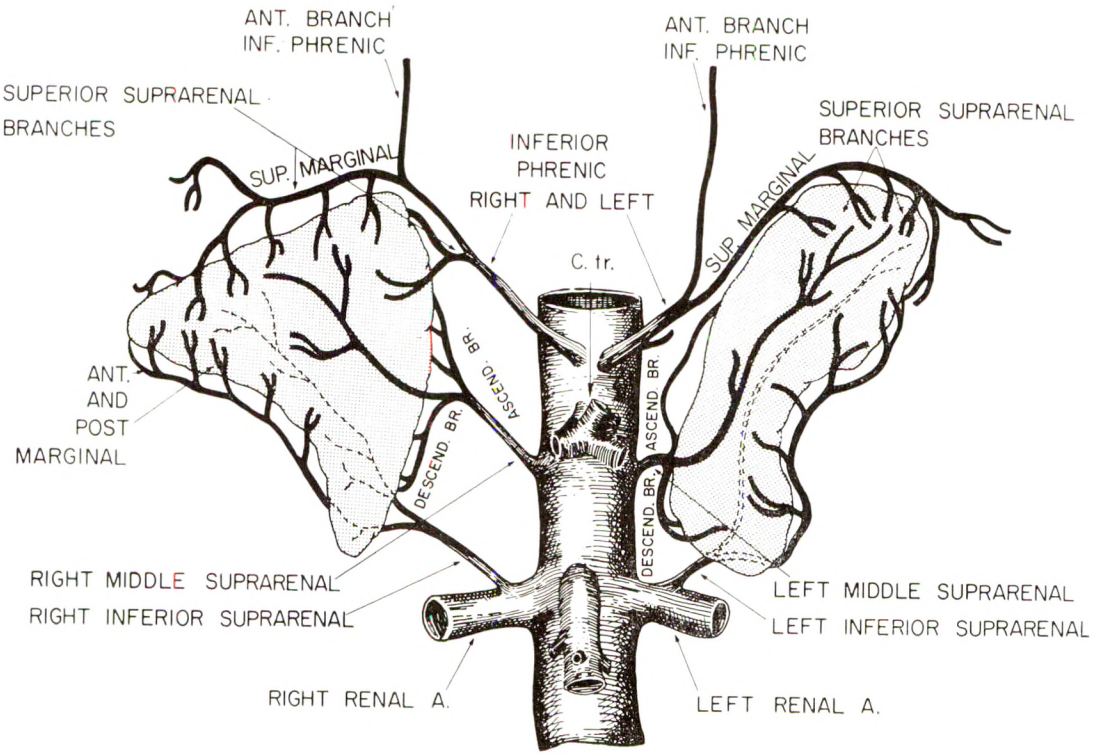


FIG. 5. Retouched schematic drawing of the arterial supply to the adrenal glands[17] (modified from Gerard 1913).

quately demonstrated the adrenals.[14] However, aortography remains a valuable procedure as the initial contrast examination. It may reveal increased vascularity or tumor stain and provides information about the origins of the adrenal arteries, thus expediting subsequent selective arterial studies. Bilateral selective renal arteriography following intrarenal administration of 10 micrograms of epinephrine may provide very satisfactory demonstration of the adrenal glands.[8] However, superselective catheterization of the inferior adrenal arteries affords the best visualization of the major portion of a gland (Fig. 1*C*). The inferior phrenic artery can be directly catheterized if it arises from the aorta and will result in opacification of the upper half of the gland (Fig. 1*B*). A selective celiac arteriogram following intraceliac injection of epinephrine frequently gives good results in cases where the inferior phrenic artery arises from this artery.[9] Direct catheterization of the middle adrenal artery is relatively difficult because of its small size.

Functioning adenomas are moderately vascular and demonstrate a variety of tumor vessel configurations. These tumors may have a fine or coarse reticulated vascular network in the arterial phase and a localized, sharply outlined blush in the capillary phase. Early filling of veins (Fig. 1*B*) is not a common finding in benign adenomas and has been reported only once in the literature.[1] If the adenoma is avascular, displacement or distortion of normal vascular structures or the presence of a round filling defect in the capillary phase is frequently demonstrated.[10] The majority of aldosteronomas are small in size and avascular.[3,10] The arteriographic findings of non-functioning adenomas (Case IV) are similar to those of functioning adenomas.[15]

Adrenal carcinomas are best evaluated by arteriography because they are large

and generally quite vascular.[20] They are characterized by numerous irregular tumor vessels, pooling of contrast material, early venous filling and a patchy capillary blush. The feeding vessels are rather large and occasionally show invasion. Celiac arteriography will frequently identify liver metastases.

It must be emphasized that the roentgenologic as well as the histopathologic differential diagnosis between benign and malignant cortical tumor is impossible. Because of their large size, primary adrenal carcinomas tend to have more abundant tumor vascularity and early venous filling. However, early venous filling was found in 1 of our benign cases (Fig. 1*B*). The arteriographic findings of pheochromocytomas are similar to those of adenomas, but the former is often more vascular.[2,20] The differentiation between adenoma and pheochromocytoma frequently depends on the clinical manifestations.[20]

Cortical hyperplastic nodules often attain a size of 3 or 4 cm. in diameter and are usually multiple and bilateral.[11,17] In contrast, an adenoma is usually single, unilateral and without the regularity of cellular arrangement found in hyperplastic nodules. In hyperplasia the enlarged gland shows convexity of its lateral border instead of the normal lateral border concavity. Hyperplasia is frequently diagnosed clinically as an adenoma because of the failure of suppression with dexamethasone.

The surgeon and the radiologist must try and differentiate adenoma from hyperplasia since the surgical approach and subsequent treatment are completely different.[6,17] The radiologist should carefully evaluate the contralateral gland if he finds either a vascular or avascular cortical tumor in a patient with Cushing's syndrome. If the contralateral gland is either of normal size or reveals adenomatous changes, the patient has adenomatous hyperplasia rather than a true cortical adenoma. In the latter instance, the contralateral gland is usually atrophic (Case 1).[7,17,21] This is not true in the adrenogenital syndrome where only 30 per cent of the cases show contralateral glandular atrophy.[7]

SUMMARY

Three cases of adrenocortical adenoma and 1 case of cortical hyperplasia correctly diagnosed preoperatively by arteriography are presented.

The various arteriographic patterns are discussed.

Arteriography provides the most accurate information in the evaluation of cases with possible adrenal disease. Frequently, meticulous superselective arteriography is required to adequately demonstrate the pathologic changes in the adrenal glands. It is the responsibility of the radiologist to understand the differential diagnostic signs and implications of adrenal disorders and perform the correct sequence of arteriographic studies.

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UNUSUAL ASPECT OF RENAL ANGIOGRAPHY IN URETERIC DUPLICATION

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VARIATIONS in the urinary tract are common. One of the most frequent is duplication of the collecting system[5,7,8] with nonfunction of the upper pole of the kidney. The diagnosis can usually be made from the intravenous urographic findings. The calyces are fewer in number than in the normal kidney and the uppermost calyx is farther from the outline, *i.e.*, there is an obvious lack of collecting system extending into the upper pole parenchyma. The uppermost calyx is often short and appears to be more the type associated with the middle of the kidney than the long necked infundibulum and calyceal placement of the upper and lower poles. Angiographically, there is stretching of the segmental arteries around the dilated, nonfunctioning calyces and a corresponding radiolucent defect is generally present during the nephrographic phase.

On rare occasions, however, this lesion may present an atypical urographic and angiographic appearance, as illustrated by the following 3 cases.

REPORT OF CASES

CASE 1. This was the first hospital admission of a 25 year old white female with the chief complaint of incontinence. The patient stated that she had noted dribbling of urine since childhood. Intravenous pyelogram showed prompt functioning collecting systems bilaterally. The axis of the upper pole collecting system of the right kidney appeared slightly lateral in position and the amount of upper pole parenchyma seemed in excess for the collecting system visualized (Fig. 1). The patient was admitted for urologic work-up. Physical examination and laboratory data were unremarkable.

During the first hospital week, the patient underwent aortography and selective right renal arteriography. The aortogram showed no evidence of ectopic kidney. The left kidney appeared to be normal. On the right side, a selective renal arteriogram showed a small area of irregular avascularity at the upper pole (Fig. 2). This appeared to be almost a cap at the top of the right kidney (Fig. 3). There was no

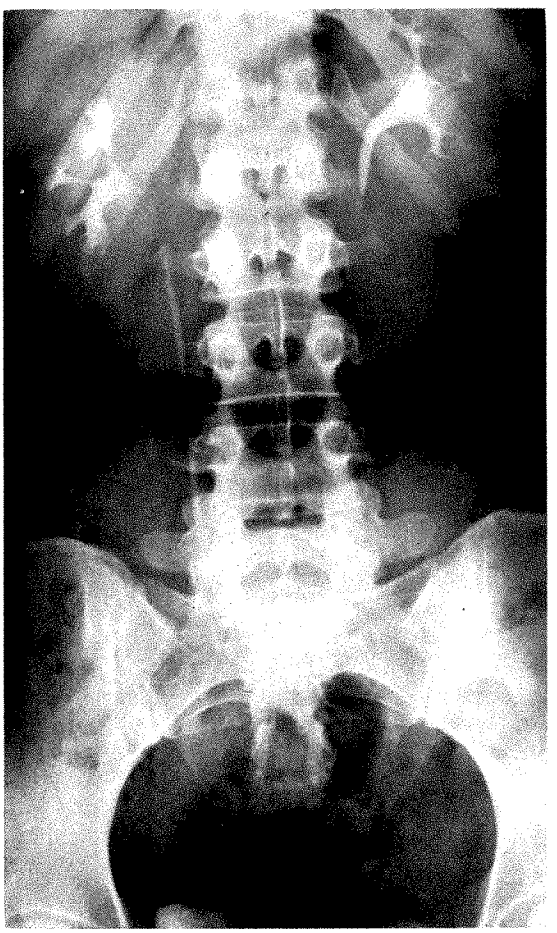


FIG. 1. Case 1. Intravenous pyelogram shows prompt functioning systems. The right upper pole collecting system does not appear to be visualized. The parenchyma at the upper pole seems to be in excess for the collecting system seen.

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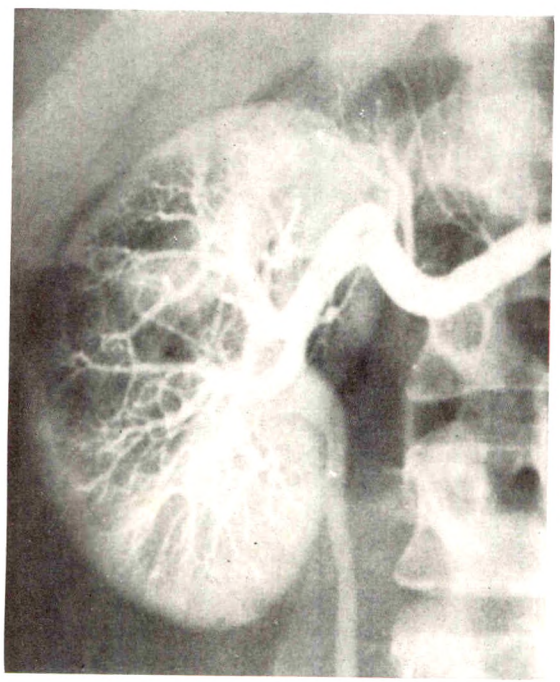


FIG. 2. Case 1. Selective right renal arteriogram shows a small area of irregular avascularity at the upper pole.

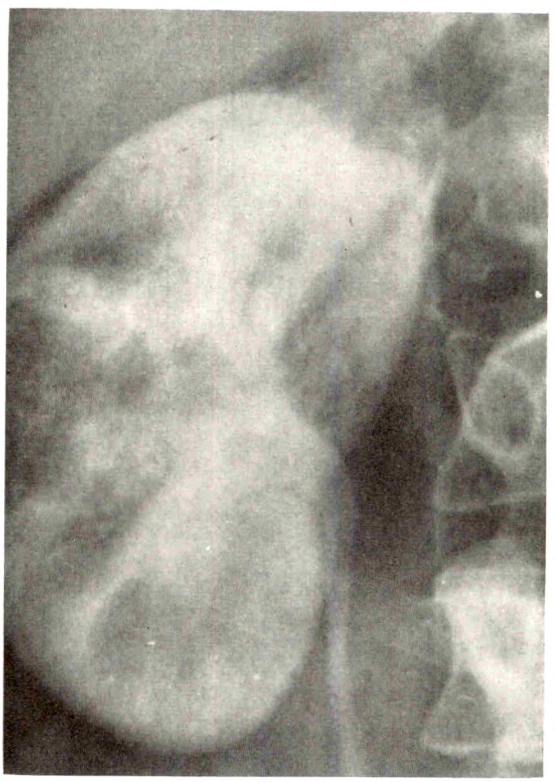


FIG. 3. Case 1. Later arterial phase shows some staining in a small amount of tissue, cap-like in shape, at the top of the right kidney. No tumor vascularity and only minimal stretching of several small vessels are noted.

tumor vascularity and there was only minimal stretching of several small renal arteries in this region. However, no clearly defined rim was seen and the radiolucent defect was not sharply rounded. Thus, this lesion did not satisfy the criteria for typical subcapsular cyst. Other possibilities such as an avascular tumor, localized inflammatory mass or localized area of hydronephrosis were considered.

At operation the right kidney with its known small cap of tissue at the upper pole was seen to be quite high in position. Two small arteries, about 1 mm. in diameter, went to this cap of tissue, which appeared to be a hydronephrotic sac. The 2 small arteries were easily ligated and divided. The cap portion immediately became cyanotic and was resected. A separate ureter was seen draining this tissue. It was later noted that the normal ureter in the lower portion of the kidney was of regular caliber and about one-third the size of the one to the upper pole. The ectopic ureter was dissected downward well below the lower pole of the kidney, probably to about 3–4 cm. above the pelvic brim. The ureter was then divided. Inspection of the vagina indicated that the opening of this ectopic ureter was in the vaginal vault.

Pathology reported a resected hydronephrotic upper pole of kidney and ureter. The renal tissue contained patchy areas of fibrosis and focal features suggesting a dysplastic kidney. The ureter was considered unremarkable.

The patient was discharged uneventfully with the initial complaint cured.

CASE II. This was the second hospital admission of this 45 year old female complaining of left lower quadrant abdominal pain and vaginal discharge. A diagnosis of urethral diverticulum had been made 4 years prior to this admission and the patient had had a resection of this blind sac-like structure which emptied into the urethra. The patient had temporary relief of symptomatology, but was readmitted for further evaluation when vaginal discharge recurred. Positive physical findings were confined to the pelvic examination where a cystic mass was felt on the ventral surface of the urethra approximately 1 inch from the meatus measuring approximately 2 inches across.

An intravenous pyelogram revealed prompt functioning collecting systems bilaterally. A calcification was noted associated with the upper medial margin of the left kidney. The cortical nephrogram effect in this region was slightly irregular (Fig. 4). Nephrotomography confirmed the calcification and the cortical nephrogram defect (Fig. 5).

The patient underwent cystoscopy and a sinus opening in the vagina was identified. Introduction of contrast material into this sinus revealed an ectopic ureter which led to a small hydronephrotic collecting system (Fig. 6). Selective renal arteriography revealed a relatively avascular area. Stretching of a few small vessels was seen (Fig. 7). The calcification appeared to be centered in this area of decreased contrast (Fig. 8), presumably the dilated upper pole ectopic collecting system.

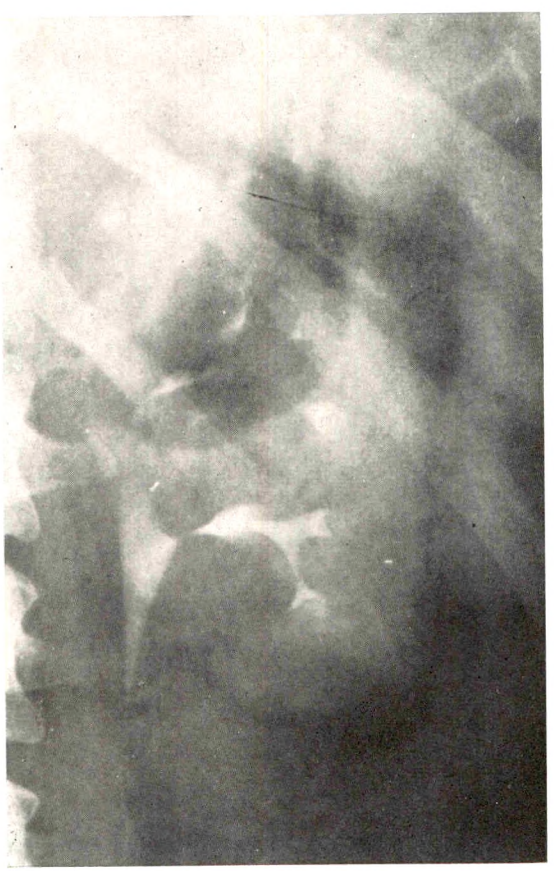


Fig. 4. Case II. Intravenous pyelography shows prompt functioning collecting system. A small calcification is noted associated with the upper medial margin of the left kidney. The nephrogram effect in this region medially is somewhat irregular.

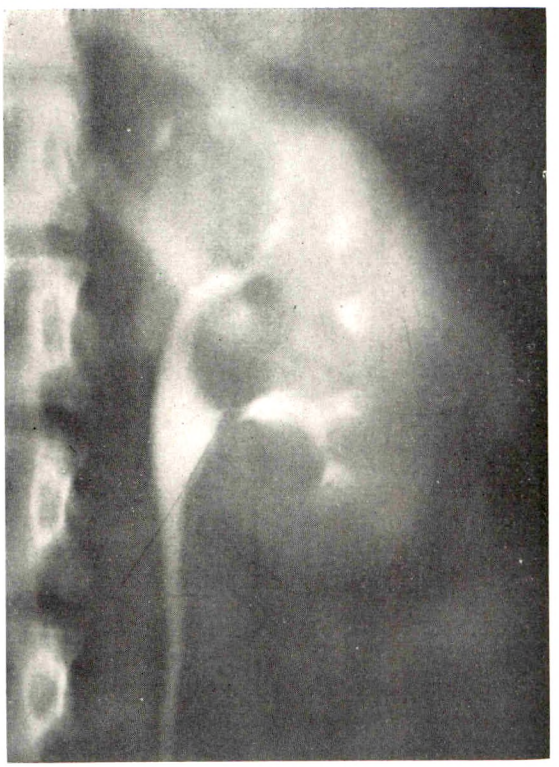


Fig. 5. Case II. Nephrotomography confirms the calcification and the cortical nephrogram defect.

At operation the ectopic ureter led to a remnant of kidney occupying the upper pole of the left kidney. Removal of this upper pole and ectopic ureter resulted in complete cessation of symptoms.

Case III. This was the first St. Luke's Hospital admission of a 42 year old female with a 24 hour history of hematuria. The patient was seen 2 months prior to this admission at an urology clinic where an intravenous pyelogram revealed a double ureter on the right side with reflux. An oval calcification associated with the upper pole contour was seen. There appeared to be an upper pole collecting system which was not visualized (Fig. 9). Physical examination was normal. When admitted to the hospital, a right retrograde pyelogram showed no filling of the right upper pole collecting system by catheterization of a single ureter identified in normal position on the right side of the bladder (Fig. 10). A second ureteral orifice was identified in the bladder on the right and retrograde filling of this ureter revealed a hydronephrotic upper

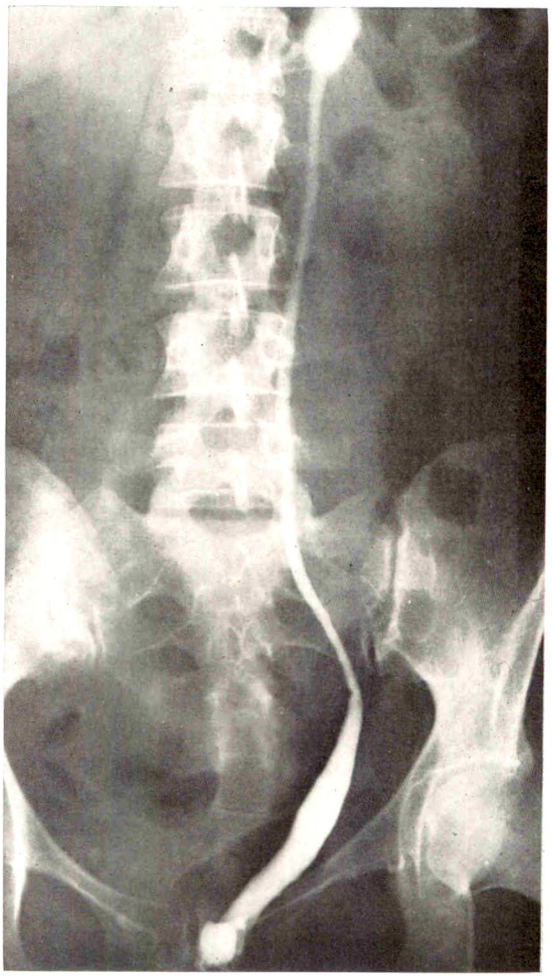


FIG. 6. Case II. Retrograde pyelogram reveals an ectopic ureter which leads to a small hydronephrotic collecting system.

pole collecting system which was nonfunctioning on intravenous pyelogram (Fig. 11).

Selective right renal arteriography demonstrated areas of avascularity in the upper pole. Stretching of small vessels and disorganization of the normal vascular pattern were seen (Fig. 12). In the later arterial phase the medial contour of the upper pole appeared irregular with vessels projecting medial to the main cortical nephrogram (Fig. 13).

In the late nephrogram phase, irregular staining was seen adjacent to the medial upper pole border (Fig. 14).

The patient underwent resection of the upper pole nonfunctioning hydronephrotic kidney tissue and the ectopic ureter. The patient recovered rapidly and was discharged promptly.

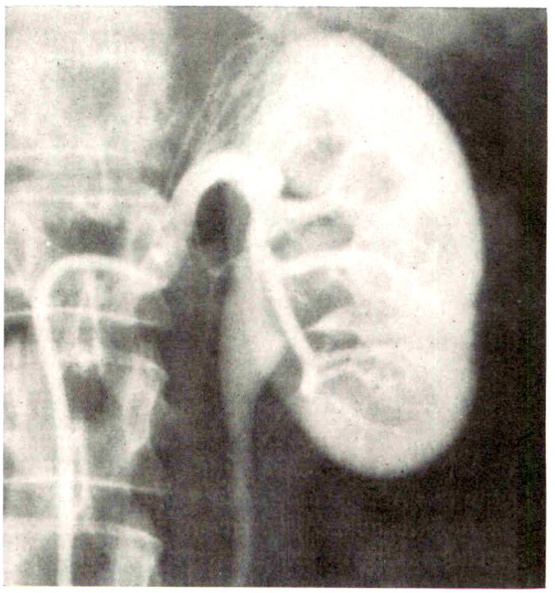


FIG. 7. Case II. Selective renal arteriogram reveals a relatively avascular area. Stretching of a few small vessels is seen.

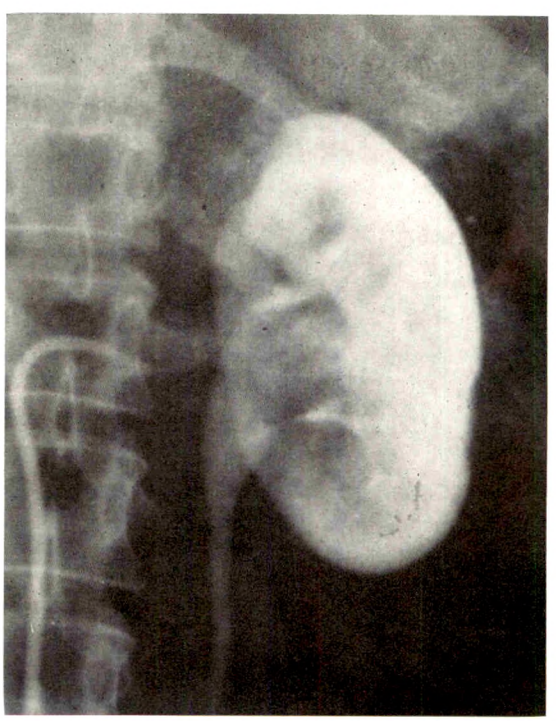


FIG. 8. Case II. Calcification appears to be centered in this area of decreased contrast. This is the area of the dilated upper pole ectopic collecting system. The avascular area is slightly medial and is cap-like in shape.

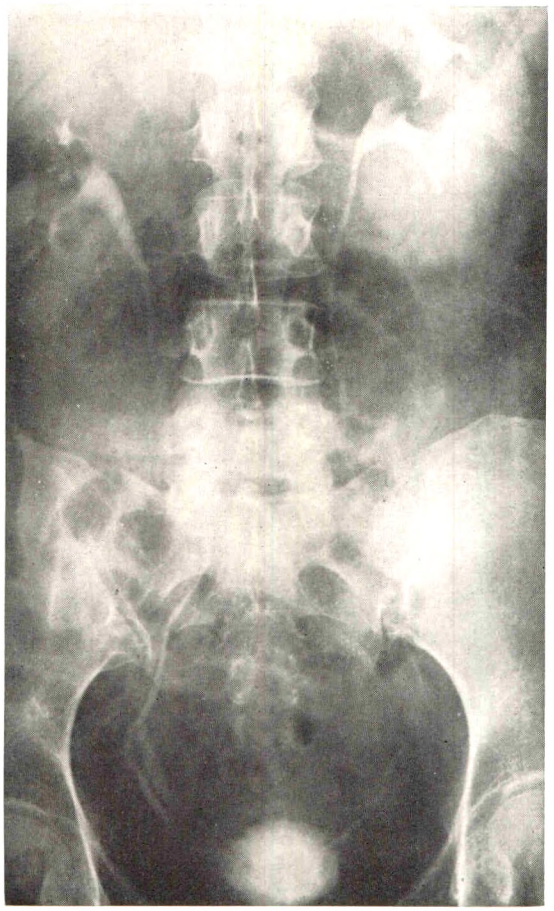


Fig. 9. Case III. Intravenous pyelogram reveals a double ureter on the right side demonstrated by reflux into the more dilated medial distal ureter.

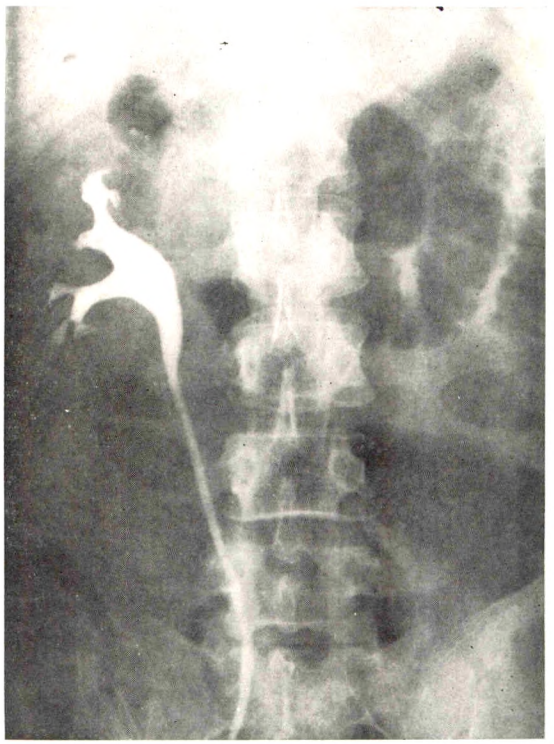


Fig. 10. Case III. Retrograde pyelogram shows no filling of the upper pole collecting system by catheterization of a single ureter identified in the right side of the bladder.

DISCUSSION

In one series[6] complete ureteral duplication was found in 109 cases in 16,000 autopsies. It was more common in females and more likely to exhibit pathologic complications in females. Bilateral duplication was common as well.

In most instances of duplication of the collecting system, the kidney remains undivided except in the rare instance of supernumerary kidney.[2] Therefore, in the usual circumstances, the kidney has a superficially normal appearance except perhaps for some increase in length. Slight irregularity in the contour of the kidney with 2 systems of drainage may be interpreted as pyelonephritic scarring or slight hydronephrosis of one element. Usually the upper system is the smaller of the 2 consisting of 1 or 2 calyceal groupings. The higher ureter usually finds an opening in the more caudal position in the bladder relative to the normal ureter. Therefore, in the ordinary uncomplicated case, the orifice that is normally placed in the corner of the trigone corresponds to the lower and main portion of the collecting pelvis, while the upper ureter opens through a slit-like orifice more distally.

A duplicated ureteric opening may not empty into the bladder, but may remain in the bladder neck area or posterior urethra. These accessory ureters most often drain the upper pole of the kidney. When the ectopic ureteric opening is further afield, the ureter is almost always dilated, sometimes severely, and the renal element which it drains is often dysplastic or pyelonephritic.[1,4,9]

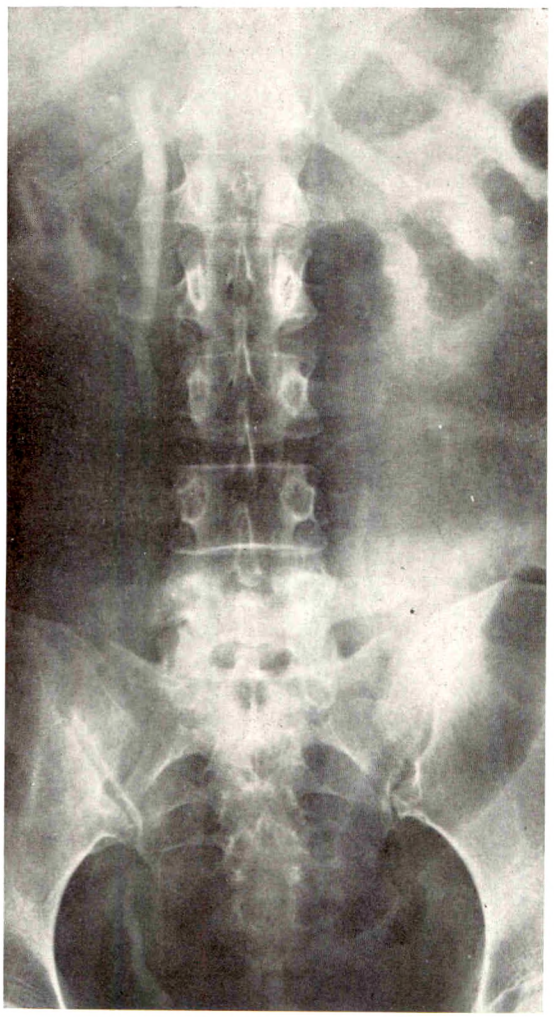


FIG. 11. Case III. A second ureteral orifice was identified in the bladder on the right and retrograde filling of this ureter revealed the hydronephrotic upper pole collecting system which is nonfunctioning on the intravenous pyelogram.

When the ureter opens into the lower urethra or vagina, dribbling incontinence is the rule, and infection is less common. The characteristic complaint is one of continual wetting despite normal micturition at normal intervals. When it opens into the urethra, the dribbling incontinence may be due to the return of urine refluxing into the ectopic ureter during micturition and the symptom may therefore be present even though the upper part of the ectopic ureter is atretic and the renal element without function. Such an ectopic ureter is of course functionally a diverticulum of the urethra.[3]

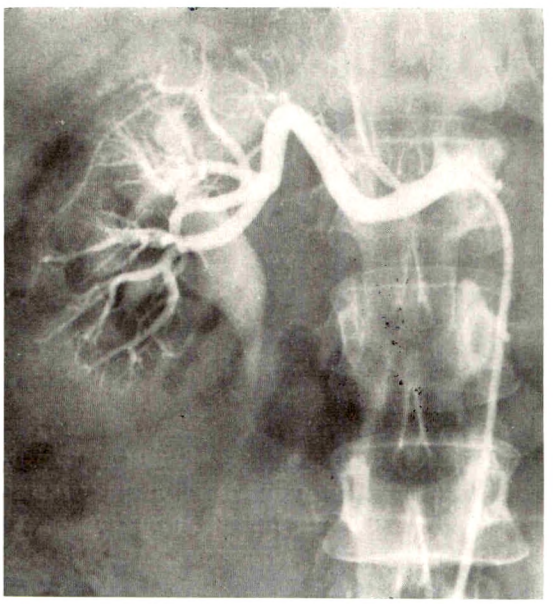


FIG. 12. Case III. Selective renal arteriogram demonstrates areas of avascularity in the upper pole of the right kidney. Stretching of small vessels and disorganization of the normal vascular pattern are seen.

Although the urographic and angiographic changes are often typical, our 3 cases illustrate the fact that occasionally the intravenous pyelographic findings may be subtle and the upper pole collecting system that is drained by an ectopic ureter may

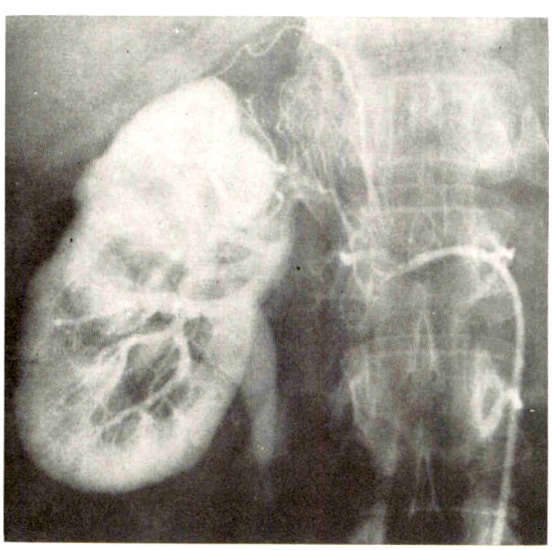


FIG. 13. Case III. In the later arterial phase the medial contour of the upper pole of the right kidney appears irregular with vessels projecting medial to the main cortical nephrogram.

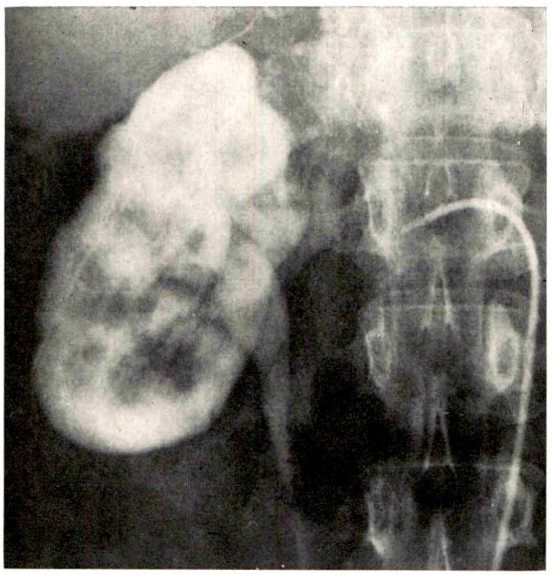


Fig. 14. Case III. In the late nephrogram phase irregular staining is seen adjacent to the medial upper pole border.

not be appreciated. This is especially true if it drains only a small area of dysplastic, hydronephrotic tissue which caps the tip of the upper pole. Angiography in this setting can also be misleading. A small area of relative avascularity at the tip of the upper pole results from the caliectasis present. This may also produce an atypical distribution of vessels with stretching and displacement. Compression of adjacent parenchyma secondary to caliectasis in the nonfunctioning collecting system can result in an area of increased density following selective angiography and can be misinterpreted as tumor stain. In the absence of clearly defined criteria for a benign cyst, tumor must be included in the differential diagnosis. This inevitably results in a surgical exploration and possible nephrectomy.

The purpose of this report is to familiarize radiologists with the angiographic appearance of this unusual form of ureteral duplication with hydronephrosis. As opposed to the more common form of the entity, this lesion produces a small, irregular cap of poorly opacified renal tissue at the tip of the upper pole, usually with stretching of adjacent segmental arteries. Once suspicion has been aroused, re-evaluation of the more basic intravenous or retrograde urograms, or a repeat of the latter study, may identify the ectopic collecting system.

SUMMARY

A common variation in the urinary tract is duplication of the ureter. Pathologic complications are frequent.

Three examples of an unusual form of this lesion, in which the duplicated ureter drains a small, dysplastic, hydronephrotic cap of tissue at the very tip of the upper pole, are presented.

The angiographic findings may be misleading, unless one is aware of the characteristic appearance. Once suspicion is aroused, review of the intravenous pyelogram, retrograde pyelogram and cystoscopic findings, may help in identification of the ectopic system and help save the major portion of a well functioning kidney.

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RETROPERITONEAL FIBROSIS: AN ANALYSIS OF THE CLINICAL SPECTRUM AND ROENTGENOGRAPHIC SIGNS*

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RETROPERITONEAL fibrosis presents with a clinical and roentgenographic spectrum involving the ureters, inferior vena cava, iliac vein and rectum. The clinical and roentgenographic aspects of this spectrum are, as a result, quite variable. Most of this spectrum is discussed in the literature in scattered reports, many of which are case reports.

Twenty-one cases of retroperitoneal fibrosis seen at the Hospital of the University of Pennsylvania from 1960 to 1973 illustrate this wide spectrum.

Medial deviation of tapered obstructed ureters in the lower lumbar spine region is described as a "classic sign" of retroperitoneal fibrosis in most reports.[21,22,24,28,40,49] A comparison statistical analysis of the position of the ureters relative to the lower lumbar spine and sacrum was done comparing the 21 cases of retroperitoneal fibrosis with 60 consecutive normal cases. This analysis showed a lack of significant difference between the 2 groups.

This suggests that in retroperitoneal fibrosis, the ureter (normally variable in its course) is caught in the fibrotic process rather than being deviated medially or pulled medially by the fibrotic process.

HISTORICAL REVIEW

In 1905, Albarran[2] first described ureteral obstruction due to fibrotic changes in the retroperitoneal space. Bachrach[4] in 1928 distinguished 2 groups of fibrosis—a generalized and a localized form.

In 1953, Vest and Barelare[57] described a localized form which they called periureteritis plastica. Additional cases were described in 1937[42,46] and in 1942.[10,27]

Ormond[39] in 1948 published 2 cases of bilateral ureteral obstruction due to an inflammatory fibrotic retroperitoneal process. In 1968, Packham and Yates-Bell[40] surveyed 130 previously reported cases and reported on 8 new ones. Additional cases were reported by Kerr *et al.*[24] (16 cases) in 1968 and by Saxton *et al.*[49] (14 cases) in 1969.

PATHOLOGY

On gross examination, there is a pale, plaque-like mass with indefinable margins which envelops the adjacent structures, usually the ureters and the vena cava. On microscopic examination, there is a fibroblastic proliferation with a pleomorphic inflammatory cell infiltrate. This involves the fascial and adipose investments of the retroperitoneal space in an indifferent fashion. Fat cells, nerves, muscle and vessels are all encased. The fibrotic reaction is limited to the outer wall and no ureteral wall inflammatory process is seen.

Although most authors characterize the process as a proliferative fibrotic reaction of retroperitoneal tissues associated with chronic inflammatory response, some authors report an active chronic phlebitis.[1,17,20,43,44,51]

ETIOLOGY

There have been multiple etiologic theories as to the cause of retroperitoneal fibrosis. Various authors have reported cases and given their etiologic theories. These include: (1) pyelonephritis;[25,56] (2) lower intestinal tract infections;[9,19] (3) pancreatitis;[6] (4) thrombophlebitis;[58] (5) renal tuberculosis;[31] (6) lymphopathia vener-

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TABLE I

CLINICAL SYMPTOMS: RELATIVE FREQUENCY OF SYMPTOMS IN 189 CASES

Symptoms	Kerr *et al.*[24] (16 cases)	Packham and Yates-Bell[40] (138 cases)		Saxton *et al.*[49] (14 cases)	HUP* (21 cases)
		No.	Per Cent		
Nausea and vomiting		30	22	7	4
Weight loss	6	45	33	6	3
Frequency and dysuria	3	23	17		4
Increased blood pressure	1	39	29	10	3
Anemia		43 of 43 reported			4
Elevated sedimentation rate	4	34 of 34 reported		12	
Pain in the loin		64	45	5	
Abdominal pain		56	41		
Back pain	16	34	25	2	18
Mass		15	11		4

* HUP = Hospital of University of Pennsylvania.

TABLE II

SUMMARY OF CASES

Case and Age (yr.)	Clinical Features	Side	Other Involvement
1. FW59	Abdominal pain—leg swelling—constipation—sansert	right and left	Inferior vena cava—rectum
2. FN54	Constipation—left pelvic mass	left	Orbital granuloma
3. FN63	Left flank pain—vomiting—weight loss	right and left	
4. MW41	Right abdominal pain	right	
5. FW18	Right lower quadrant abdominal pain—frequency—dysuria	right	
6. FW62	Left lower abdominal pain—left abdominal mass—abdominal aneurysm	right and left	
7. MW64	Abdominal pain—jaundice	right and left	Pancreas—porta hepatis—mediastinal lymph nodes
8. FN82	Weight loss—mass anterior to rectum encasing the rectum—urinary incontinence	no urinary obstruction	Left iliac vein—mediastinal lymph nodes
9. MN59	Back pain—anorexia—weight loss	right and left	
10. FW38	Nocturia—right lower quadrant pain	right and left	
11. FN50	Thyroid nodule—left lower quadrant pain	left	
12. MN44	Low back pain	right and left	
13. MW53	Nausea—vomiting—weight loss—suprapubic pain	right and left	
14. MW56	Abdominal aneurysm—hypertension	right and left	
15. FN14	Anemia—hypertension	right and left	
16. FN58	Right and left abdominal pain—diverticulosis	right and left	
17. FW38	Abdominal and back pain—nocturia	right and left	
18. MW53	Right flank pain	right	
19. FW57	Sansert therapy for 3 years—leg edema—back pain—anemia	left	
20. FW41	Nausea—vomiting—lower abdominal pain—anemia—hypertension—pelvic mass	right and left	
21. MN48	Left flank pain—anemia	left	

eum;[12] (7) urine extravasation;[35] (8) aqueous contrast extravasated in retroperitoneum;[26] (9) previous surgery;[2,52] (10) hematomas from multiple causes;[8,25,37,54] (11) generalized arteritis;[18] (12) collagenosis;[29] (13) association with malignancies causing fibrosis;[23,25,55] and (14) drug induced by methysergide, ergotamine, dihydroergotamine and hydralazine.[21]

INCIDENCE

In 4 series totaling 215 cases,[24,28,40,47] the age range was 8 to 80 years. Over-all, the male incidence was 3 times that of females. However, under the age of 30, the female incidence was twice that of males. Sixty per cent presented in the fourth to fifth decades. Fifty to 75 per cent were bilateral at presentation.

Our 21 cases showed an age range of 14 to 82 years. Thirteen of 21 were females. Thirteen of 21 were bilateral and 12 of 21 presented in the sixth and seventh decades.

Gelford *et al.*[15] reported 50 to 60 cases of retroperitoneal fibrosis secondary to sansert out of over 500,000 being treated with the drug for migraine headaches.

DISCUSSION

The variable and often vague presentation of retroperitoneal fibrosis is characteristic. The clinical picture ranges from symptom onset of 2 days to over 1 year. Back or abdominal pain is the most common symptom. Other presenting complaints include nausea, vomiting, weight loss, frequency of urination, dysuria, hypertension, anemia, elevated sedimentation rate, palpable mass, constipation, leg edema, incontinence, anorexia, nocturia, and anuria.

The relative frequency of the most com-

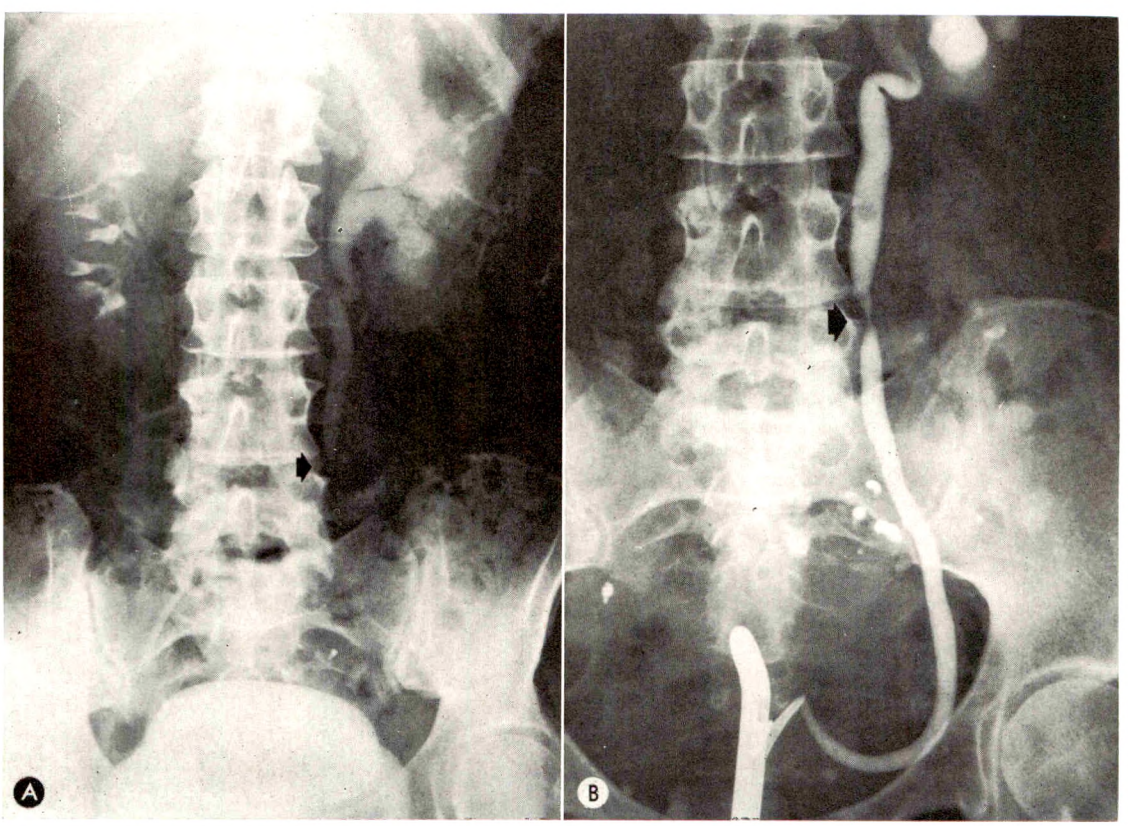


FIG. 1. (*A*) One hour roentgenogram of 58 year old Negro female shows left hydronephrosis and obstruction of ureter (closed arrow). Note normal incompletely filled right kidney. (*B*) Retrograde ureterogram shows small area of ureteral narrowing due to retroperitoneal fibrosis causing the left obstruction (closed arrow).

mon complaints in the spectrum is shown in Tables I and II.

The extent of demonstrable roentgenographic abnormalities elucidates the cause of this variable presentation. Hydronephrosis and hydroureter with delayed excretion is one roentgenographic finding. This may be unilateral (Fig. 1, *A* and *B*; and 2, *A* and *B*) or bilateral (Fig. 3, *A* and *B*; and 4*A*) when first seen. Other patients initially have one ureter involved and later present with involvement of the other ureter (Fig. 5, *A* and *B*). On retrograde study, the catheter passes easily beyond the obstruction site.[15,34,40,49] The fibrotic process may extend to and encase the inferior vena cava and iliac vessels (Fig. 4*C*; and 5*C*).

Venography was abnormal in 2 of Saxton and co-worker's[49] cases. Only 2 of our 21 patients had venograms and both of these were abnormal. Rectal encasement is shown in Figure 4, *B* and *D*. The fibrosis may extend even further, involving other organs such as the pancreas (Fig. 6, *A* and *B*).

Only a few authors have reported lymphangiographic changes. There is obstruction of lymphatics at the site of fibrosis.[28,40] There is some discrepancy in the literature as to the helpfulness of plain film roentgenographic changes. Loss of psoas muscle outline is felt to be significant by some authors.[11,32,48,50,56] Absence of the renal outline has been described.[41,48,50] Other authors feel that the plain film roentgenograms are seldom helpful.[22,49] Our experience was that they were not helpful in our 21 cases.

In a small group of patients, there are additional areas of the body involved by a similar fibrotic process. This is exemplified by the fibrotic mediastinal mass in our Cases 7 and 8 (Table II) and the fibrotic orbital granuloma in our Case 2 (Table II).

To evaluate the "classic sign" of medially deviated ureters in retroperitoneal fibrosis, an analysis of the course of the ureters in both normal and in patients with retroperitoneal fibrosis was done. The differences of the medial side of the ureters

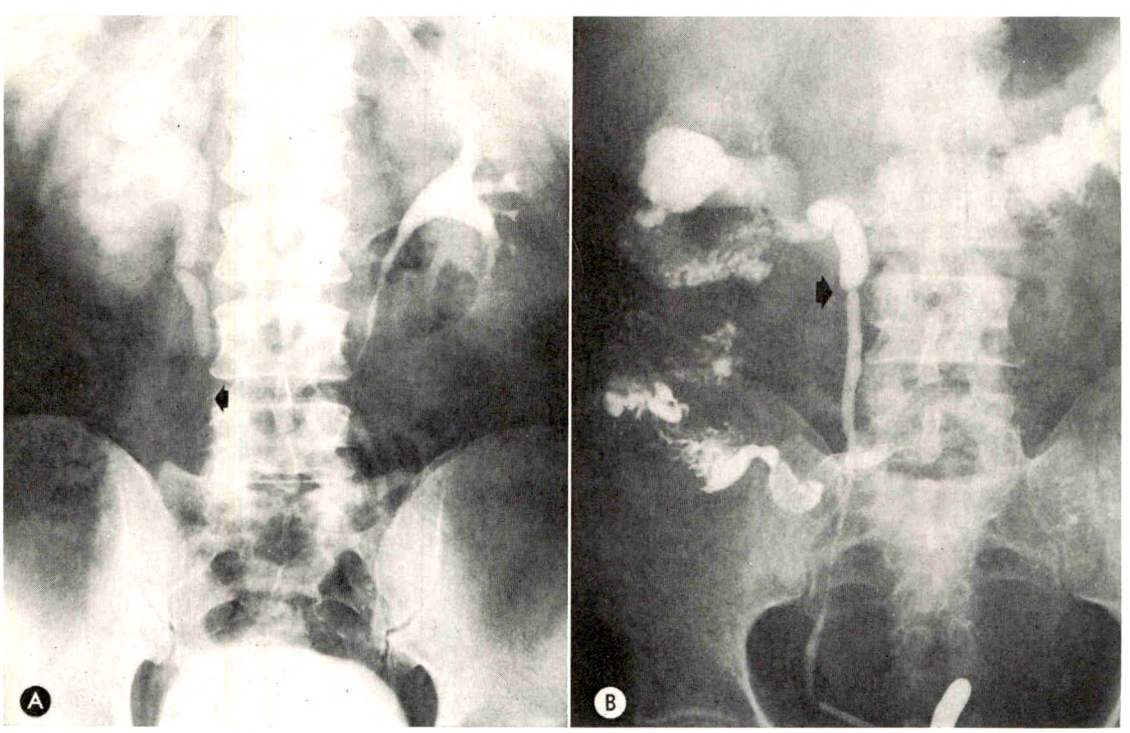


Fig. 2. (*A*) A 53 year old white male with unilateral right hydronephrosis secondary to retroperitoneal fibrosis. Closed arrow shows point of obstruction. Note normal left kidney and ureter. (*B*) Right retrograde ureterogram shows site and extent of involvement (closed arrow).

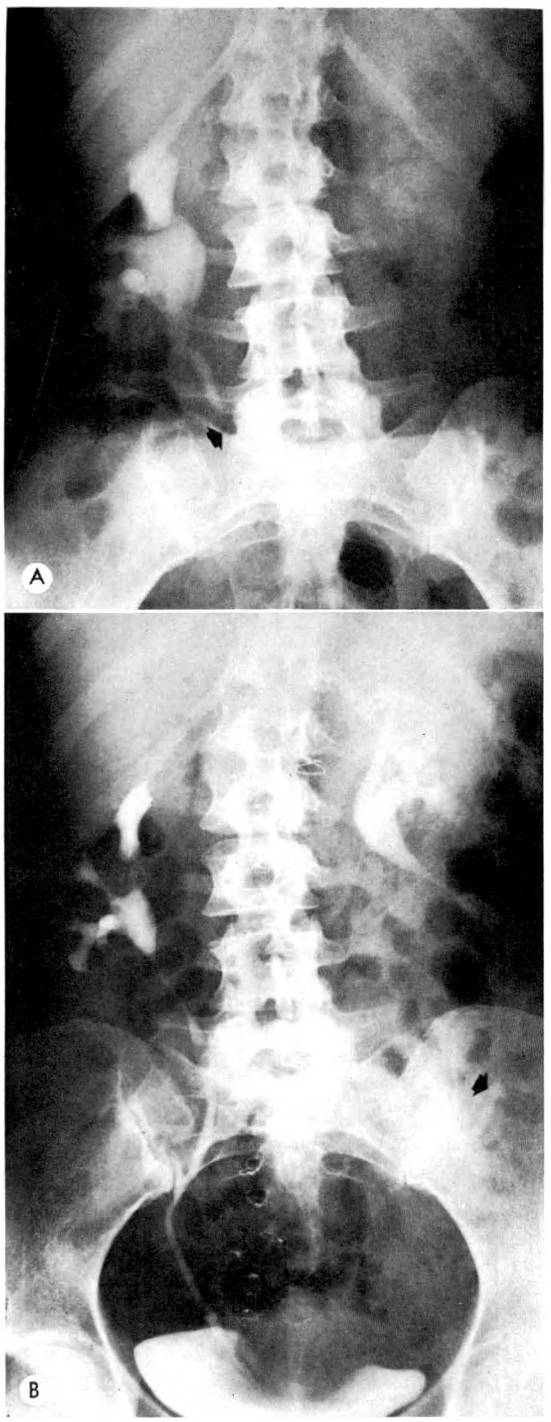


FIG. 3. (*A*) A 38 year old female with bilateral obstruction. Faint visualization is seen on the left. The less obstructed right kidney best demonstrates the level of obstruction (closed arrow). (*B*) Roentgenogram made 2 years postureterolysis shows good bilateral function with some residual blunting of the pelvocalyceal systems. The course of the left ureter is due to the surgical procedure (closed arrow).

from the midpoint of the spine at 3 arbitrary levels were measured. These levels were chosen in the region of most frequent involvement. Level 1 and level 2 were at the inferior border of the pedicles of the lumbar vertebral bodies 4 and 5. The third was 2.5 cm. below the upper margin of the sacrum. Sixty consecutive normals in whom the entire course of the ureters could be seen were compared to the 21 cases of retroperitoneal fibrosis (Fig. 7; and 8). A "t" test between the normals and the abnormals at the 3 levels was done. At no level was there a statistically significant difference observed. The measurements are compared and summarized in Figure 9. This analysis indicates that there is no medial deviation of the ureters. It seems more likely that the ureters are caught in the fibrotic process in their normal variable course.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes: (1) retroperitoneal tumors; (2) metastatic carcinoma; (3) lymphoma; (4) ureteral carcinoma; (5) uric acid stones; (6) aneurysms; (7) infection secondary to tuberculosis and/or diverticulitis; (8) periarteritis nodosa; (9) blood clots of the ureter; and (10) retrocaval ureter.

THERAPY

The treatment of choice in retroperitoneal fibrosis is lysis of the ureters with repositioning of the ureters intraperitoneally. In patients whose fibrosis is due to methysergide, just stopping the drug may relieve the obstruction.[14,15] One of our 2 patients receiving sansert therapy responded to stoppage of the drug, but the other patient required surgery.

Morandi and Grob[33] feel that steroid therapy in the early stages may prevent

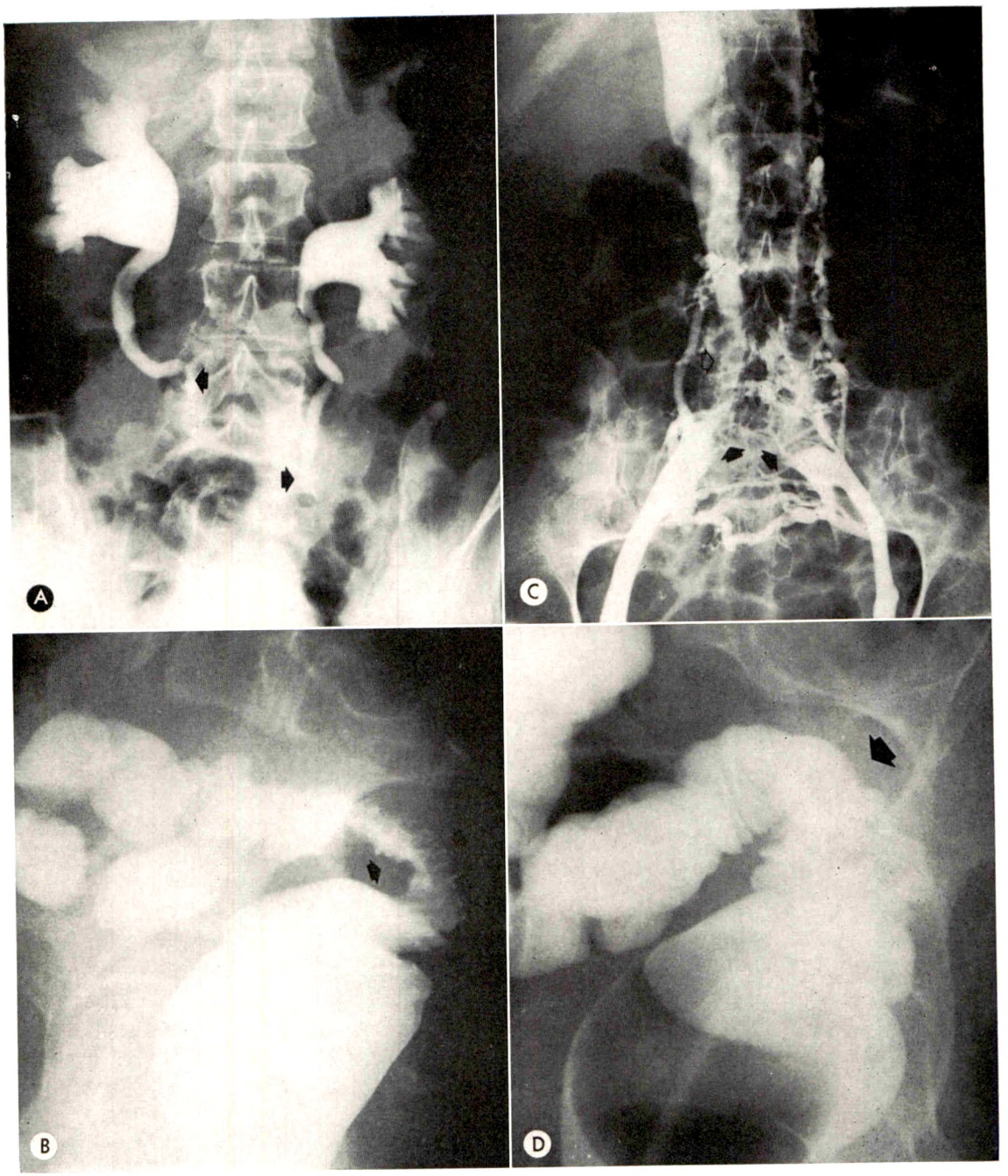


Fig. 4. (*A*) Delayed roentgenogram of 59 year old white female shows bilateral obstruction. The closed arrows show a difference in the level of obstruction on each side. (*B*) A barium enema examination shows the marked extent of the retroperitoneal fibrosis. The closed arrow demonstrates a persistent area of narrowing in the upper rectum due to the fibrotic process. (*C*) Venogram shows obstruction of each common iliac vein (closed arrows). The right open arrow demonstrates the narrowing and partial obstruction of the inferior vena cava. (*D*) A barium enema examination done 3 months after discontinuing sansert and surgical lysis shows the narrowed area to have regressed markedly. A near normal sized lumen is present (closed arrow).

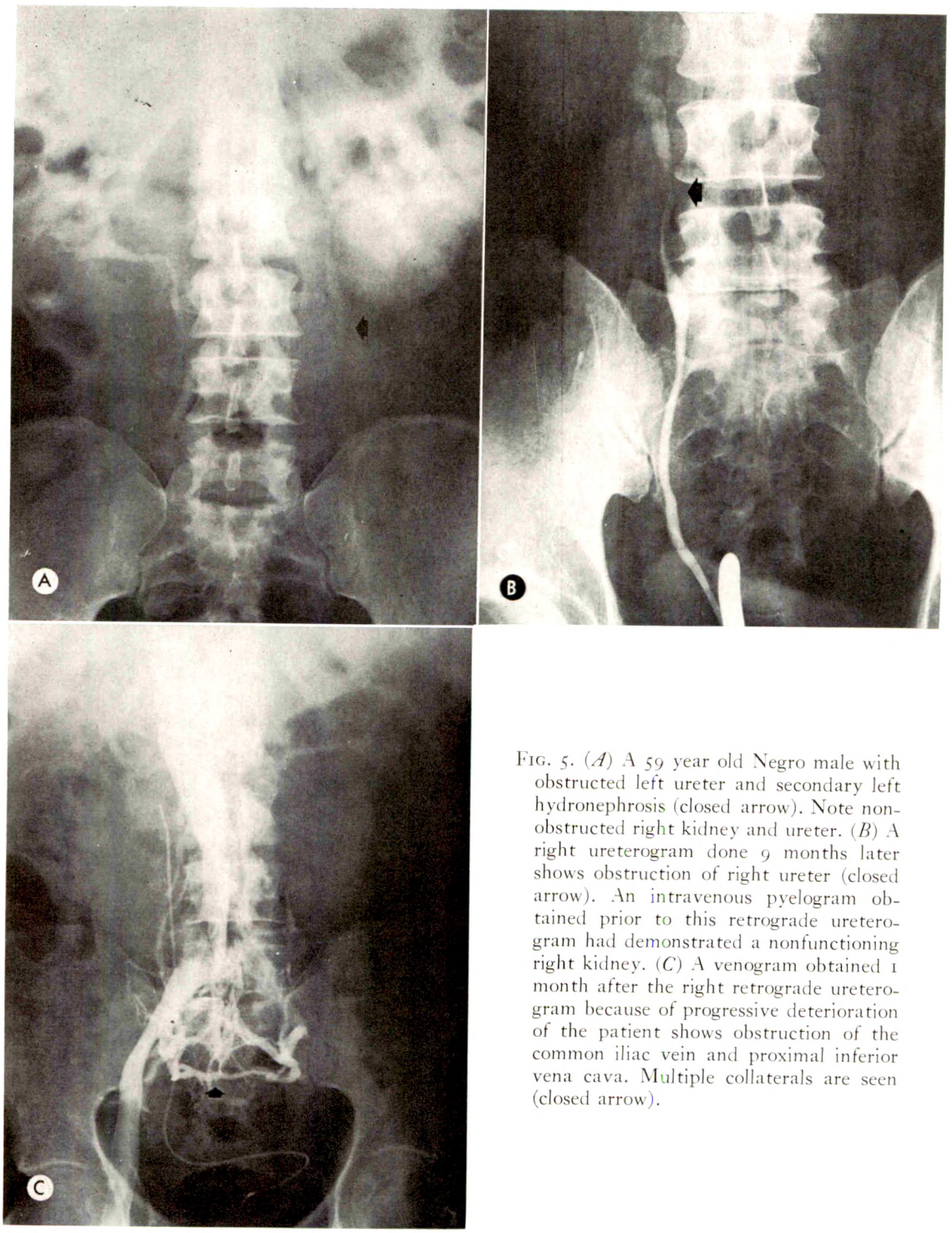


FIG. 5. (*A*) A 59 year old Negro male with obstructed left ureter and secondary left hydronephrosis (closed arrow). Note non-obstructed right kidney and ureter. (*B*) A right ureterogram done 9 months later shows obstruction of right ureter (closed arrow). An intravenous pyelogram obtained prior to this retrograde ureterogram had demonstrated a nonfunctioning right kidney. (*C*) A venogram obtained 1 month after the right retrograde ureterogram because of progressive deterioration of the patient shows obstruction of the common iliac vein and proximal inferior vena cava. Multiple collaterals are seen (closed arrow).

progression of the process if it has not reached a hyaline fibrotic stage. There is some conjecture in the literature as to whether steroid therapy alone will improve the degree of obstruction present.[3,5,13,16,30,36,38,44,47]

SUMMARY

Retroperitoneal fibrosis presents with a clinical and roentgenographic spectrum involving the ureters, inferior vena cava, iliac vein and rectum.

The clinical and roentgenographic as-

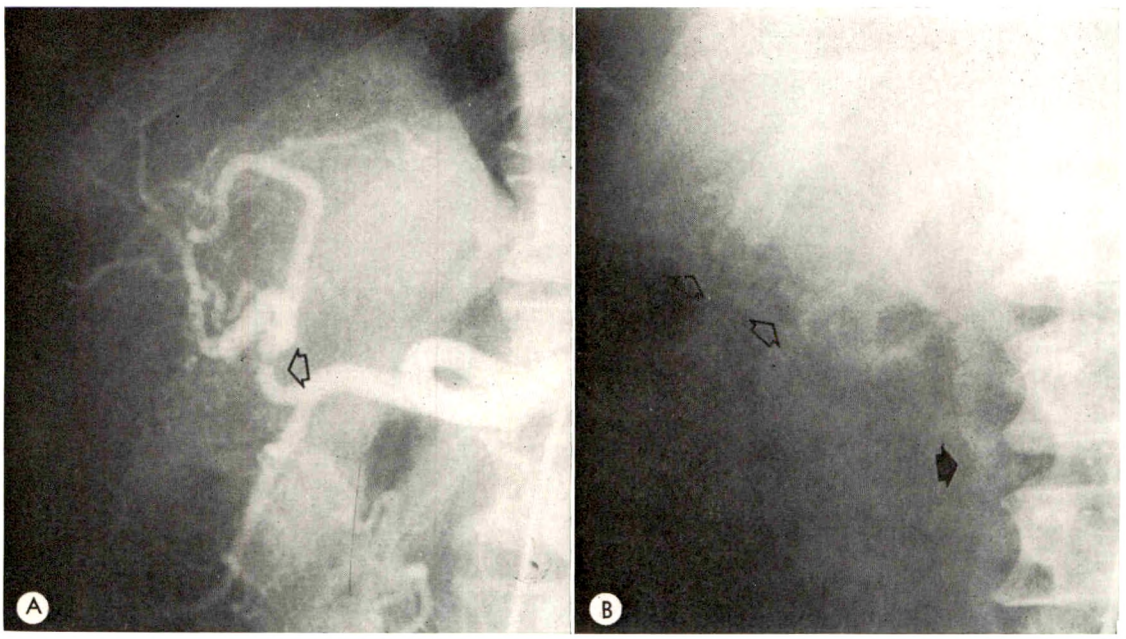


Fig. 6. (*A*) A 64 year old male with abdominal pain and obstructive jaundice. Celiac artery injection shows extrinsic constriction of hepatic artery (open arrow). (*B*) Venous phase shows superior mesenteric vein (closed arrow) and collateral veins (open arrows) indicating obstruction of proximal portal vein.

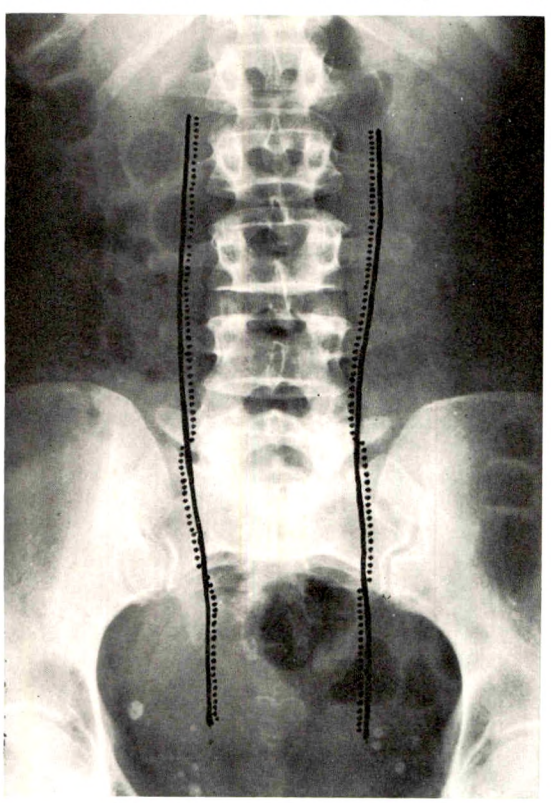


Fig. 7. The solid line represents the average course of the 60 normal ureters which were measured from L4 through the upper sacrum. The broken line represents the average course of the ureters involved with retroperitoneal fibrosis.

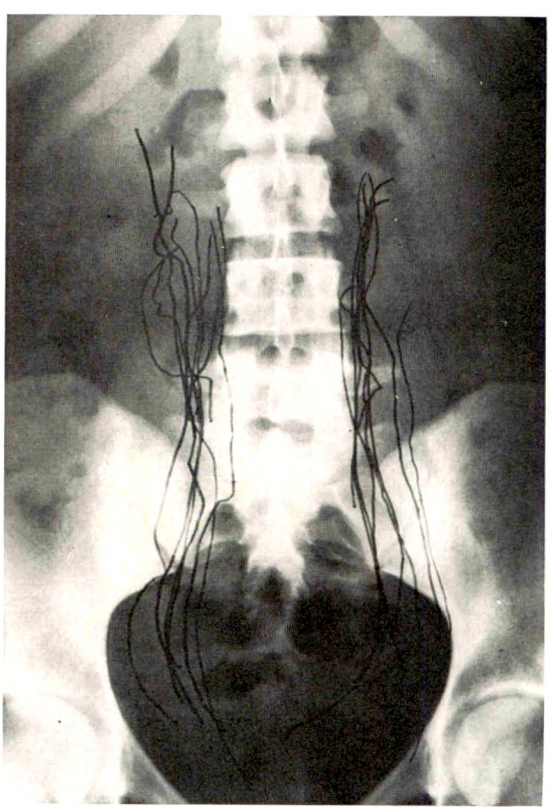


Fig. 8. The multiple lines demonstrate the individual course of the ureters involved with retroperitoneal fibrosis in our series.

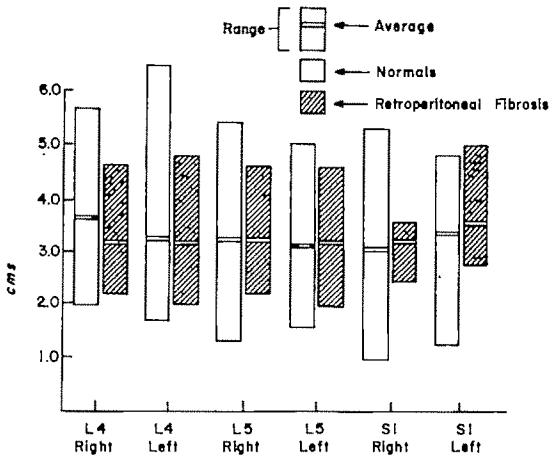


FIG. 9. Barograph of measurements shows lack of significant difference between normal and abnormal.

pects of this spectrum are discussed and illustrated.

The medial deviation of tapered obstructed ureters in the lower lumbar region is described as a "classic sign" of retroperitoneal fibrosis. A comparison statistical analysis of the position of the ureters relative to the lower lumbar spine and sacrum was done comparing 21 cases of retroperitoneal fibrosis with 60 normal cases. This analysis is shown and the lack of significant difference between the 2 groups is demonstrated.

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URETHRAL STRICTURE AND URETHRAL RUPTURE*

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IT has been estimated that 2,000,000 new cases of gonorrhea were contracted in the U.S.A. during 1970.[12] The appearance of strains of gonococci with increased resistance to antibiotics is well documented.[1,12,13,15] It is reasonable to infer that the incidence of the complications of untreated gonorrhea will also increase, and this will include the eventual development of urethral stricture in the male. The gonococcus is not the only cause of urethral stricture.[14] Indeed, it may be difficult to determine in many cases whether the stricture, or strictures, have occurred secondary to gonococcal infection, instrumentation, or to trauma.

The treatment of urethral stricture is dilatation or surgery,[8,14,20] and the majority of patients are usually managed with repeated dilatations. However, repetitive, difficult, painful dilatations, the presence of fistula or sinuses or episodes of repeated postinstrumental fever are indications for consideration of surgical reconstruction of the urethra. One hundred and twenty urethroplasty operations were performed at Jackson Memorial Hospital in the past four years, 1968–1971, and 80 of these were during the 12 months of 1971. In each case, the roentgenologic findings have been a major factor in the decision to operate and in determining the precise surgical approach. A search of the recent American radiologic literature has failed to reveal a comprehensive description of the roentgenology of the male urethra, apart from a short review in 1961.[24] Various techniques, maneuvers, and protocols have been described in other journals[2,6,10,19] and some confusion might reasonably arise as to which approach and what equipment should be used in the investigation of urethral disease.

We will describe our experience with the suggested techniques, and will outline the urethrographic findings and the investigative protocol we now use. Since there are similarities, but also important differences in the roentgenologic investigation of urethral stricture and urethral rupture, the roentgenology of both of these conditions will be described.

MATERIAL

Approximately 100 retrograde urethrographies and 200 male micturating cystourethrographies are performed at Jackson Memorial Hospital each year. Although the majority of the patients investigated have known, or suspected, urethral stricture or rupture, urethrography is also performed in some cases of paraplegia and prostatic disease. We have reviewed the roentgenographic appearances and correlated these with the clinical and, where applicable, the operative findings in 100 patients who have had recent urethrography at this institution. The age of the patients in this series ranged between 11 years and 85 years. In the patients with urethral stricture, symptoms had been present from between a few weeks to 20 years, and in the majority it was not possible to determine reliably the precise sequence of events leading to stricture—whether it was due solely to previous gonorrhea, instrumentation, external trauma, or to a combination of several of these factors.

ANATOMY OF THE MALE URETHRA

The male urethra has been divided descriptively in the urologic and anatomic

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literature into various regions.[5,9,22,24] However, many of these descriptive divisions have limited value in relating to the clinical management of urethral stricture. It is more appropriate to divide the urethra into 4 easily identified anatomic parts: prostatic; membranous; bulbous; and pendulous (Fig. 1, *A* and *B*). The bulbous urethra may be further divided into proximal bulbous and distal bulbous when referring to the position of a stricture. The proximal bulbous urethra is that part of the urethra within 2 cm. of the membranous portion; the distal bulbous urethra is all of the remainder up to the peno-scrotal junction. This subdivision of the bulbous urethra is of importance to the surgeon when a urethroplasty is being considered. As described by Turner-Warwick,[20] if a stricture is limited to the distal bulbous urethra or beyond, then a technically relatively simple urethroplasty may be performed. However, if the stricture extends to involve the region of the proximal bulbous or membranous urethra, then a more technically complex urethroplasty will be necessary. Although this selection of the urethra 2 cm. beyond the membranous portion as being the proximal bulbous urethra represents an arbitrary distance, its purpose is to serve as a preoperative indication to the urologist as to the probability of a technically simple or difficult urethroplasty.

The prostatic urethra is approximately 3 cm. in length and extends from the bladder neck to the external sphincter (synonyms: triangular ligament, urogenital diaphragm) (Fig. 1*A*). This portion of the urethra is surrounded throughout its length by the prostate gland. In the floor of the prostatic urethra there is a longitudinal elevation of the mucosa known as the verumontanum. The multiple orifices of the prostatic ducts (Fig. 2; and 8, *A* and *B*) are situated on either side of the verumontanum. The orifices of the ejaculatory ducts, which are the common ducts for the termination of the seminal vesicles and the vas deferens (Fig. 3) are situated at the apex of the verumontanum. On the posterior aspect of the verumontanum is the opening for the utricle, the vestigial remnant of the Müllerian duct which corresponds to the female vagina and uterus.

The membranous urethra is approximately 1 cm. in length and is situated immediately distal to the prostatic urethra (Fig. 1*A*). It

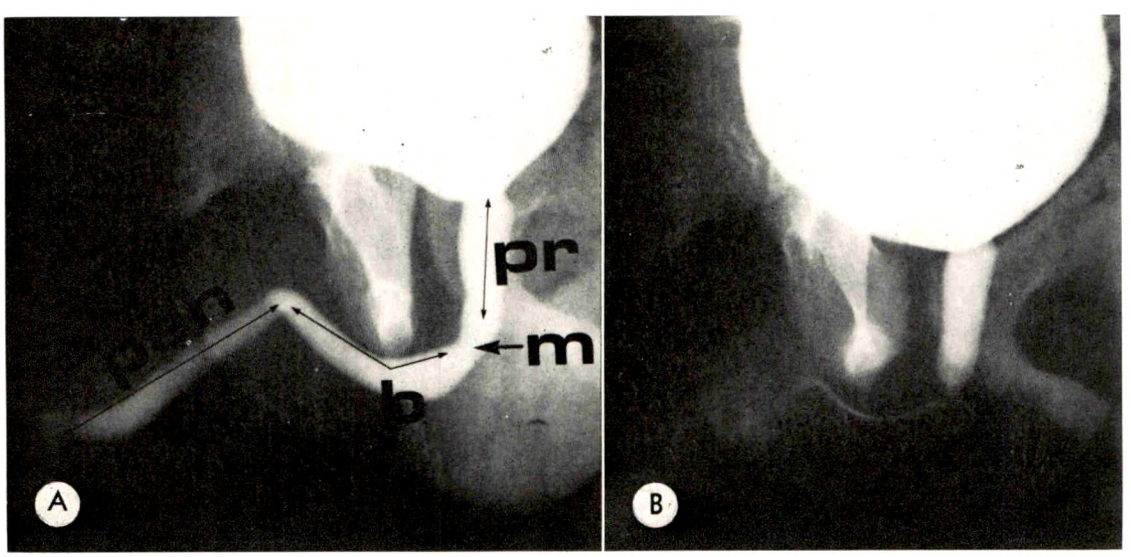


Fig. 1. (*A* and *B*) Normal urethra (pr = prostatic; m = membranous; b = bulbous; pen = pendulous). Micturating cystourethrograms. (*A*) The urethra during micturition. (*B*) The same patient with closure of the external sphincter indicating the site of the membranous urethra.

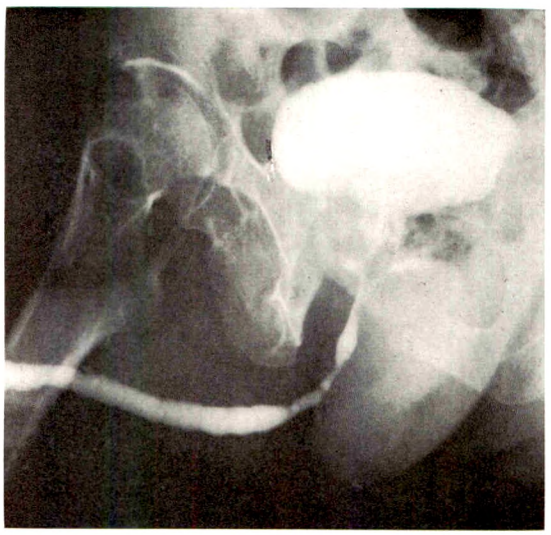


FIG. 2. Retrograde urethrogram. Several strictures of the distal bulbous urethra. Filling of the glands of Littré and contrast medium in the prostate gland.

is surrounded throughout its length by the layers of the urogenital diaphragm which forms the external sphincter. The position of the membranous urethra and the external sphincter are identified on urethrography as the narrowing of the urethral lumen immediately distal to the verumontanum. In addition, on the 45° oblique roentgenogram, a line joining the junction of the upper and middle thirds of both inferior pubic rami represents approximately the attachment of that portion of the external sphincter which traverses the urethra and thus helps in localizing the position of the membranous urethra.[20]

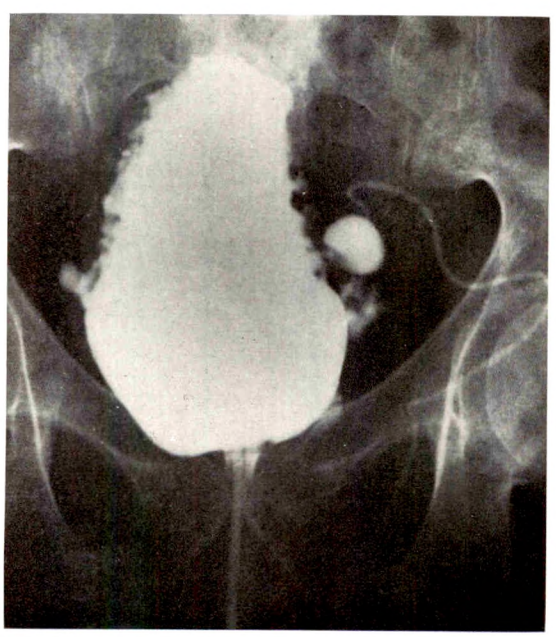


FIG. 3. Cystogram. Neurogenic bladder and filling of the vas deferens.

The bulbous urethra is between 4 cm. and 6 cm. in length and lies between the external sphincter and the peno-scrotal junction (Fig. 1*A*). As described above, the subdivision of the bulbous urethra into proximal and distal portions (Fig. 2; 4; 5; 6; and 7) is of clinical importance to the surgeon. There are 2 Cowper's glands, 1 on either side of the midline and each empties by an individual duct into the proximal bulbous urethra (Fig. 4*A*).

The pendulous urethra (synonym: penile) is the mobile portion of the urethra (Fig. 1*A*) and includes the fossa navicularis which is a small dilatation immediately proximal to the urethral meatus. The glands of Littré are multiple small submucosal glands which are situated along the entire length of the pendulous urethra (Fig. 2). The lacunae urethrales should not be confused with urethral glands; these are normal small folds of mucosa on the surface of the relaxed pendulous urethra.

TECHNIQUES OF URETHROGRAPHY

There are 2 roentgenographic methods for examination of the urethra: retrograde urethrography; and micturating cystourethrography.

RETROGRADE URETHROGRAPHY

This procedure may be performed by using a clamp,[3,11] a Foley catheter,[10] or a vacuum apparatus.[2,16] The majority of our examinations are performed with the Brodny Penile clamp.[3] This has proved very satisfactory, but there are 3 occasional circumstances in which it is difficult to use. Firstly, it is important during the examination to stretch the pendulous urethra in order to obliterate the peno-scrotal junction and to efface the lacunae urethrales. There does come a point at which a further increase in traction may

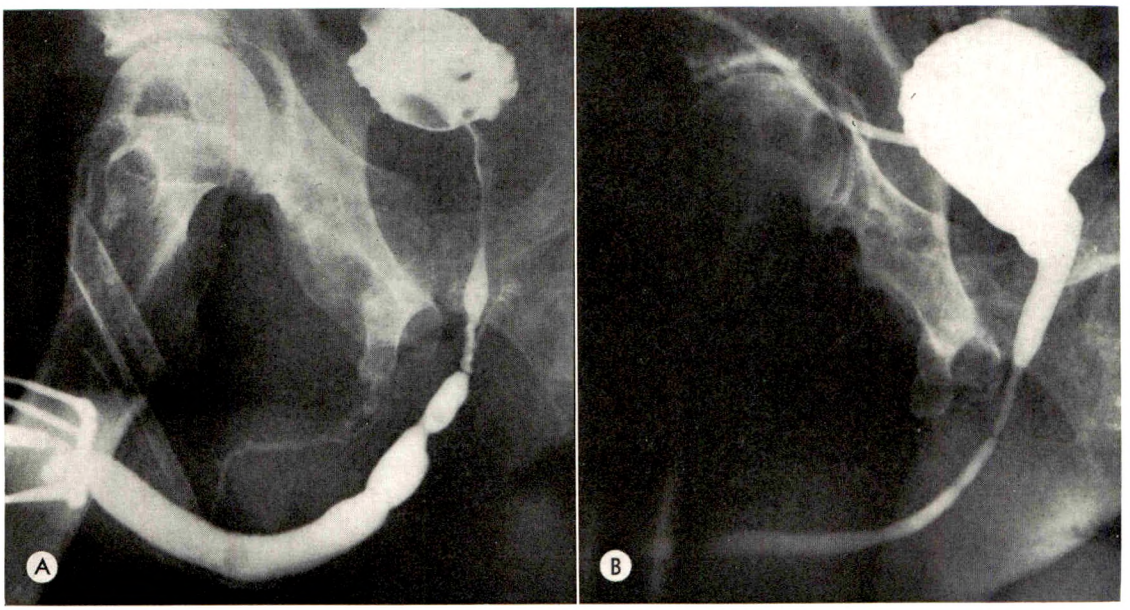


Fig. 4. (*A*) Retrograde urethrogram. Long stricture of the proximal bulbous and membranous urethra and 2 short strictures of the distal bulbous urethra. There is filling of a Cowper's gland and some venous intravasation of contrast medium. (*B*) Micturating cystourethrogram. The prostatic urethra is better demonstrated than on the retrograde examination. The proximal bulbous and membranous stricture is well demonstrated, but the 2 distal bulbous strictures are not identified.

pull the clamp off the penis. This occurs most frequently in noncircumcised patients. If this problem does occur, a Foley catheter with the balloon distended in the fossa navicularis[10] is used, as this will provide excellent traction. However, should the balloon catheter be inadvertently pulled out of the urethra, then the meatus can be traumatized, and although this happens infrequently, it is because of this that the Brodny clamp is used for the routine examination. The second circumstance in which it is difficult to use the clamp is in patients with phimosis. In the

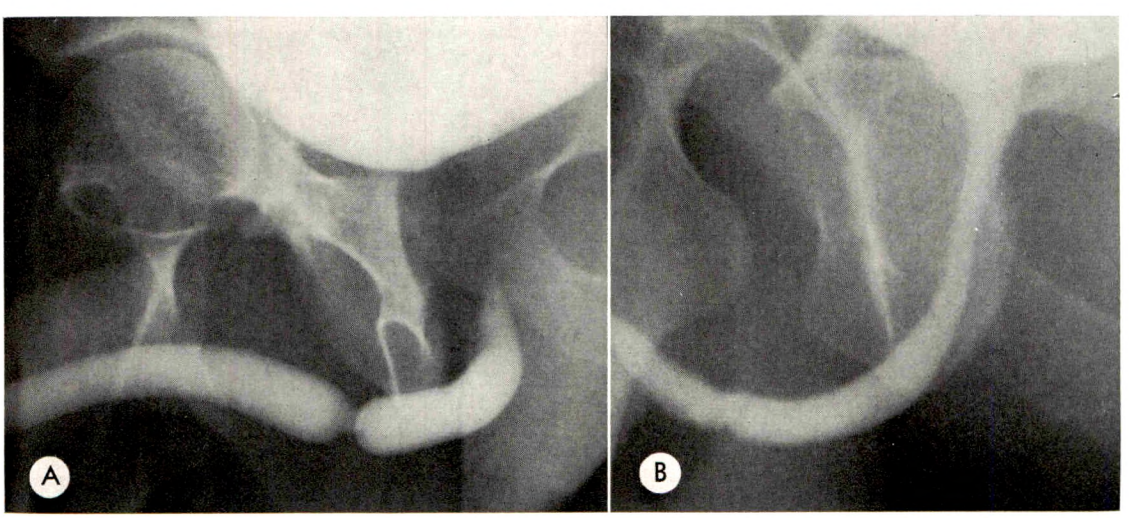


Fig. 5. (*A*) Retrograde urethrogram. Stricture of the distal bulbous urethra, but the prostatic urethra is not adequately filled. (*B*) Micturating cystourethrogram in the same patient. The prostatic urethra is shown to be normal.

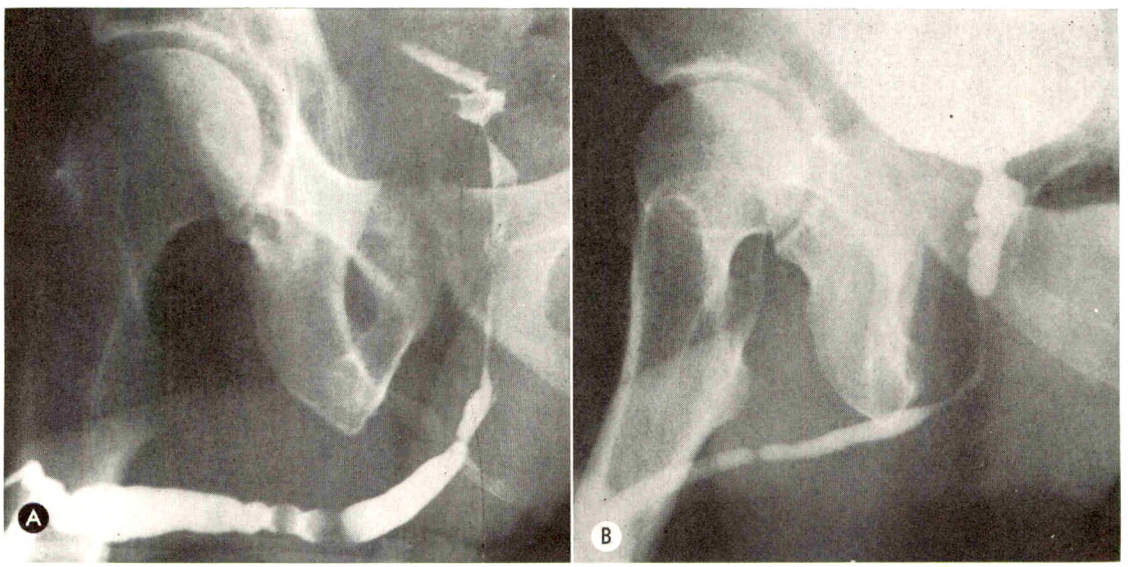


FIG. 6. (*A*) Retrograde urethrogram. Multiple strictures of the pendulous urethra (the filling defects are air bubbles), and a stricture of the proximal bulbous urethra. (*B*) Micturating cystourethrogram in the same patient. This demonstrates that there is also an additional extensive stricture of the bulbous and membranous urethra.

latter patients, the Foley catheter is an excellent alternative. The third situation in which the Brodny clamp is difficult, and sometimes impossible to use, is in patients with hypospadias. This particular problem may be overcome by using a vacuum apparatus,[2,16] developed initially for hysterosalpingography. However, not all of the available cervix adapters are suitable in practice for male urethrography, and those that are satisfactory are often considerably more expensive to use than the Brodny clamp. There is not any advantage in the routine use of a vacuum apparatus over a Brodny clamp that makes this additional expense worthwhile.

Prior to scheduled urethrography, instrumentation is not performed as this will increase the risk of mucosal trauma and of periurethral contrast material extravasation and its complications. However, in our series there were many patients in whom urethrography was performed immediately after instrumentation, either because a stricture had not been previously suspected or for diagnosis after traumatic instrumentation had occurred. This accounts for many of the patients in whom contrast material extravasation or venous intravasation occurred at urethrography.

The patients' urine is examined before the urethrography, and if infected, the examination is postponed. The patient attempts to empty his bladder and is then placed supine on the table and a scout roentgenogram obtained. The Brodny clamp is attached to the penis and the patient is placed in the 45° right posterior oblique position. The penis is placed under traction and 20–40 cc. of contrast material is injected gently; however, if any resistance is felt the injection is immediately stopped and an overhead roentgenogram is taken. By this means, the risk of extravasation due to excess pressure is reduced. A second injection of contrast medium is made in this oblique position employing a modification of the choke cystourethrography described by Pearman and Miller;[19] *i.e.*, without further injection of contrast material the patient is requested to micturate with the injection apparatus still in position. In a small number of cases, this maneuver results in a greater distention of the prostatic urethra than is obtained by the simple retrograde injection alone. The

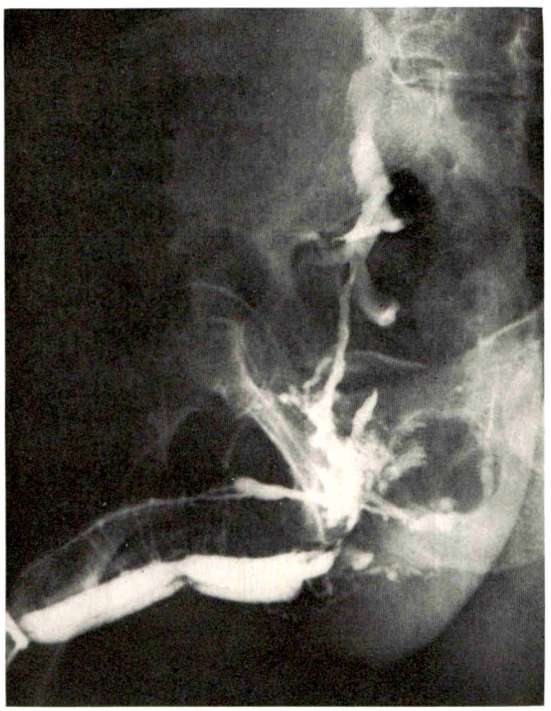


Fig. 7. Retrograde urethrogram. Proximal and distal bulbous urethral strictures. Contrast medium has extravasated into the corpus cavernosum and thence into the venous system. The prostatic ducts also fill with contrast medium.

patient is not given routine antibiotic coverage, but the urine is examined after the urethrography and if it is infected, or if extravasation has occurred, appropriate antibiotic treatment is given.

All injections are carried out with an aqueous contrast medium (Conray 60% or Hypaque 50%) and this is preferred to an oily medium because of the potential hazard of pulmonary oil embolism.[21] A viscous water soluble contrast medium[7,18] (*i.e.*, one that contains a thickening agent such as carboxymethylcellulose) may provide greater distention of the prostatic urethra than is obtained routinely with the low viscosity medium that we use; however, the choke roentgenogram taken during retrograde urethrography and more importantly the micturating cystourethrogram obtained later provide a completely adequate demonstration of the prostatic urethra. It has been suggested that the use of viscous contrast media reduces the risk of periurethral extravasation of the contrast medium.[22] We cannot refute this possibility, but the frequency of extravasation does depend most particularly on the degree of urethral inflammation, recent instrumentation and on an incautious, forceful technique by the inexperienced urethrographer (Fig. 7).

It might seem that all retrograde urethrographies should be performed under fluoroscopic control. However, like Willwerth,[24] it has been found that fluoroscopy is only occasionally an advantage. The majority of our retrograde urethrographies are performed without fluoroscopy but with the overhead tube alone. Fluoroscopy is reserved for those rare occasions when the overhead roentgenograms have not delineated the pathologic anatomy satisfactorily. Routine fluoroscopy has the disadvantages of a more cumbersome technique and increased radiation exposure to the patient and urethrographer, and it has not been found that these disadvantages are outweighed by the advantage of injection under direct vision.

MICTURATING CYSTOURETHROGRAPHY

Following a scout roentgenogram, the bladder is filled with water soluble contrast medium, Hypaque 25%, the total volume varying in different patients between 200–500 cc. The contrast medium is introduced into the bladder *via* a transurethral catheter. In many patients, the urethral stricture is often so severe that suprapubic cystostomy has been performed and then this catheter is utilized. In an occasional patient, it is necessary to perform a suprapubic percutaneous needle puncture of the bladder in order to introduce the contrast agent. An overhead roentgenogram is taken in the supine 45° right posterior oblique position during micturition. Next, the patient is instructed to hold his breath, and to actively attempt to cease micturition at which point an additional film is exposed. This maneuver will cause the external sphincter to contract (Fig. 1*B*) and thus identify the membranous portion as well as providing further

distention of the prostatic urethra. Finally, a postvoiding overhead roentgenogram is taken.

THE ROENTGENOLOGIC PROTOCOL FOR THE INVESTIGATION OF A URETHRAL STRICTURE

A retrograde urethrography is performed. The Brodny clamp is routinely used, but if there is any difficulty with traction or phimosis, an 8F Pediatric Foley catheter is utilized. At the same examination, an intravenous pyelogram is obtained to assess the proximal urinary tract, the main purpose being to show any evidence of hydronephrosis or chronic pyelonephritis.

A micturating cystourethrogram is obtained at the same time if a suprapubic catheter is *in situ*. Brooks and Bateson[4] perform the retrograde urethrography and the micturating cystourethrography as immediately consecutive procedures with introduction of all of the contrast medium *via* the urethra. However, this is frequently impossible when a severe stricture is present, and it only represents additional urethral trauma in those patients in whom periurethral collections of contrast material may have occurred on the retrograde study. Unlike the method of Fumerton and Macewan,[6] the micturating cystourethrography is rarely performed by solely using the intravenous injection of contrast medium; we do not reliably obtain sufficient contrast density by this means, and on one occasion the findings were misleading and a short stricture was not detected. However, this method can be useful on those occasions when it might be difficult to otherwise fill the bladder with contrast material.

Although the retrograde urethrography or the micturating cystourethrography alone may, on occasion, satisfactorily distend the entire urethra proximal and distal to a stricture, this is a relatively infrequent occurrence. The 2 examinations are complementary, and both are essential in order to give the urologist the maximum information as to the extent of the urethral disease (Fig. 4; 5; and 6).

THE ROENTGENOLOGIC PROTOCOL FOR THE INVESTIGATION OF A URETHRAL RUPTURE

Scout roentgenograms of the pelvis and abdomen are taken, and a retrograde urethrography is performed. It is very important that the urethrography be carried out prior to insertion of a catheter into the bladder, as this can by-pass a small tear and mask extravasation, or it can make a small laceration more extensive or even complete. If the retrograde urethrogram is normal, then a catheter is passed and a cystogram obtained without micturition. The catheter is left in the bladder, and finally an intravenous pyelogram is obtained.

ROENTGENOLOGIC FINDINGS AND THEIR SIGNIFICANCE IN URETHRAL STRICTURE

Urethrography will demonstrate the presence, site, multiplicity (Fig. 6, *A* and *B*), extent, and caliber of strictures. In addition to the stricture, complications may be detected in the periurethral tissues, the bladder, or kidneys.

As described earlier, the site of a urethral stricture is of importance to the surgeon. In our series of 68 patients with urethral stricture, 108 strictures were demonstrated at urethrography. Of these 108 strictures, 62 (57 per cent) were in the bulbous, 42 (39 per cent) were in the pendulous, 2 (2 per cent) were limited to the membranous, and 2 (2 per cent) were in the prostatic urethra. Further, of the 62 bulbous strictures, 28 (45 per cent) were designated as involving the proximal bulbous urethra and 34 (55 per cent) were situated solely in the distal bulbous urethra. Additionally, of the 68 patients with stricture, 48 (70 per cent) had single strictures and 20 (30 per cent) had multiple strictures.

The periurethral complications include: filling of the prostatic ducts (Fig. 2; 7; and 8), ejaculatory ducts, Cowper's ducts (Fig. 4), glands of Littré (Fig. 2), abscess, diverticulum (Fig. 8), sinus or false passage (Fig. 8; and 9), fistulae (Fig. 10), extravasation, venous intravasation (Fig. 4; and

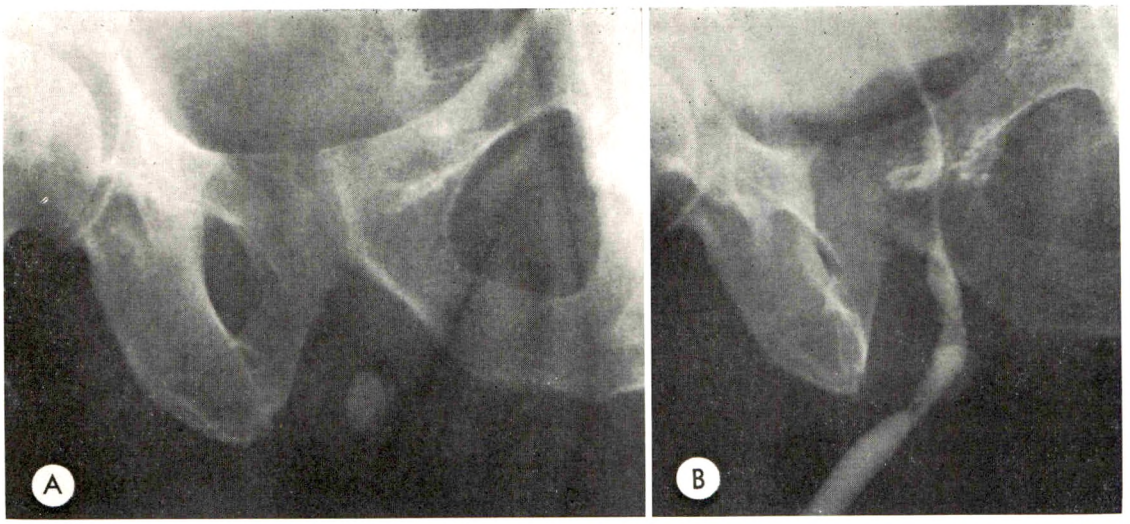


Fig. 8. (*A*) Scout roentgenogram shows a calculus. (*B*) Retrograde urethrogram. The calculus is in a urethral diverticulum proximal to a distal bulbous stricture. A false passage is present immediately distal to the diverticulum. There is also an additional stricture of the proximal bulbous urethra.

7) or urethral calculus (Fig. 8). In this series of 68 patients with urethral stricture 47 (69 per cent) showed at least one of these periurethral collections of contrast material, and the most frequent was filling of the prostatic ducts. None of these appearances are seen with a normal urethra,[17,18] and none were seen in our series of 20 clinically and roentgenologically normal urethras. When demonstrated, these appearances imply either a stricture, inflammatory urethral disease, recent instrumentation, or previous surgery (frequently prostatic) to the lower urinary tract.

Furthermore, the demonstration of these periurethral collections of contrast medium is of clinical importance. Dilated ducts, sinuses, diverticula, and abscesses act as potential sources of infection as well as hazards during dilatation. Their presence or absence will be important factors in the consideration of conservative or surgical management.

In this series of 68 patients with urethral stricture, 49 (72 per cent) showed varying degrees of bladder trabeculation or diverticula formation, 4 (6 per cent) showed roentgenographic changes of chronic pyelonephritis, and 6 (9 per cent) of hydronephrosis. There were no examples of renal calculi, and only 1 case with bladder calculi.

Roentgenologic Findings in Urethral Rupture

Urethral rupture may result from either instrumentation or from external injury. Instrumental rupture may occur during attempted catheterization when an un-



Fig. 9. Retrograde urethrogram. Stricture of the proximal bulbous urethra and a false passage. It is not possible to determine which is the true lumen in the region of the stricture.

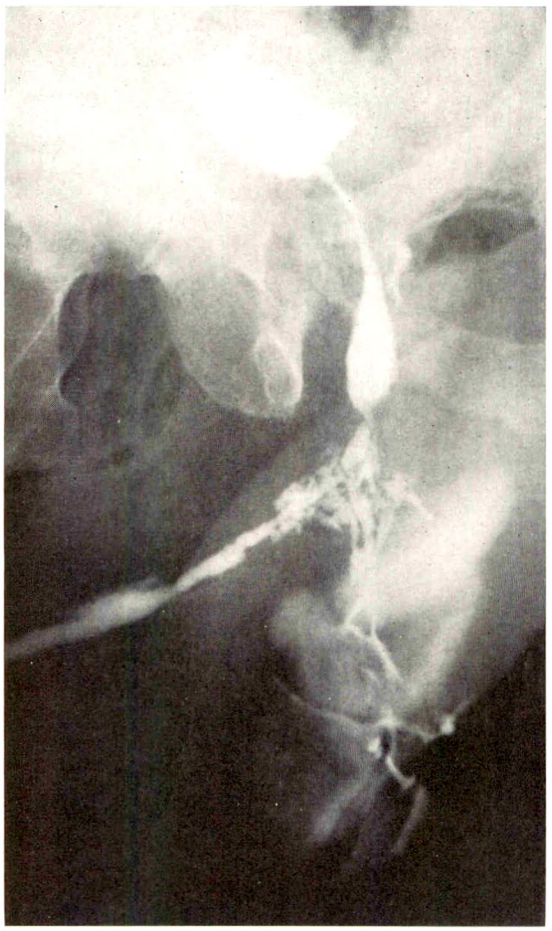


FIG. 10. Retrograde urethrogram. Extensive stricture throughout the bulbous urethra with urethrocutaneous fistulae. There is contrast medium within the prostate gland.

suspected stricture is present, during attempted dilatation of a known stricture, or during unskilled endoscopy. The site of rupture may be either in the region of a stricture, in the bulbous portion where the urethra bends, or in the prostatic urethra; however, most do occur in the bulbous urethra.

Pelvic fractures most frequently result in rupture of the urethra either at the junction of the prostatic and membranous portions, or in the prostatic urethra itself. The urogenital diaphragm has a very strong attachment to the bony pelvis and its fascial layers make the membranous urethra a rigidly fixed structure. Thus, a fractured pelvis may cause complete transection of the apex of the prostatic urethra at this fixed point. Straddle injuries, on the other hand, frequently cause rupture at the bulbous-membranous junction as the urethra is struck against the overlying symphysis pubis, but they may also, on occasion, rupture or traumatize any part of the bulbous, membranous or prostatic urethra.

Rupture of the urethra proximal to its membranous portion may cause extravasation which can be indistinguishable from that caused by an extraperitoneal rupture of the bladder.[23] The rupture may result in either a very small or a large extravasation of contrast material which diffuses into the tissues of the pelvic floor and prevesical space. A large extravasation often appears as a "sunburst" collection (Fig. 11) and there may be an associated indentation of the bladder by the collection of blood and urine. It must be noted, however, that a similar bladder indentation can be caused by a large hematoma without an associated urethral rupture. In this circumstance, although it is possible that a very small urethral tear may rapidly seal over, a normal retrograde urethrogram will strongly suggest that such an appear-

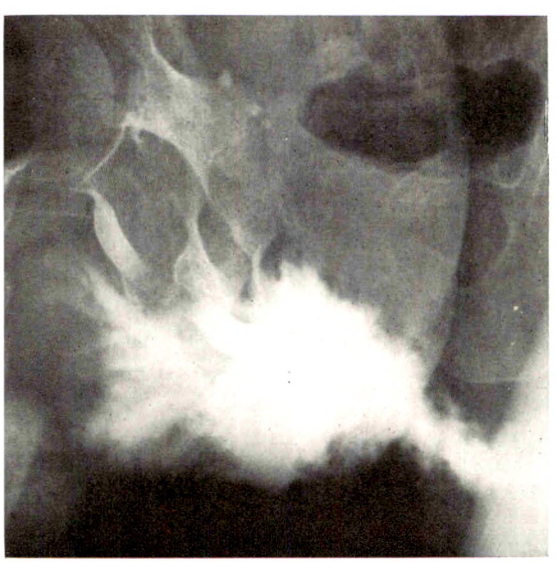


FIG. 11. Retrograde urethrogram. Pelvic fracture. The contrast medium extravasates from the urethral transection at the prostatic-membranous junction.

ance is due solely to hematoma from the pelvic fracture.

SUMMARY

The incidence of gonorrhea is increasing, and this may well be accompanied by an increase in the frequency of eventual urethral strictures in the male. At the present time, there has also been a marked increase in the number of plastic operations being performed for urethral stricture.

Radiology provides accurate preoperative assessment of the entire urethra. Bulbous urethral strictures are the most common, and their location in the bulb as seen at urethrography will indicate the technical difficulty that will be encountered at urethroplasty. In this regard, both retrograde urethrography and micturating cystourethrography are essential for the preoperative assessment of stricture.

The investigation of urethral rupture requires a different approach to that of urethral stricture. The protocol suggested will prevent a small rupture from being undiagnosed, and will also prevent such a rupture from being made more extensive or even complete.

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ACUTE REACTIONS TO UROGRAPHIC CONTRAST MEDIUM*

INCIDENCE, CLINICAL CHARACTERISTICS AND RELATIONSHIP TO HISTORY OF HYPERSENSITIVITY STATES

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ACUTE reactions to the intravenous injection of iodinated contrast medium for excretory urography pose a hazard familiar to most physicians. The clinical characteristics and incidence of reactions to these agents have been documented in the medical literature by Ochsner and his associates,[3,7–9] Ansell[1] and others.[5,6,11,12] But in spite of the availability of this large body of detailed information, differences of opinion, confusion and misunderstanding persist as regards the relative incidence of acute reactions in large patient populations, their significance in specific clinical settings and their relationship to pre-existing "hypersensitivity" or "allergic" states.

It is the purpose of this study to add to the body of available data observations on the incidence and clinical characteristics of reactions to urographic contrast medium in a large, uniform series of patients and to explore the relationship between a history of "allergy" and acute reaction to contrast medium.

METHOD

The study group consisted of 32,964 consecutive outpatients at the Mayo Clinic who were referred to the Department of Diagnostic Roentgenology for excretory urography during a 27 month period. Included were 15,594 males and 17,370 females who ranged in age from infancy to 91 years. No patient on whom excretory urography was undertaken was excluded from the study.

The first 9,934 consecutive patients were studied to determine what relationship exists between a history of known or suspected allergy or hypersensitivity and acute reactions to urographic contrast medium.

Prior to injection of contrast material, each patient in this group was questioned in detail on the following relevant points and the data tabulated:

1. Presence or absence of a history of "allergy" or "hypersensitivity."
2. Specific allergen or allergens if known (*i.e.*, pollens, foods, drugs, iodides, urographic medium, etc.).
3. Clinical characteristics of previous hypersensitivity response if known (*i.e.*, hives, asthma, nausea and vomiting, hay fever, etc.).

Among the remaining 23,030 patients in the study, detailed historical data relating to past history of allergy or hypersensitivity were obtained prior to injection but were tabulated for analysis only in those patients who developed acute reactions. Reactions in all 32,964 patients were recorded as to clinical characteristics, severity and treatment for later analysis.

TECHNIQUE OF EXAMINATION

All patients were examined in a single radiologic facility under highly uniform conditions. The contrast medium used in all examinations was a mixture of sodium and meglumine diatrizoate 69 per cent (Reno-

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vist-Squibb). A routine intravenous test dose of approximately 1 ml. of this medium was employed. The dose administered ranged from as little as 1 ml. test dose only to as much as 150 ml. Standard doses in children ranged from 10 ml. to 30 ml. depending on age and weight. In adults, a standard dose of 30 ml. was used for most patients with known or suspected urologic disease, and a dose of 50 ml. was employed for patients studied because of hypertension and some large patients with suspected urologic disease. Larger doses ranging to 150 ml. were used on a routine basis in patients with chemical evidence of depressed renal function. In an occasional patient, failure of adequate venopuncture or extravascular extravasation of contrast medium during injection resulted in administration of doses under 30 ml.

DEFINITIONS

The response of patients to injection of contrast medium was tabulated in one of 3 categories:

1. No clinically significant response.
2. Minor side effects.
3. Acute reactions.

For the purpose of this study, certain arbitrary definitions were necessary. Patients considered to have *no clinically significant response* include those with no symptoms from injection as well as those who experienced mild transient symptoms which appeared to be the result of normal physiologic effects of injection of the contrast medium itself and were judged to be of no clinical significance. These included mild hot flush, metallic taste in the mouth, mild sense of nausea without retching or vomiting, "peculiar" sensation over the body, tingling in the face or extremities and pain in the arm associated with extravasation of contrast medium. It is worthy of note that one or more of these minor symptoms were experienced by most patients.

Similar symptoms were recorded as *minor side effects*, when unusually prominent or severe. For example, nausea with retching or vomiting was recorded as a minor side effect as was pain in the arm associated with venospasm.

Acute reactions were defined as symptoms and signs considered to be true idiosyncratic responses to contrast medium. These included urticaria in all its forms, mucous membrane and cutaneous edema, bronchospasm, convulsions, hypotension, shock, cardiac arrest and death.

RESULTS

INCIDENCE AND CLINICAL CHARACTERISTICS

The incidence of reactions and the types encountered are tabulated in Table 1.

No clinically significant response was recorded in 30,713 patients (93.2 per cent).

Minor side effects were observed in 1,683 patients (5.1 per cent). These included nausea with retching and vomiting in 1,626; a strong but transient hot flushing or sense of warmth, either alone or with nausea and vomiting, in 37; subjective complaint of "faintness" not found to be associated with hypotension in 7; cough associated with transient "fullness" in the throat and flushing in 7; and pain in the arm secondary to venospasm but not the result of extravasation in 6. No patient in this group with minor side effects required treatment and all symptoms were transient.

Acute reactions were recorded in 568 (1.72 per cent) of the 32,964 patients. The reaction developed during the injection or

TABLE 1

REACTIONS
INCIDENCE AMONG 32,964 PATIENTS

Type	No.	Per Cent
None	30,713	93.2
Minor side effects	1,683	5.1
Acute reactions total	568	1.72
Dermal	476	1.44
Nasal and mucosal	41	0.12
Cardiovascular	24	0.07
Respiratory	16	0.05
Neurologic	3	0.01
Other	8	0.02

within 5 to 10 minutes after completion of the injection in all patients but 2. In these the reaction developed within 1 hour of injection. No examples of delayed reactions to injection of contrast medium were recognized but no special effort was made to identify reactions of this type.

The clinical characteristics of acute reactions are tabulated in Table II. While more than one symptom was recorded in a large number of patients, it was necessary to oversimplify the analysis and tabulate reactions by the predominant symptom or sign. Three hundred thirty-four were classified as mild, 204 as moderate and 30 as severe.

Dermal reactions were present in 476 with hives the dominant finding in 444 and a diffuse erythematous rash in 32. Hives varied in extent from solitary to generalized and were accompanied by itching and in severe cases by moderate cutaneous edema. (Erythematous rash associated with cardiovascular symptoms is discussed below.)

Nasal and mucosal symptoms were observed in 41. Of these, periorbital edema with or without itching of the eyes was most frequent with 19 examples recorded. Nasal congestion with sneezing and rhinitis was present in 17 and angioneurotic edema with severe diffuse swelling of the skin and mucous membranes in 5.

Cardiovascular reactions were encountered in 24 (Table III). These were of 3 types: syncope associated with transient hypotension in 8; hypotension (shock) accompanied by a diffuse erythematous rash in 15; and cardiovascular collapse with cardiac arrest and death in 1.

Respiratory symptoms developed in 16. These included symptoms of bronchospasm or bronchial asthma in 9 and episodes of laryngeal edema with signs of airway obstruction of mild to moderate severity in 7.

Neurologic symptoms were seen in 3. All were grand mal seizures not accompanied by other signs or symptoms.

Miscellaneous other symptoms thought to represent idiosyncratic reactions were seen in 8 patients. In 3, shaking chills accompanied by diarrhea and profuse sweating developed within one hour of injection. These symptoms were terminated by injection of antihistamine and epinephrine on an empiric basis. In 3, parotid swelling developed within minutes of injection. In 2 marked restlessness with severe nausea and vomiting were observed and a single patient developed profuse sweating with no other recognizable symptoms.

TABLE II

CLINICAL CHARACTERISTICS
568 ACUTE REACTIONS

Type	Severity		
	Mild	Moderate	Severe
Dermal (476)			
Hives	271	170	3
Erythema	10	21	1
Nasal; mucosal (41)			
Periorbital edema	13	6	
Sneezing, congestion	17		
Angioedema			5
Cardiovascular (24)			
Syncope	8		
Shock			15
Cardiac arrest			1
Respiratory (16)			
Asthma	5	3	1
Laryngeal	6		1
Neurologic (3)			
Grand mal seizure			3
Other (8)	4	4	

TABLE III

CARDIOVASCULAR REACTIONS

Syncope	
No treatment	8
Shock with erythema	
Moderate—not hospitalized	5
Severe—hospitalized	10
Cardiac arrest	
Death	1

SEVERE REACTIONS

Reactions classified as severe occurred in 30 patients (Table IV). They occurred at a rate of about 1 for each 1,100 patients examined and represented 5 per cent of the total number of acute reactions in this series. Only about one-third appeared life threatening at the time they occurred, but 1 of these proved fatal in spite of prompt and vigorous treatment.

Fifteen patients with reactions classified as severe developed a diffuse erythematous rash accompanied by hypotension as the major symptoms. Bradycardia (pulse 30 to 40/min.) accompanied the hypotension in 9 and tachycardia in 6. The reaction appeared within 3 to 5 minutes after injection of medium in all cases. It was preceded by nausea and vomiting and the patients almost invariably lost consciousness. Airway obstruction due to relaxation of the tongue developed in 6. Many patients had urinary and fecal incontinence during the episode. Neither myocardial infarction nor cardiac arrest was observed in this series but subsequently, we have seen an acute myocardial infarction develop in several patients with this syndrome.

The cutaneous rash which accompanied these reactions was diffuse, of moderate to severe intensity, and usually accompanied by perioral palor. Neither bronchospasm nor pulmonary edema was observed.

Each of the patients with this syndrome required vigorous treatment. All recovered within 30 minutes to 2 hours. In 5, the recovery was so prompt and complete that the patient was dismissed from the Radiologic Department after a period of rest. Ten patients with more severe reactions, or reactions which cleared more slowly, were hospitalized for overnight observation, but all recovered with no residual effects.

Five patients developed angioneurotic edema with diffuse mucocutaneous swelling over the entire body. Hives or cutaneous erythema accompanied the edema in all patients and 1 patient had associated parotid swelling. None was accompanied by bronchospasm or cardiovascular symptoms and all responded promptly to treatment with epinephrine and hydrocortisone. No late sequelae were noted.

TABLE IV

SEVERE REACTIONS TO UROGRAPHIC MEDIUM

Type	Patients
Shock with erythema	15
Angioneurotic edema	5
Hives or erythematous rash	4
Generalized convulsion	3
Asthma	1
Laryngospasm	1
Cardiovascular collapse with cardiac arrest and death	1
Total	30

Severe hives or cutaneous rash was present in 4 patients. One was accompanied by mild bronchospasm with moderate wheezing. The remainder had associated mucocutaneous edema, pruritus and rhinitis. All responded promptly to treatment. None required hospitalization.

Three patients developed grand mal seizures not accompanied by other signs or symptoms suggestive of idiosyncratic response to the contrast agent. Two of these patients had a history of similar seizures and were on anticonvulsive therapy. The third patient had no prior history of convulsive disorder and when last seen had had no repetition of seizures. No specific treatment was given to these patients. All 3 were hospitalized for observation and completion of medical evaluation.

One patient developed a severe asthmatic attack relieved by aminophyllin and one acute laryngeal edema with mild cyanosis, dyspnea and hives relieved by epinephrine.

Cardiovascular collapse with cardiac arrest and death occurred once. The patient was a 52 year old male studied for complaints suggesting urinary outlet obstruction from enlarged prostate. Moderate nausea and retching occurred early during the injection and injection was temporarily interrupted until these symptoms subsided. The reaction developed precipitously im-

mediately after the injection was completed. It consisted of loss of consciousness, apnea and cardiac arrest with deepening cyanosis. Immediate resuscitative measures were undertaken. The airway was opened, cardiac massage instituted, and appropriate drug therapy was underway within 2 to 3 minutes of onset. The therapy was effective and the patient regained consciousness, but on arousal was panic stricken and pulled out his airway and intravenous needles. Subsequently, difficulty was encountered reestablishing his airway and in administration of intravenous drugs. Cardiac arrest recurred and in spite of the availability and use of all appropriate resuscitative techniques, he died.

RELATIONSHIP OF DOSE TO REACTION

The relationship of dose to reaction is seen in Table v.

The test dose alone induced nausea and vomiting in 28 and acute reactions in 9 patients. The acute reactions were mild in 3, moderate in 4 and severe in 2. One of the severe reactions was a grand mal seizure and the other severe generalized erythematous rash. In all 28 patients who experienced nausea and vomiting the examination was continued with injection of the full dose of contrast medium after the initial symptoms subsided. The nausea and vomiting recurred in only 6 of these and subsided promptly after injection was completed. In 6 of 9 patients with acute reactions to the test dose, the examination was terminated but in 3, all of whom had mild to moderate hives, the injection was completed following administration of intramuscular antihistamine because of the importance of the clinical problem under study. No further reaction developed in any of these patients.

With doses under 30 ml., minor side effects were recorded in 4.1 per cent; with doses of 30 ml., in 5.0 per cent; and with doses of 50 ml., in 5.3 per cent. With doses of 30 to 50 ml., acute reactions were recorded in 1.67 and 1.73 per cent, respectively, with no apparent difference in the relative incidence of mild, moderate and severe reactions with these doses. The number of reactions recorded in patients receiving doses under 30 ml. or over 50 ml. is small and the calculated incidence rates of doubtful statistical significance.

TABLE V

RELATIONSHIP OF DOSE TO REACTION

	Patients	Minor Side Effects		Acute Reaction				
Dose (ml.)	No.	No.	Per Cent	Mild	Moderate	Severe	Total No.	Per Cent
1 (test)	32,968	28	0.09	3	4	2	9	0.03
<30	823	33	4.1	9	3	0	12	1.5
30	21,168	1,043	5.0	205	129	20	354	1.67
50	10,626	562	5.3	111	65	8	184	1.73
>50	247	13	5.3	4	2	0	6	2.4
Unknown	37	4	11.0	2	1	0	3	7.8
	Total	1,683	5.1	334	204	30	568	1.72

RELATIONSHIP OF HISTORY OF ALLERGY TO ACUTE REACTION

Nine thousand, nine hundred thirty-four patients were studied with specific reference to the relationship between a history of allergy or hypersensitivity and the occurrence of reactions (Table VI): 7,445 (75 per cent) gave no allergic history (negative history); 2,489 (25 per cent) reported symptoms which the patient thought or had been told represented an allergic or hypersensitivity state (positive history). On close scrutiny many of these complaints are not true "allergies." For example, many patients reported symptoms of nausea and vomiting associated with injection of narcotic drugs for pain relief as "allergies." However, the data as recorded accurately represent the incidence of a history of "allergy" as obtained from the patient in day to day practice.

Three hundred forty-five (4.6 per cent) of the patients with a negative history experienced mild side effects consisting primarily of nausea and vomiting during examination and 89 (1.2 per cent) developed acute reactions. One hundred sixty-six (6.9 per cent) patients with a positive history developed minor side effects and 74 (3.0 per cent) developed acute reactions.

Table VII relates the incidence of reactions to a number of common allergens and types of allergic response claimed by patients. It is, of course, usual to find patients with multiple allergies. In this analysis, major allergens or types of symptoms are tabulated separately and cause a discrepancy between the total number of patients reporting "allergies" in this series (2,489) and the number of patients listed by type of "allergic" history in this table (2,815). Acute reactions were associated with a history of asthma in 6 per cent; hay fever in 4 per cent; hives of unknown cause in 7 per cent; miscellaneous food allergies in 6 per cent; seafood allergy in 6 per cent; inorganic iodides in 13 per cent; and previous reaction to injection of urographic contrast medium in 20 per cent. Among 1,854 other "allergic" states were 1,374 patients with sensitivity to drugs such as penicillin, tetracycline, chloromycetin, sulfa, morphine, codeine, aspirin and 480 with sensitivity to a wide variety of other substances. The incidence of reactions in these patients was 2 per cent, which is less than twice the incidence expected in the patients who reported no history of "allergy."

Of special interest were 121 patients who claimed sensitivity to a previous injection of iodinated contrast medium for excretory urography. On careful questioning or review of the recorded history prior to injection, it was determined that 66 had had reactions consisting of hives, orbital edema, nasal congestion, mild asthma or other mild forms of acute reaction. The remaining 55 patients had had episodes of nausea and vomiting which they or their physician had interpreted as a reaction. In many instances these patients had been advised never to have urography again. No patient who gave a clear history of previous severe reaction such as hypotension, severe asthma or severe hives was examined again in this series. Forty-three of the 121 patients were

TABLE VI

INCIDENCE OF HISTORY OF ALLERGY AND REACTIONS
(9,934 Patients)

Allergic History	No. of Patients	Minor Side Effects		Acute Reactions	
		No.	Per Cent	No.	Per Cent
Negative	7,445	345	4.6	89	1.2
Positive	2,489	166	6.9	74	3.0

TABLE VII

INCIDENCE OF REACTIONS RELATED TO TYPE OF ALLERGIC HISTORY*

Allergy Reported by Patient	No. of Patients	Patients with Reactions	
		No.	Per Cent
Asthma	140	9	6
Hay fever	316	13	4
Hives (unknown cause)	69	5	7
Food allergy	212	12	6
Seafood	64	4	6
Urographic contrast medium	121	24	20
Iodine (iodide)	39	5	13
Other†	1854	37	2

* Many patients reported multiple allergies. These are tabulated separately and cause a discrepancy between total number of patients reporting allergies in this series (2,489) and the number of patients listed here by type of allergic history.

† Primarily sensitivity to antibiotics, sulfa drugs and narcotics, but includes many other drugs and substances.

premedicated with antihistamine prior to examination. None reacted to the intravenous test dose.

Among the 55 patients with a history of nausea and vomiting only 1 developed a reaction. This consisted of mild hives. Of the 66 patients with a definite history of hives or other reaction, 23 (35 per cent) developed an acute reaction to examination in this study. Reactions in these patients consisted of hives in 22 and mild asthma in 1. The hives ranged from a single hive to moderate generalized hives. A diffuse, mild erythematous rash accompanied hives in 3 patients and mild periorbital edema in 1 patient. Nineteen of the patients who developed a reaction had been premedicated.

COMMENT AND CONCLUSIONS

Acute reactions to injection of urographic contrast medium remain one of the most vexing problems faced by any physician who does excretory urography in his day to day practice. Minor side effects were recorded in 5.1 per cent of this series and acute reactions in 1.72 per cent. The vast majority of these reactions were temporarily unpleasant but limited in duration and severity and did not represent a serious threat to the patient. Severe reactions on the other hand, developed in 0.09 per cent of the patients (1 of each 1,100 examined) and the severity of their symptoms made prompt and vigorous treatment necessary. One-third appeared life threatening and 1 patient died.

Much of the difficulty encountered in dealing with reactions results from the fact that they cannot be predicted by any known means and attempts at prevention by premedication have not proved effective in reduction of the incidence of severe reactions or death.[1,9]

In this series all patients had a preliminary intravenous test dose. Nine had a positive response in the form of an acute reaction to the test dose, 2 of which were severe. In the remaining 559 patients with acute reactions, the test dose gave no hint that a reaction would occur. Thus, the results in this study confirm the opinion of many authors: that the intravenous test dose is of no value in prediction of reactions; that it is potentially hazardous to the patient; and that its continued use cannot be justified on medical grounds. It follows then that as no medical justification for pretesting exists, likewise no medico-legal justification exists and pretesting should be abandoned.

The association between a history of "allergy" and acute reactions to contrast medium was investigated to gain further insight into the nature and extent of this association. The incidence of reactions in patients with a history of "allergy" was 2.5 times that in patients who claimed no such history (3.0 per cent as compared with 1.2 per cent). When specific types of allergic responses or allergens are considered (Table VII), it is evident that in the presence of asthma, hay fever, hives of unknown etiology, certain food allergies, sensitivity to urographic contrast medium and iodides, there is a substantial increase in incidence

of reactions but that among other common allergic or hypersensitivity states the increase is minor. The type and severity of reactions among patients with a history of allergy was not tabulated separately for inclusion in this report but was observed to be essentially the same as for patients with no history of allergy. Only 4 (13 per cent) of the 30 severe reactions occurred among patients with a history of allergy, whereas allergy was claimed by 25 per cent of patients.

Twenty-three of 66 (35 per cent) patients with prior reaction to urographic contrast medium and 1 of 55 (1.8 per cent) with a prior "reaction" consisting of severe nausea and vomiting developed reactions when re-examined. No reaction in this group was more severe than the previously recorded reaction or reactions and in most instances was identical in clinical characteristics. In contrast to the observations of Ansell[1] we have not encountered a patient in whom a second, third or even fourth reaction was more severe than the initial reaction. Thus, there is no evidence in this series or in our over-all experience outside this study that would suggest an increase in sensitivity to contrast medium with repeat examination.

A striking feature of reactions in a given patient with known sensitivity to urographic contrast medium was the inconstancy with which reactions occur from examination to examination. Five patients in this group had had multiple urographic examinations (as many as 6) since their first reaction. Each experienced reactions with some but not all subsequent examinations even when the contrast medium and the dose were unchanged. In a typical example, a patient with a history of 6 previous examinations had experienced hives on his face on 3 occasions and no hives on 3. He was premedicated with antihistamine for only 2 of the 6 examinations and both times developed mild hives. On examination during this study, no reaction developed.

We concluded, as have others,[4,10] that neither a history of specific allergy nor a history of prior mild to moderate reaction from injections of contrast medium is a contraindication to excretory urography. Thus, we believe that these patients should not be denied examination by excretory urography when sound medical indications for its use exist.

SUMMARY

Thirty-two thousand, nine hundred sixty-four consecutive patients were studied with reference to the incidence and clinical characteristics of reactions to a single urographic contrast medium. Mild side effects were recorded in 5.1 per cent and acute reactions in 1.72 per cent. Severe reactions occurred in 0.09 per cent with 1 death from cardiac arrest.

Acute reactions are unpredictable on the basis of history or any known pretesting technique. Pretesting itself is hazardous and gives a false sense of security to both physician and patient. As a consequence of these facts, it is evident that the presently available pretesting techniques serve no useful purpose and should be abandoned.

The first 9,934 patients were studied with special reference to an association between a history of "allergy" or "hypersensitivity" and acute reactions. Reactions were 2.5 times (3.0 per cent as compared to 1.2 per cent) more frequent in patients with a positive history of "allergy." No increase in severity of reactions was noted. With a history of prior mild to moderate reaction to urographic contrast medium, reactions occurred in 20 per cent. No evidence of increasing sensitivity with repeated use was found.

It is concluded that neither a positive history of previous "allergy" nor previous mild to moderate reaction from contrast medium is a contraindication to excretory urography.

Acute life threatening reactions, although very rare, can occur unexpectedly in any patient, at any time. The radiologic team doing excretory urography must be well trained in their recognition and treat-

ment. Emergency equipment, adequate to cope with any reaction,[2] must be on hand and available for immediate use in every radiologic suite where excretory urography is done.

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RADIOLOGIC ASPECTS OF AN ABORTION PROGRAM*

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ABORTION laws have been liberalized in New York and several other states in recent years. In New York, with the most liberal law, abortion is now available on request up to 24 weeks of gestation. The radiologist is in a position to play a helpful role in an abortion program and very likely will be called upon to do so.

The radiologic aspects of abortion may be divided into 2 main categories: (1) first trimester (up to 12 weeks), in which dilatation and curettage, including suction curettage, is the most frequent method for interrupting pregnancy; and (2) second trimester, in which the intraamniotic injection of hypertonic saline is the method generally used to induce abortion between 15 and 24 weeks.

1. DILATATION AND CURETTAGE

Uterine curettage may be followed by immediate and/or delayed complications. The immediate complications are perforation of the uterus, particularly beyond the first trimester (Fig. 1), with intraperitoneal or retroperitoneal hemorrhage and various postabortal infections (endometritis, salpingitis, parametritis, peritonitis). With the development of abortion programs in hospitals and properly equipped clinics, the number of these mishaps will undoubtedly decrease. Delayed sequelae of dilatation and curettage, which the radiologist may expect to encounter, include the development of uterine adhesions or synechiae and incompetence of the internal os.

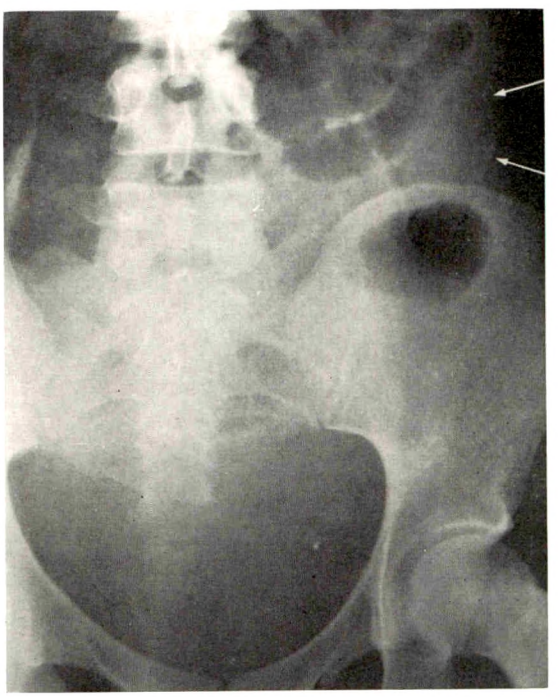


FIG. 1. Perforation of the uterus during uterine curettage, with hemoperitoneum and escape of fetal parts into the peritoneal cavity. The faintly calcified fetal head (arrows) is in the peritoneal cavity. The gestational age was underestimated in this case.

UTERINE SYNECHIAE

Although uterine adhesions consequent to curettage were described by Hald[11] in 1949, the subject was popularized by Asherman[2] in 1950 and has become known in the gynecologic literature as the Asherman syndrome. The adhesions vary in extent, causing partial or complete obliteration of the uterine cavity. In mild cases there are no clinical symptoms and normal menstruation is maintained. With increasing atresia of the uterine cavity, there may be oligomenorrhea, hypomenorrhea, dysmenorrhea, amenorrhea, infertility and abortion.

Roentgen examination is the key to the diagnosis of the Asherman syndrome. The uterine cavity shows bizarre filling defects (Fig. 2*A*), frequently linear or ovoid, which

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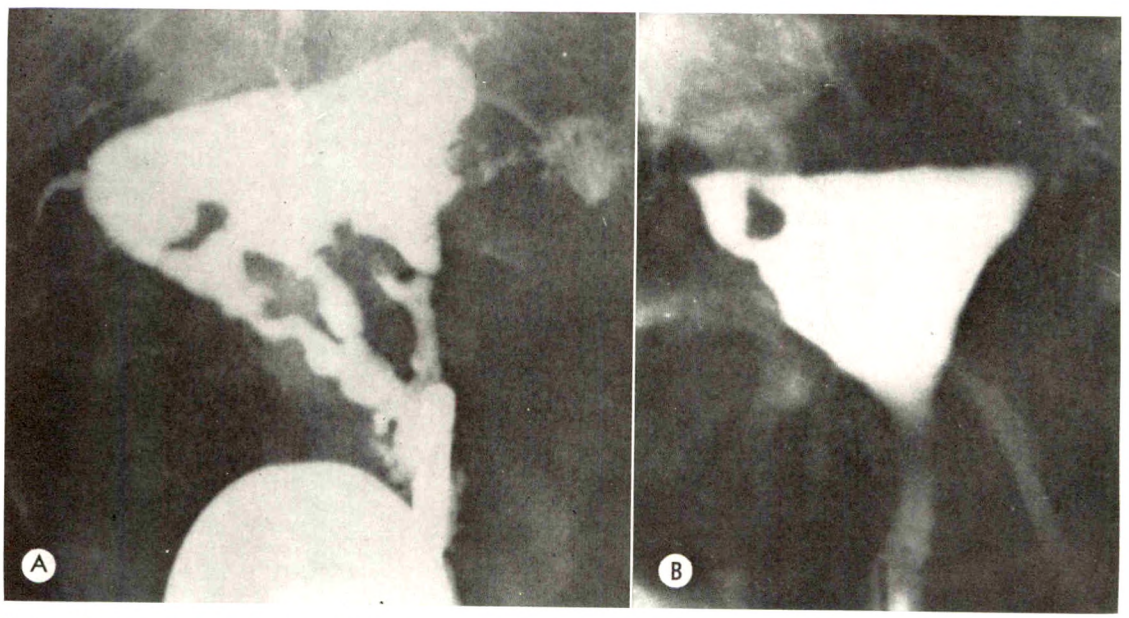


FIG. 2. Postcurettage adhesions (Asherman syndrome). (*A*) Multiple bizarre filling defects are characteristic of uterine synechiae. (*B*) A solitary pear-shaped defect near the right uterine cornu due to adhesions.

may be single (Fig. 2*B*) or multiple, large or very small, generally well demarcated and not effaced by increasing amounts of contrast medium. The radiolucent defects may occasionally branch like a tree, or may resemble the shape of a heart or a window.[2] The distinction from air-bubbles is readily made since the latter are round and inconstant images. Uterine adhesions may develop after one or more curettages of the puerperal uterus.[11] Asherman[2] encountered intrauterine adhesions in 44 out of 65 women who had undergone 2 or more curettages.

Pathologically, the adhesions are formed by delicate strands or by thick fibrous bands; microscopically, they consist of fibrous tissue, endometrial glandular elements or smooth muscle, individually or in various combinations.[8] While curettage is the most common cause of uterine synechiae, they may result also from cesarean section, myomectomy, metroplasty and intracavitary radium insertion. Spontaneous synechiae may develop after healing of an endometritis. Treatment consists of surgical separation of the adhesions, which can be accomplished with a uterine sound. The insertion of a Foley catheter or a contraceptive device into the uterine cavity for 1 to 2 weeks has been advocated to keep the uterine walls apart and prevent recurrence of the adhesions.[5]

CERVICAL ATRESIA

This is the counterpart of the Asherman syndrome in the cervical canal, which becomes distorted and partly obliterated. When attempting a hysterogram, the radiologist has difficulty identifying the external os and cannulating the cervix. The examination usually has to be deferred pending careful dilatation of the cervical canal.

INCOMPETENCE OF THE UTERINE ISTHMUS

The width of the uterine isthmus (internal os) varies during the menstrual cycle; it increases under the influence of estrogens and decreases under the influence of progesterone.[3] Average values range from 2 mm. in the secretory phase to 6 mm. in the proliferative phase of the endometrium.[3] An internal os wider than 10 mm. suggests incompetence (Fig. 3, *A* and *B*), particularly if studied in the pre-

menstrual phase, when physiologic constriction of the uterine isthmus is maximal.[24] Palmer[17] established "gaping" of the isthmus, if an 8 mm. Hegar dilator traversed the internal os without resistance in the nonpregnant uterus.

Various roentgenologic techniques have been described to study the internal os. The simplest method, which we have employed, is a conventional hysterogram in which the lower cervical canal is obturated by means of a rubber acorn.

In the balloon technique, 3–4 ml. of contrast medium is introduced into a balloon within the uterine cavity. Traction is exerted on the balloon and a roentgenogram is obtained. A small amount of contrast medium is then removed from the balloon and further traction is exerted while the roentgenogram is repeated. The balloon now will usually assume the shape of the isthmus and upper cervical canal, thus permitting their evaluation.[19]

In the technique of direct hysterography, which may be difficult and hazardous, a long, fine needle is inserted into the uterine cavity *via* the posterior fornix or through a small posterior colpotomy incision, and 2–3 ml. of contrast medium is injected. In case of isthmic incompetence, there is prompt leakage of the contrast medium through the internal os and cervix into the vagina. Such immediate leakage is considered abnormal, particularly in the luteal phase of the cycle.[26]

Incompetence of the internal os rarely may be congenital or dysfunctional, but is usually due to trauma from repeated dilatation and curettage, criminal abortion, overzealous cervical dilatation, traumatic delivery, high multiparity or incision of the uterine cervix. The incompetent isthmus is an important cause of spontaneous, habitual abortion in the second trimester (Fig. 3, *A* and *B*).

The delayed sequelae of uterine dilata-

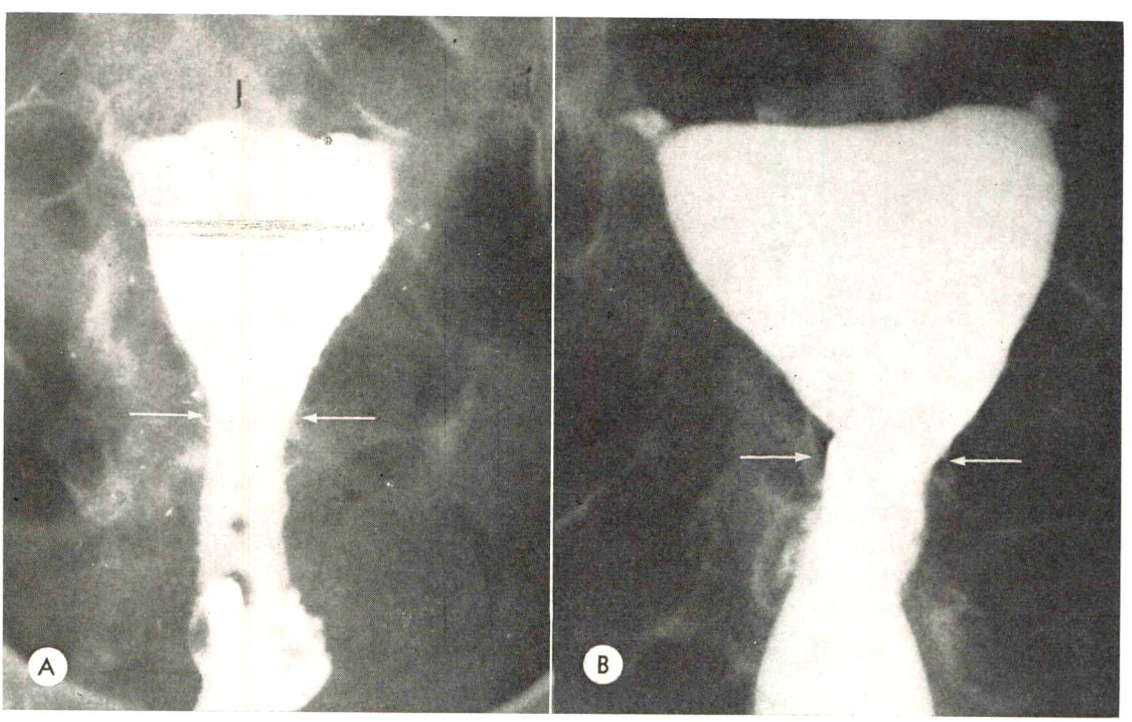


Fig. 3. Incompetence of the internal os due to multiple previous dilatations and curettages. (*A*) A 27 year old patient with miscarriage in the second trimester. The internal os measures 11 mm. (arrows). (*B*) A 26 year old patient with 3 previous dilatations and curettages developed an incompetent internal os and had two mid-trimester miscarriages. The internal os is 12 mm. wide (arrows). A measurement greater than 10 mm. is suggestive of incompetence (see text).

tion and curettage illustrated in Figures 2 and 3 were not part of the current abortion program, since the follow-up of patients from this program has been too short. It is hoped that the use of suction curettage, with or without supplementary scraping of the endometrium, will reduce the incidence of the Asherman syndrome and of the incompetent internal os.

2. SALINE INDUCTION OF ABORTION

New York State's abortion law was liberalized on July 1, 1970. At Morrisania City Hospital (Montefiore-Morrisania Affiliation), 3,569 elective abortions were performed between July 1, 1970 and June 30, 1972. Of these, 813 (23 per cent) were induced by intraamniotic injection of hypertonic saline (Table 1). The duration of pregnancy in the bulk of this group (81 per cent) was between 15 and 20 weeks. Abortion occurred from 12 hours to 4 days following the injection of saline, with the largest group delivering in 24 to 36 hours.

In the "salting out" procedure, about 200 ml. of amniotic fluid is usually removed and replaced with a similar amount of 20 per cent saline. The removal of amniotic fluid prior to injection of saline is not essential. In 110 women a new simplified technique, without removal of fluid (other than for testing the position of the needle), was safely accomplished by direct injection of 250 ml. of 20 per cent saline. Amniocentesis and the saline injection are performed with a 16 gauge Jelco catheter placement unit (Jelco Laboratories, Raritan, New Jersey). Following successful puncture of the amniotic sac, the inner metallic needle is withdrawn, leaving the flexible plastic outer catheter *in situ*. The injection of hypertonic saline is performed over a period of 5 to 10 minutes, during which multiple aspirations are made to ascertain a proper return of fluid.

Amniocentesis is usually a simple and easy procedure. However, in 4.5 per cent of cases there was difficulty in obtaining amniotic fluid. Obesity, uterine fibromyomata, a small uterus, overestimation of gestational age, oligohydramnios, various malpositions of the needle tip or a hydatidiform mole may account for the failure to obtain amniotic liquid.

The introduction of 20 per cent saline into the amniotic fluid is painless. In 3–4 per cent of cases the injection was inadvertently intramuscular (uterine wall), intraperitoneal or intravascular, due to dislodgement of the needle or catheter tip from an initially proper position. Such dislodgement can be caused by contraction of the uterus following removal of amniotic fluid. Injection into the uterine musculature is associated with severe pain over the lower abdomen. Intraperitoneal spill of saline may give delayed hyperosmolality with severe thirst. The patient should be encouraged to drink much water to correct the hyperosmolality; rarely peritoneal dialysis may be required. An intravascular injection of the saline solution is accompanied by pelvic pain, a flushing sensation, tinnitus, mental confusion, hypotension,[10]

TABLE I

ABORTION PROGRAM AT MORRISANIA CITY HOSPITAL
(July 1, 1970—June 30, 1972)

Method Employed	Stage of Gestation	No. of Patients	Per Cent
Dilatation and suction curettage	Up to 12 weeks	2,691	75
Intraamniotic injection of saline	15 to 24 weeks	813	23
Hysterotomy	12 to 24 weeks	65	2
Total elective abortions		3,569	100

and apneic episodes and may lead to coma, severe brain damage,[4] and death,[4,16] if the injection is not stopped immediately.

Whenever difficulty is encountered in an amniocentesis, the injection of water soluble contrast medium (through the same Jelco needle) will enhance and confirm proper placement of the needle in the amniotic sac (Fig. 4, *A* and *B*; and 5). Roentgenologic monitoring is particularly helpful in the early stages of an abortion program when extensive experience is lacking. Malpositions of the needle tip in the peritoneal cavity are readily recognized (Fig. 6, *A–C*). Similarly, an inadvertent intraplacental or intravascular injection (Fig. 7, *A* and *B*) can be seen under fluoroscopic control and serious reactions are averted. Goodlin *et al.*[10] report inadvertent intravascular injections in 8 out of 309 patients (2.6 per cent). These authors took great care to alert their patients to the possible warning symptoms of an intravascular injection. Under those circumstances the injection was immediately discontinued and measures were instituted to prevent central nervous system reactions (oxygen and intravenous infusion of 5 per cent dextrose in water). With an intelligent and perfectly alert, non-sedated patient this practice may be satisfactory. In other patients, when there is difficulty in withdrawing amniotic fluid during the injection, or if the aspirate is bloody, roentgenologic control *via* injection of contrast material is recommended.

Failure to obtain amniotic fluid during an attempt at saline induction may be due to a *hydatidiform mole* (Fig. 8), in which case the amniotic cavity does not develop properly. Instead, the uterus is filled with a friable mass of hydropic chorionic villi. The injection of radiopaque contrast medium into the uterus (properly called transabdominal hysterography rather than amniography since there is no amniotic sac) results in a highly characteristic appearance of multiple small spheroid radio-

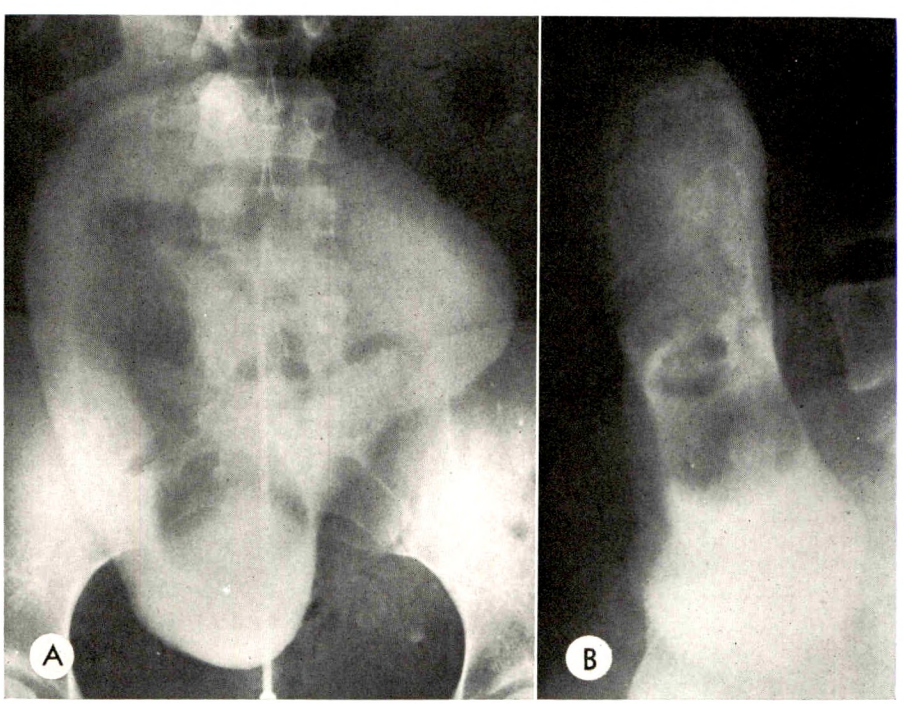


Fig. 4. Normal amniography at 17 weeks of gestation. The study ruled out hydatidiform mole and twin pregnancy, which had been suggested clinically because of an unduly large uterus. Fetal parts could not be clearly identified on the plain roentgenogram. (*A*) Frontal projection. Filling defect along the left lower uterine wall is due to the placenta. The fetus is well outlined. (*B*) Normal lateral projection.

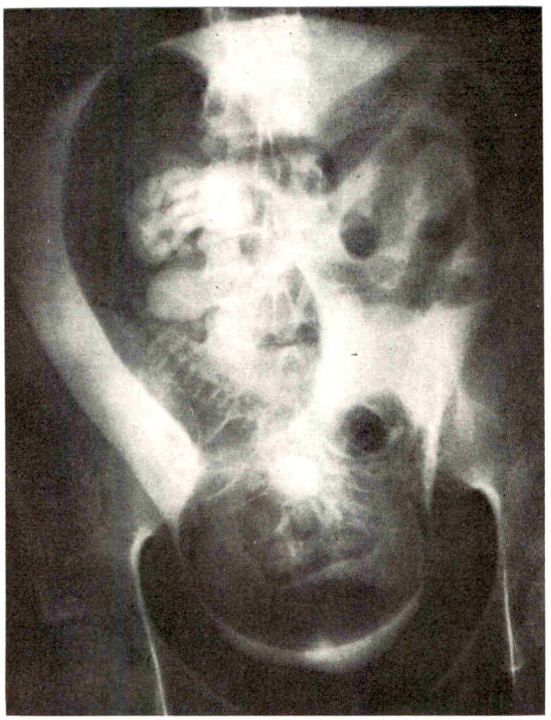


FIG. 5. Normal amniography at 21 weeks gestation. A 17 year old patient in whom amniocentesis was initially difficult (mild oligohydramnios). After withdrawal of amniotic fluid and injection of water soluble contrast medium, there is excellent visualization of the fetus. The fetal gastrointestinal tract is seen, due to rapid swallowing of opacified amniotic liquid by the fetus.

lucent images (Swiss cheese, sponge or honeycomb pattern) due to the cystic, grape-like villi surrounded by a reticulated network of contrast medium in the intervillous spaces. It is sufficient to recognize this typical pattern over a portion of the uterus to establish the diagnosis. About 30 ml. of 50 per cent sodium (or meglumine) diatrizoate is injected. The contrast medium disappears from the intervillous spaces rather rapidly and is excreted into the maternal urinary tract in a few minutes. Fluoroscopic control of the injection, with spot film roentgenographic documentation of the molar pattern at the right moment, is therefore advisable. In view of its high specificity, the roentgen diagnosis of a mole makes it possible to initiate a course of prophylactic chemotherapy (actinomycin D) for 3 days before evacuating the mole. This practice has decreased the incidence of both metastatic and local sequelae in molar pregnancy.[9] Transabdominal hysterography is a simpler method than pelvic arteriography for the diagnosis of hydatidiform mole. However, angiography helps to differentiate between benign and malignant trophoblastic disease[25] and identifies extrauterine extension of malignant disease. Ultrasonography has recently been used for the detection of molar pregnancy. A speckled multiple-echo ("snow flakes") pattern has been noted, believed to be due to the multiple interfaces produced by the grape-like villi.[13]

Inability to withdraw or inject any fluid at amniocentesis suggests that the needle tip is in a uterine fibromyoma. The salting-out procedure is occasionally difficult and may fail in twins, especially in the biamniotic variety; the diagnosis and management of such a situation (injection of both sacs) can be facilitated by amniography.

In this series of 813 patients undergoing saline induction for abortion, there were no fatalities. There were no instances of water intoxication or pulmonary embolization.[10] The most significant complication, occurring in 8 per cent of patients, was postabortal vaginal bleeding due to incomplete abortion, requiring (in 2 per cent of patients) 1 or 2 transfusions and curettage. The occasional onset of premature labor which has been noted after intraamniotic injection of contrast medium later in pregnancy is of no concern in an abortion program.

Rarely an ovarian cyst, mistaken for a gravid uterus, has been injected with saline, representing one of the causes for a "failed saline induction." Pelvic pneumography will help to clarify the nature of the pelvic mass (Fig. 9, *A* and *B*).

DISCUSSION

In addition to its role in an abortion program, amniography has been useful in several other areas of obstetrical diagnosis: (a) localization of the placenta, which ap-

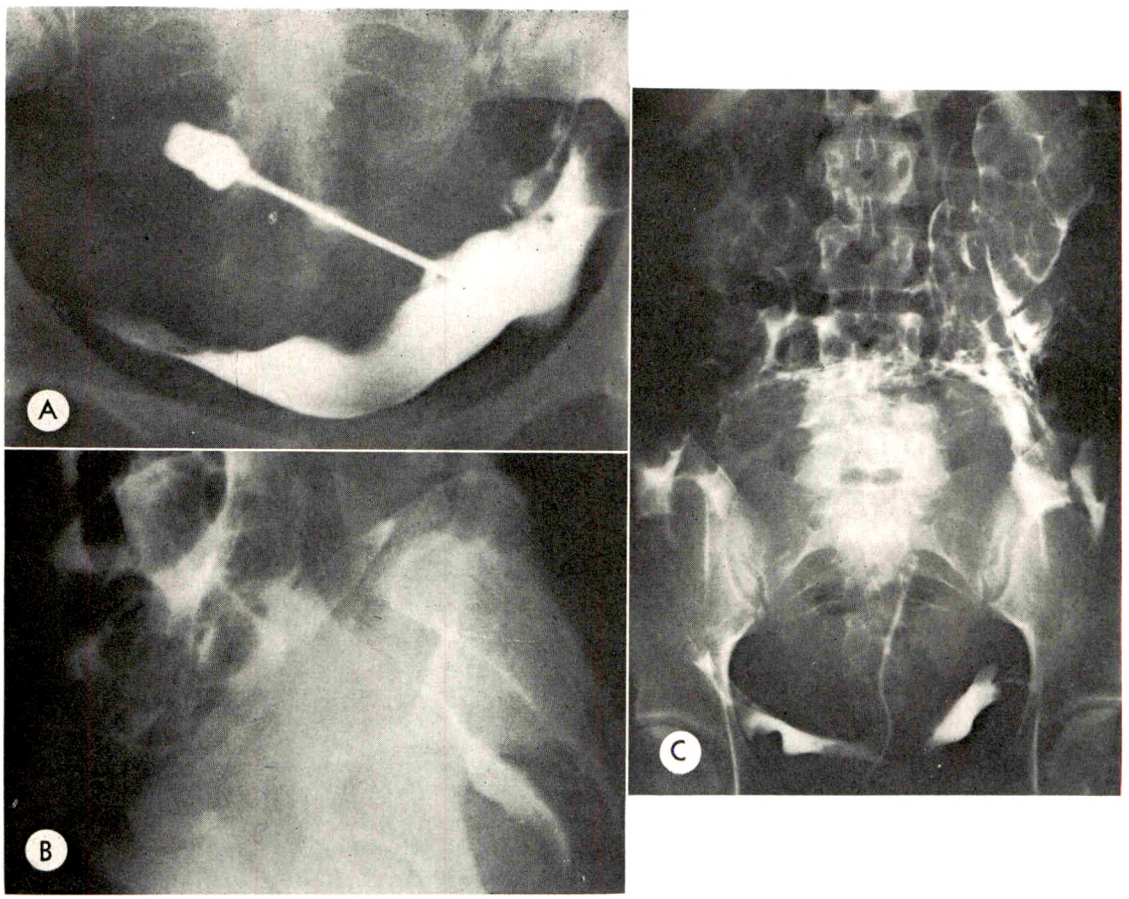


Fig. 6. Malposition of needle tip in the peritoneal cavity. (*A*) Injection of contrast medium during a difficult amniocentesis simulates entry of the needle into the bladder. (*B*) However, in a lateral projection the contrast medium is predominantly posterior, in the cul-de-sac. (*C*) Malposition of catheter in the peritoneal cavity in a different patient. The needle tip may become dislodged from an initially proper position due to contraction of the uterus.

pears as a filling defect in the contour of the opacified amniotic sac (Fig. 4 *A*); (b) determination of fetal life or death: the normal fetus swallows the opacified amniotic liquid, resulting in visualization of the fetal gastrointestinal tract (Fig. 5). The absence of swallowing is highly suggestive of fetal death. Major gastrointestinal anomalies (*e.g.*, duodenal atresia) may be detected by the lack of progression of contrast medium in the fetal digestive tract;[14] (c) detection of various gross fetal abnormalities, *e.g.*, fetal hydrops, meningocele, anencephaly, Pierre-Robin syndrome;[6] (d) guide to intrauterine transfusions into the fetal abdomen in cases of Rh incompatibility; (e) demonstration of polyhydramnios or oligohydramnios, which are associated with a high incidence of fetal anomalies; (f) recognition of ruptured membranes by leakage of contrast material;[14] and (g) estimation of fetal maturity.[6]

Certain authors have used oily contrast agents for amniography.[1,6,15] The oily substance impregnates the vernix caseosa and outlines the skin surface of the fetus. This can be helpful in measuring scalp edema—a presumptive roentgen sign of fetal death[1]—and in the determination of fetal sex.[1,6] A combination of oily and water soluble contrast medium has been used to facilitate the puncture of the fetal peritoneal cavity for purposes of transfusion.[1] We have used water soluble contrast medium exclusively

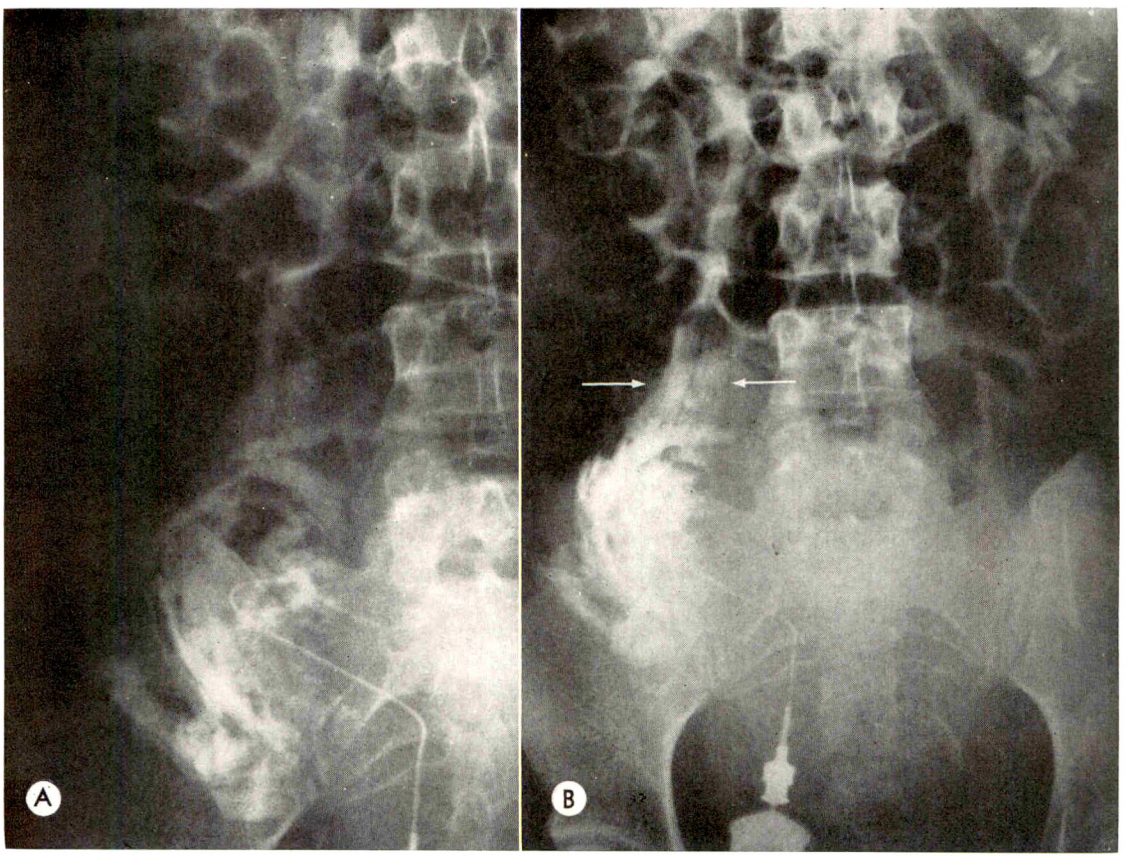


FIG. 7. Intravascular position of catheter. Following successful amniocentesis and during the injection of saline, aspiration did not yield a proper return of amniotic fluid. (*A*) Injection of contrast medium shows the catheter to be in a venous network, most likely within the placenta. (*B*) Slightly delayed roentgenogram showing drainage by paired ovarian veins (arrows) and excretion of contrast medium *via* maternal urinary tract. Prompt recognition of an intravascular injection of hypertonic saline is important to avoid acute hypernatremia and its serious consequences.

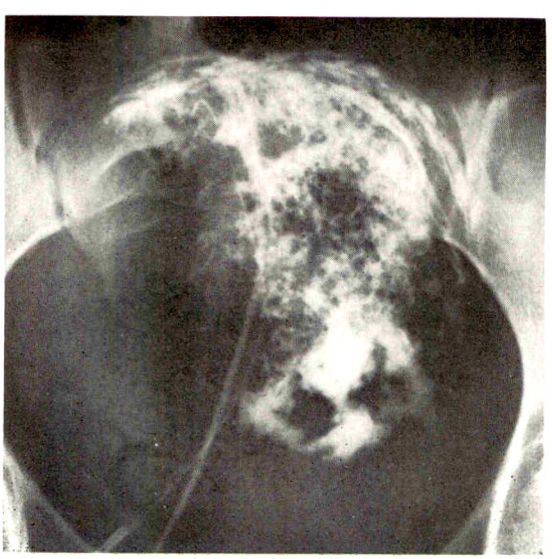


FIG. 8. Hydatidiform mole. Amniocentesis failed to yield fluid in this 23 year old patient who requested an abortion. The size of the uterus suggested a 16 week gestation. Injection of contrast medium shows the characteristic honeycomb appearance of a mole, with multiple spheroid lucencies due to hydropic villi. On the basis of this roentgen diagnosis, chemotherapy was instituted prior to evacuation of the mole.

because of the potential hazard of intravascular injection. Oily contrast medium is resorbed from the amniotic sac much more slowly than water soluble material.

Hysterotomy was the method of terminating pregnancy in 2 per cent of pa-

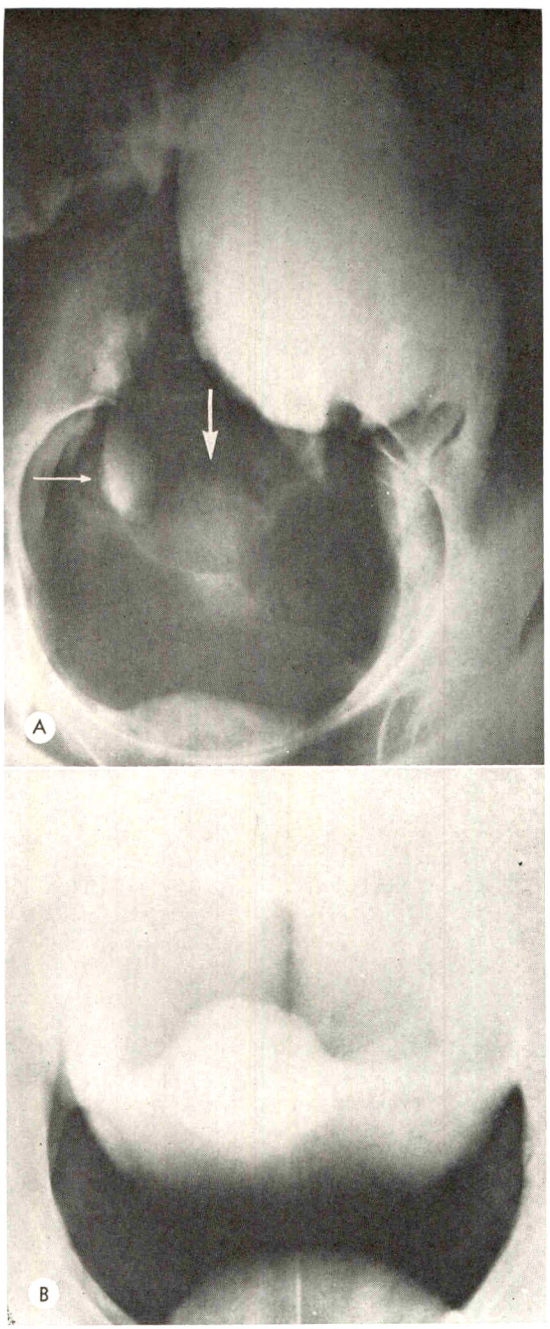


Fig. 9. Inadvertent injection of hypertonic saline into a left paraovarian cyst. A 19 year old female with amenorrhea for 2 months and a pelvic mass corresponding in size to a 14 week gestation, requested an abortion. Withdrawal of fluid from the mass and injection of hypertonic saline did not result in abortion. The patient was referred to Morrisania Hospital, where (*A*) pelvic pneumography showed a normal uterus (large arrow), normal right ovary (small arrow) and a large mass separate from the uterus. The latter proved to be a left paraovarian cyst. (*B*) Different projection during pelvic pneumography, showing normal uterus overlapping large ovarian mass. Dome-shaped density at bottom of figure is urinary bladder.

tients in this abortion program (Table 1). This method is used when saline induction of labor has failed or is undesirable (cardiorenal disease or previous cesarean section). Hysterotomy is also performed between the 12th and 16th week of gestation in patients who desire a tubal ligation in addition to an abortion.

The roentgenologic assessment of fetal age on a plain abdominal roentgenogram can help, in cases of vague menstrual history or inconsistent uterine size, to determine whether the gestation is within the legal time limits (24 weeks for New York), during which an abortion may be performed. Although an accurate evaluation of fetal age below 32 weeks is generally impossible, a reasonable estimation can be made from the crown-rump distance and from the longitudinal diameter of the skull. Thus, at 24 weeks, the sitting height, measured from the vertex to the proximal end of the femur, is about 21 cm., while the fronto-occipital diameter of the skull measures about 8 cm.[23]

A chest roentgenogram is performed routinely prior to admission for abortion. The yield of positive chest findings in this program has been very low.

A different radiologic aspect of abortion is the problem of the woman who has been exposed to ionizing radiation early in her pregnancy. Such radiation will not directly induce an abortion, but the embryo (particularly its neuroblastic elements) is highly radiosensitive.[20] The peak vulnerability to radiation in the production of congenital malformations appears to be from 20 to 45 days of gestation.[20] At what point is a therapeutic abortion indicated? Experience with human material is quite limited and quantitative extrapolations from experimental data in animals are probably in-

valid. The following preliminary suggestions have been made[12] regarding irradiation during the first trimester of pregnancy: for fetal doses below 1 rad, abortion is not indicated; doses between 1 and 10 rads require an abortion only in the presence of additional indications; for fetal doses above 10 rads a therapeutic abortion is presumably always indicated. The National Bureau of Radiological Health is currently gathering all the available information in this regard.[21]

Despite occasional complications, liberalized legal abortion, from a medical viewpoint, has proven to be a highly successful procedure, accompanied by a distinct drop in maternal mortality[7] and morbidity. In view of the probable extension of abortion on demand to other states and nations in the near future, an increasing number of radiologists is likely to encounter the problems described in this report.

SUMMARY

The radiologic aspects of abortion are divided into 2 main categories: (1) sequelae of uterine dilatation and curettage; and (2) problems related to intraamniotic injection of saline.

The first category includes uterine synechiae (Asherman syndrome) and incompetence of the internal os, the roentgen features of which are described and illustrated.

In the second category, proper placement of the needle in the amniotic sac can be verified by the injection of water soluble contrast medium (amniography). Malpositions in the placenta, vascular system or peritoneal cavity are easily recognized and serious consequences can be averted. The diagnosis of hydatidiform mole is readily established.

The use of amniography in other areas of obstetrical diagnosis, the roentgenologic assessment of fetal age and the problem of therapeutic abortion because of exposure to ionizing radiation early in pregnancy are briefly discussed.

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IMPROVEMENT OF THE CUPS USED WITH THE MALMSTRÖM THORËN VACUUM UTERINE CANNULA IN UTEROSALPINGOGRAPHY

By TOM M. FULLENLOVE, M.D.*
SAN FRANCISCO, CALIFORNIA

THE use of the vacuum uterine cannula in uterosalpingography has made the procedure much easier on the patient and consequently easier for the doctor. During the number of years of using the apparatus, the author had difficulty with the plastic cups that are used for the suction. They are made in Germany of a plastic called Makrolon which can be sterilized up to a temperature of 125° C. The difficulty was that the cups cracked around the hole in the base and gradually spread until a vacuum could no longer be maintained. Occasionally, using great care, several examinations could be done before the crack appeared and sometimes several after the crazing or cracking began, but eventually they were useless. All causes were contemplated and investigated. The obvious reasons were, of course, known—mishandling and side pressure during placement, rough handling during removal and cleaning. But even with one person designated for the cleaning, fissures would eventually appear and gradually increase. The rubber grommets, the bolts and washers were all varied in position and number to no avail.

The plastic was tested by the manufacturer for resistance to over 275 substances —from Acetaldehyde through Worcester sauce to Zephiran!—and while the plastic was not resistant to cresol or phenol it was to ethyl alcohol and 2 per cent Zephiran. So, not trusting autoclaving of the plastic, we sterilized the cups in an aqueous solution of Zephiran 1 part to 750 with perfect safety. We still continued to have fissuring.



FIG. 1. Comparison of the plastic and aluminum cups for the vacuum uterine cannula.

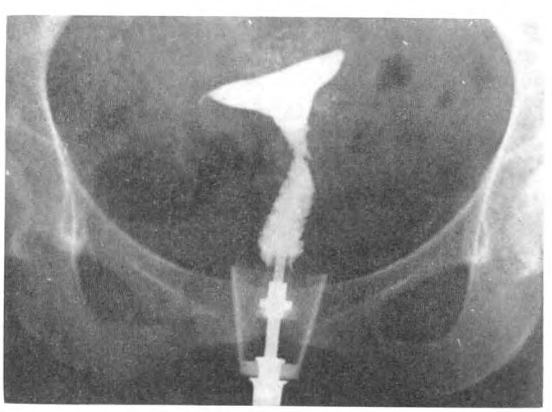


FIG. 2. Roentgenogram of the vacuum uterine cannula in use showing the radiolucency of the aluminum cup.

* Clinical Professor of Radiology, University of California, School of Medicine, San Francisco, California.

NEW DEVICE

To correct this situation the author had a metal worker make aluminum cups of exactly the same shape and size by turning them out of blocks of aluminum on a lathe (Fig. 1). These cups have worked perfectly for over 2 years, even though they block the view in placing the acorn in the cervix: with a little practice, this difficulty was easily overcome. They are translucent to roentgen rays and do not obscure the cervical canal (Fig. 2).*

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San Francisco, California 94108

The author acknowledges the technical assistance of Joe Campton, machinist and of Ruth E. Cames, R.T.

*AB Vacuum Extractor, Gothenburg, Sweden: Personal correspondence.

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Fifty-sixth Annual Meeting: Surfer Hotel, Maui, Hawaii, April 21–25, 1974.

EDITORIALS

SEVENTY-FOURTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Seventy-fourth Annual Meeting of The American Roentgen Ray Society was held for the third time in its history in Montréal, metropolis of Canada. To quote the chronicler "Montréal is a city on an island and is built around a mountain." The mountain is Mont Royal, the royal mount, of which Mont-réal is no more than a variant form, reminiscent of southern France. It has been called the city's crown and glory and it is.

In this superbly panoramic city the Annual Convention was accommodated in the ultramodern "La Reine Elizabeth" Hotel and the Scientific Sessions were held Tuesday through Friday, September 25–September 28, 1973.

President J. Scott Dunbar of Vancouver, British Columbia, Canada, called the meeting to order at 8:30 A.M. Tuesday. Immediately thereafter outgoing President Dunbar assisted by Dr. Ralph M. Scott, Louisville, Kentucky, Chairman of the Executive Council, installed Dr. William B. Seaman, of New York City, as the new President of the Society. In so doing, outgoing President Dunbar presented to Dr. Seaman the working Erskine gavel made from memorabilia of Röntgen, Coolidge and Snook and the symbolic Pfahler gavel, fashioned from a mastodon's tooth recovered from Alaskan glacial ice. Concluding the ceremony, Dr. Dunbar pinned on Dr. William B. Seaman the traditional gold emblem of the Society engraved "President of the American Roentgen Ray Society."

President Seaman as his first function, remitted to Dr. J. Scott Dunbar the Past Presidents' Book, inscribed with his name and containing a list of all Past Presidents, as well as color photographs of the historic Erskine and Pfahler gavels. He then gave his scholarly Inaugural Address on "At This Point in Time," which as the title indicates is a very timely subject and is published *in extenso* as the leading article in this issue of the JOURNAL.

In his Inaugural Address, Dr. Seaman quoted the famous words of Charles Dickens written in the opening paragraph of the "Tale of Two Cities" to describe the year of 1775: "It was the best of times, it was the worst of times; it was the age of wisdom, it was the age of foolishness; it was the epoch of belief, it was the epoch of incredulity; it was the season of light, it was the season of darkness; it was the spring of hope, it was the winter of despair; we had everything before us, we had nothing before us."

These words—said President Seaman—have a certain relevance at this *Point in Time*, when we will soon celebrate the 75th Anniversary of the foundation of the American Roentgen Ray Society, the oldest radiologic society in North America.

In appraising the many complex problems confronting Radiology today he quoted appropriate words of wisdom from The Presidential Address of Dr. Harold Reineke in 1960 "Whither Bound?" and The Presidential Address of Dr. James C. Cook in 1969 "In Union There Is Strength."

In conclusion President Seaman cited the closing sentence of Lewis Mumford's book "The Transformation of Man" which is: *Every goal man reaches provides a new starting point, and the sum of man's days is just a beginning.* "What new goals should this Society, soon to be 75 years old, be striving for?"

Prior to the beginning of the Scientific

Sessions, Byron G. Brogdon, Chairman, Committee on Continuing Evaluation of Postgraduate Education of ACR, announced that for the first time the Scientific Program of the American Roentgen Ray Society is being co-sponsored by the American College of Radiology for the purpose of obtaining credit for the AMA Physician Recognition Award in Category 1 or for other official use. The procedural instructions, the schedule of credit claimed for Category 1, as well as the requirements for 5 other categories of credit (6 altogether) were given and copies of application forms were made available.

The First Scientific Session began at 9:00 A.M. Tuesday with President William B. Seaman in the Chair. Paper 2 was the *Award Paper of the Society for Pediatric Radiology*, which was selected the preceding day by the Society for Pediatric Radiology as being of greatest interest to all radiologists. Its author was David L. Gilday, M.D., Toronto, Canada, and its title was "The Various Radionuclide Patterns of Cerebral Inflammation in Infants and Children." Dr. Gilday stated that the diagnosis of different cerebral inflammatory lesions is difficult due to their varied clinical presentation; therefore, a simple test such as a brain scan would be valuable. To reliably demonstrate cerebral inflammation, it is essential to do a routine delayed 2 hour brain scanning on all patients. The following patterns were encountered: (1) diffuse increase in radionuclide activity throughout the brain was associated with widespread encephalitis; (2) focal increase in radionuclide activity was found in patients with focal cerebritis (5 patients) or cerebral abscess (7 patients); (3) increase in activity outlining the ventricle was associated with ventriculitis (5 patients); (4) peripheral increase in radionuclide activity (often asymmetrical) with no abnormality seen on the radionuclide angiogram was associated with meningitis (8 patients); (5) focal superficial increase in activity was usually associated with osteomyelitis of the skull or subgaleal abscess (3 patients). These were best delineated by a bone scan following the brain scan. This Award Paper was followed by 12 additional papers, of which 2 dealt with pediatric subjects, 5 with problems of the gastrointestinal tract and appendages, 4 with urographic studies, and 1 with quantitative and qualitative analysis of the radiologic report.

The Second Scientific Session on Wednesday morning was divided into 2 sections: Section on Diagnostic Radiology; and Section on Therapeutic Radiology. The program of both sections started at 8:30 A.M. and ended at 11:30 A.M.

At the Section on Diagnostic Radiology which was chaired by Dr. Walter M. Whitehouse, Ann Arbor, Michigan, Second Vice-President, 11 papers were presented. The subjects of these papers were: 7 papers on intrathoracic lesions; 1 paper on mammography; 1 paper on computerized system; and 2 papers on ultrasonography.

At the Section on Therapeutic Radiology, chaired by Dr. Ralph M. Scott, Louisville, Kentucky, Chairman of the Executive Council, 10 papers were read on the following topics: treatment of orbital metastases; treatment of nonfunctioning chromophobe adenomas of the pituitary; results of treatment of carcinoma of the soft palate and mouth; squamous cell carcinoma of the nasal vestibule; medulloblastoma treated by Au^{198}; testosterone potentiated P^{32} therapy of osseous metastases in prostatic cancer; study of radiation tolerance of the lungs in dogs; protection of normal structures from high doses of irradiation; thermal enhancement of radiosensitivity of cultured human tumor cells; and electron beam therapy of head and neck tumors. The eleventh and final paper on this program was the *Award Paper of the American Radium Society* which was read by title. Its title was "Treatment of Glottic Carcinoma by Megavoltage Radiation Therapy and Results" by Dr. C. C. Wang, Boston, Massachusetts.

At 11:30 A.M. the festivities of the Caldwell Lecture, the highlight of the Scientific

Program of the Annual Meeting of the American Roentgen Ray Society, took place. This year's Caldwell Lecturer was Robert G. Fraser, M.D., Professor and Chairman, Department of Diagnostic Radiology, McGill University, and Diagnostic Radiologist in Chief, Royal Victoria Hospital, Montreal, Canada. Doctor Fraser was introduced by J. Scott Dunbar, M.D., Professor and Chairman, Department of Diagnostic Radiology, University of British Columbia, Vancouver, B.C., Canada.

Doctor Fraser, who is one of the foremost radiologic authorities on lung diseases in the world, delivered with great eloquence a brilliant Lecture on "The Radiologist and Obstructive Airways Disease," superbly illustrated and magnificently documented. The listening audience, which had overflowed the large Le Grand Salon, responded with enthusiastic recognition.

At the conclusion of the Lecture, President Seaman awarded to Dr. Fraser the Caldwell Gold Medal, the highest recognition of the Society "for meritorious scientific achievement."

The Third Scientific Session began Thursday morning at 8:30 A.M. with Dr. J. Scott Dunbar, Vancouver, British Columbia, Canada presiding. At this Session 16 papers were delivered. These papers were on such varied topics as: 5 papers on renal subjects; 5 papers on coronary artery and heart lesions; 2 on nuclear medicine; and 4 on miscellaneous topics.

On Thursday afternoon, the American College of Radiology, pursuing the tradition of the past, this year presented from 1:30 to 2:45 P.M., the following "Public Interest Programs" under the Chairmanship of Robert D. Moseley, Jr., M.D., President of the American College of Radiology as moderator: (1) You and the Feds . . . John W. Travis, M.D.; (2) About Cancer: ACR Activities . . . Gerald D. Dodd, M.D.; and (3) Standards in Practice: Various . . . Fredric D. Lake, M.D. The programs of the ACR are and must be rooted in the interests of the patient public. The panelists discussed the College activities that proved credibility to this truth. The moderator briefly noted a few individual membership benefits offered by the College.

On Thursday evening at 6:30 P.M. a Cocktail Party preceded the Annual Banquet which was held at 7:30 P.M. in the Le Grand Salon. At this Banquet, which always is the highlight of the Social Activities of the Annual Meeting, President Seaman introduced the newly elected officers and their consorts. This brief ceremony was followed by exquisite music provided by the world famous Talarico Orchestra of 14 musicians, and a pleasant evening of dancing.

On Friday morning, the Fourth and last Scientific Session, which again started at 8:30 A.M., was presided over by Dr. Robert G. Fraser, Montreal, P.Q., Canada, First Vice-President of the Society. At this session 16 papers were presented: 3 on renal lesions; 2 on nuclear medicine; 4 on middle ear, brain, and spinal cord lesions; 4 on studies of the gastrointestinal tract and appendages; and the remaining 3 on such varied subjects as arthrography, clipping injuries of football players and hysterosalpingography in infertility.

The Section of Instruction had again Dr. Harold O. Peterson, with his long experience, as Director and Dr. Richard G. Lester, as Associate Director. They assembled an outstanding Faculty of distinguished Instructors, who conducted the courses on the latest developments in radiology in a rather informal manner. There were 58 courses: 9 courses on Monday, 13 each on Tuesday, Wednesday and Thursday, and 10 on Friday. The arrangement for the courses was excellent and they attracted large audiences.

The Scientific Exhibits this year were again the important feature of the Annual Meeting. A surprisingly large number complemented the Scientific Essays on the same subject. The Society is greatly indebted to Dr. James F. Martin and his able Committee for their untiring effort in

assembling these excellent Exhibits which, together with the splendid Technical Exhibits, created unusual interest. A detailed description of these Scientific Exhibits was given in the November issue of the JOURNAL.

The Technical Exhibits, which were arranged under the expert direction of Dr. James C. Cook, Manager, and Dr. George A. Kling, Associate Manager of the Annual Meeting, were most instructive and superbly displayed. The November issue of the JOURNAL also gave an account of these exhibits, with a detailed list of the exhibiting firms.

The Chairman of the Committee on Local Arrangements was Dr. Jean-Louis Léger, with charming Mme Léger acting as Chairman of the Ladies' Committee. The outstanding social activities prepared for this year's Annual Meeting were as follows:

Monday: Guided walking tour in the narrow winding streets of "Le Vieux Montréal" with its historic atmosphere, followed by tea in "Les Filles du Roi."

Tuesday: Tour in the Laurentian Mountains with their breath-taking beauty and the maple leaves in full colors; luncheon at the Alpine Inn; and visit of one handicraft studio and Art Gallery in Sainte Adèle.

Wednesday: Visit to Radio-Canada (C.B.C. radio and T.V.) new tower building; and luncheon at Restaurant Hélène de Champlain. The City of Montréal offered a cocktail reception before the luncheon.

Thursday: Visit to the Botanical Garden, founded by Brother Marie-Victorin, B.C.S., and now scientifically affiliated with the Université de Montréal, close to the facilities of the Summer Olympic games of 1976, now under construction.

To Dr. and Mme Jean-Louis Léger, and the members of their able Committees, the Society extends its profound thanks for arranging this unique and most enjoyable program.

The Annual Golf Tournament was held on Monday, September 24, 1973, at the Royal Montréal Golf Club on Bizard Island. The tournament, which was open to both members and guests, was arranged by Doctors R. D. McKenna and Jean H. Gagnon. There was a "shot-gun start" at 9:30 A.M. with play ending for all simultaneously about 1:30 P.M. Lunch was served at 2:30 P.M., preceded by a reception, and followed by the awarding of prizes, and the show of movies of the previous tournaments.

The Annual Tennis Tournament for members and guests was also held on Monday, September 24, 1973 at Les Quatre Saisons (The Four Seasons) Tennis Club in Laval, a suburb of Montréal. The tournament was arranged by Doctors Ross Hill and Maurice Dufresne. Play began about 9:00 A.M. and ended around 1:00 P.M. This was followed by refreshments, luncheon and the awarding of prizes.

The Committee on Press Relations, with Dr. Jacques Sylvestre, M.D. as Chairman, ably assisted by Mr. Charles Honaker, Director of Public Relations of the American College of Radiology, conducted the press and public relation activities, with finesse and good judgment.

The Society expresses its sincerest thanks to the Montréal Convention Bureau and to the Management and Staff of the La Reine Elizabeth Hotel, for the efficient registration, the excellent service, and the many other courtesies gracefully rendered throughout the meeting.

The Seventy-fourth Annual Meeting in beautiful Montréal, "a city on an island built around a mountain," was a grand success in every respect. President Seaman deserves the sincere congratulations and unbounded admiration of the Society.

TRAIAN LEUCUTIA, M.D.

In Memoriam*

H. DABNEY KERR, M.D.
1893–1973

H. DABNEY KERR, a renowned radiologist and former Trustee of the American Board of Radiology, died unexpectedly at the age of 80 on July 3, 1973, following a heart attack. For the past 18 years he had made his home at Hawkeye Point, St. Michaels, Maryland. Although considered semi-retired he was extremely active in the affairs of the American Board of Radiology as its secretary from April 6, 1957 to December 31, 1968, as well as in the practice of Therapeutic Radiology.

Dr. Kerr was born in Catonsville, Maryland, and attended the Tomes School in Port Deposit, Maryland, and Davidson College. He graduated from the University of Wisconsin in 1916 and from Johns Hopkins University School of Medicine in 1919. After marrying the former Eleanore Smith in 1920, Dabney and his bride went to China to serve as Medical Missionaries. They were there under the Presbyterian Board until 1927. It is understood that a "war lord" intervened and took over the area. Mrs. Kerr and their daughter came back to the States, but Dabney went to Peking to wait out events. While there, at Peking Union Medical College, he became interested in the work of Paul Hodges and, in consequence, went on to the University of Michigan in 1928 where he worked under Preston M. Hickey at Ann Arbor. He continued his education in Radiology at various centers in the United States and in Europe. In 1930, Dr. Kerr became Professor of Radiology at the University of Iowa; in 1933, he was named Chairman of the Department and held that post until his retirement in 1955.

After retiring from his post at the University of Iowa, he returned to Maryland, and established his residence on the Eastern Shores, his favorite part of the United States. He continued productively as a therapeutic radiologist up to the time of his death, and was still participating as a Consultant in Radiation Therapy at Arundel General Hospital and Easton Memorial Hospital. He is survived by his faithful and adorable wife and two daughters who must be justly proud and appreciative of his many accomplishments and honors.

Dr. Kerr became a member of the Board of Trustees of the American Board of Radiology in 1945 and served for 24 years. He was President of the Board from 1954 to 1956 and in 1957 was selected as the Board's Secretary. He remained in that strenuous post for almost 12 years, guiding the development of the Board in a variety of aspects. As Secretary he always sought the advice of the Executive Committee, maintaining the highest level of integrity in every decision that was made by the Executive Committee and then carried forward through himself.

In his earlier years, as an Examiner, Dabney was known to be rather rigorous, yet always fair and particularly compassionate. As Secretary he chose to keep the office of the American Board of Radiology in Rochester, Minnesota, and, even from his home in Maryland, ably directed the affairs of the Organization. He made numerous trips, up to six or more per year, from Baltimore to Rochester and kept a strong guiding hand on the activities of the Board during these critical years. Throughout this period, with all its problems, Dr. Kerr maintained his usual calm and charm and never complained about the enigmas which so often confronted him.

It would be impossible to list all of the

* This "In Memoriam" tribute to Dr. H. Dabney Kerr, was presented to The Board of Trustees of The American Board of Radiology, December 2, 1973.

many honors which came to Dabney Kerr. In brief it is noted that he delivered the Janeway Lecture of the American Radium Society in 1951, received the Bronze Medal of the Iowa Division of the American Cancer Society in 1954, the Gold Medal of the Radiological Society of North America in 1964, and the Gold Medal of the American College of Radiology in 1971. Dabney was a member of Phi Beta Kappa, Sigma Xi and Alpha Omega Alpha. He published 45 articles in various journals and co-authored a book with Carl L. Gillies on "The Urinary Tract." In addition, contributions were made to Portmann's "Clinical Therapeutic Radiology" and to Pack and Ariel's "Treatment of Cancer and Allied Diseases."

Dr. Kerr did much of his early work in interstitial implantation of radon seeds and Au^{198} in the treatment of carcinoma of the prostate with Dr. Rubin H. Flocks, Professor of Surgery at the University of Iowa. Their mutual interest in prostatic cancer was abetted by their joint activities. As a matter of fact, in 1951, Dr. Flocks performed a total prostatectomy on Dr. Kerr, who remained essentially without disease until his death 22 years later.

According to Dr. Flocks, Dabney was a superb tennis player and was one of the outstanding players on the faculty in his younger days. Socially, he was a most delightful person, yet he never smoked nor took a drop of alcohol. The current writer, knowing of this, years ago presented Dabney, at one of the Board examinations, with a bottle of "Northern Comfort." When it was handed to him, sort of surreptitiously, he said "Oh no, I never have anything to do with that." (For those who are not familiar with "Northern Comfort," it is provided in what looks like a pint whiskey bottle, but contains maple syrup.) I finally convinced him that he should try it. Thus began an interchange of New Hampshire maple syrup over the years thereafter and reciprocation with wheels of Maytag cheese which, I can say, were out of this world. Actually, less than two weeks after his death, a wheel of his Maytag cheese was served to the residents and staff members at our Department picnic, and it seemed they enjoyed every moment of sampling.

Carl Gillies, one of Dabney's long time associates at the University of Iowa, pointed out that he was very alert to Chinese fellows or residents and would often speak to them in Chinese. I can also attest that I have seen him do this many times at the examination of the American Board of Radiology.

Very few radiologists have attained the stature of Dabney Kerr, and the American Board of Radiology has profited by the high level of his service for so many years. He will long be remembered for the extreme measure of integrity with which he conducted his life. Perhaps Al Good's final statement in his presentation for Dabney to the American College of Radiology best describes the man—"The Gold Medal . . . will honor a great leader and educator, a servant of the specialty of Radiology, and above all, a kind gentleman, a gracious friend, and a man of good will."

Laurence L. Robbins, M.D.

Massachusetts General Hospital
Boston, Massachusetts 02114

NEWS ITEMS

DIAGNOSTIC RADIOLOGY OF THE URINARY TRACT

(with a Session on Ultrasound)

This Aspen Seminar sponsored by the Department of Radiology, Louisiana State University School of Medicine in Shreveport, will be held at the Aspen Institute for Humanistic studies, Aspen, Colorado, February 23–27, 1974.

The outstanding Guest Faculty will include: Luther W. Brady, M.D., Philadelphia, Pennsylvania; C. Eugene Carlton, Jr., M.D., Houston, Texas; Peter Clapp, M.D., Boston, Massachusetts; Erich K. Lang, M.D., Shreveport, Louisiana; James W. Lecky, M.D., Pittsburgh, Pennsylvania; and Thomas F. Meaney, M.D., Cleveland, Ohio; in addition to the staff of the Department of Radiology and Urology of the LSU School of Medicine in Shreveport, Shreveport, Louisiana.

The Seminar is designed for radiologists and urologists alike. This year's presentations will emphasize radiologic diagnosis and evaluation of adrenal lesions, neoplastic, inflammatory, and traumatic conditions of the kidneys, bladder, urethra, and prostate. A special session on the use of ultrasound in the assessment of urologic conditions is included. The program will also offer panel discussions on renovascular hypertension, treatment of traumatic injuries to the urinary tract, and treatment of carcinoma of the prostate and bladder. These panel discussions intend to analyze and summarize other presented material, and opportunity will be offered to all registrants for free exchange of opinion with the panelists during these sessions.

For further information, please write to: E. K. Lang, M.D., Department of Radiology, LSU Medical Center, School of Medicine in Shreveport, P.O. Box 3932, Shreveport, Louisiana 71130.

ASSOCIATION OF RADIOLOGISTS OF WEST AFRICA

The Association of Radiologists of West Africa will hold the Twelfth Annual Conference on February 22–24, 1974 in Monrovia, Liberia.

For further information, please contact: C. A. Beetlestone, M.B., B.S., D.M.R.D., Honorary Secretary, c/o Department of Radiology, University College Hospital, Ibadan, Nigeria.

POSTGRADUATE COURSE IN PEDIATRIC RADIOLOGY

The Department of Radiology of Cornell University Medical College will hold a postgraduate course in Pediatric Radiology at the Barbizon Plaza Hotel, New York, New York, April 18–20, 1974.

The Guest Faculty includes: Drs. Donald Altman, David Baker, Walter Berdon, John Caffey, Herman Grossman, Herbert J. Kaufmann, M. B. Ozonoff, and Edward B. Singleton, in addition of the local Faculty of The Cornell University Medical College and The New York Hospital.

For further information, please write to: John A. Evans, M.D., Department of Radiology, Cornell University Medical College, 1300 York Avenue, New York, New York 10021.

CURRENT STATUS OF PEDIATRIC OSSEOUS RADIOLOGY

The Institute for Pediatric Radiology and Chas. E. Shopfner, M.D. will present another in the Current Status of Pediatric Radiology series of programs titled "Current Status of Pediatric Osseous Radiology," at the Marriott Hotel, Canal and Chartres Streets, New Orleans, Louisiana, February 16–17, 1974.

There is unlimited registration.

For additional information, please write: Institute for Pediatric Radiology, 4148 North Cleveland Avenue, Kansas City, Missouri 64117.

THE FIRST EUROPEAN COURSE IN CHEST RADIOLOGY

The Fleischner Society announces that it will be holding this course at The Royal College of Surgeons, London, W.C.1., May 20–23, 1974.

The speakers will be members of the Fleischner Society, a group of eminent American and European radiologists, physicians, physiologists and pathologists interested in chest diseases.

The registration is limited.

For further information, please write to: The London Secretariat of the Fleischner Society: *Hon. Secretary*, Dr. Ian H. Kerr, 156 Lambeth Road, London A.E.1.

TWELFTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The interface between heart and lung disease will be the theme of the XII International Congress on Diseases of the Chest, which will be held in London, England, July 7–12, 1974.

A select number of rigidly screened manuscripts, heretofore unpublished and original, will be presented.

The Congress, to be held at Royal Festival Hall, is sponsored by the International Academy of Chest Physicians and Surgeons, affiliated with the American College of Chest Physicians.

Two thousand physicians from 56 countries are expected to attend the Congress, which will feature medical symposia on: Airway Obstruction; Lung Defense and Lung Clearance; Coronary Heart Disease—Medical *vs.* Surgical Treatment; Lung Cancer; Congenital Heart Disease—at All Ages; Current Problems of Rheumatic Mitral Valve Disease; and The Reaction of the Respiratory Tract to Irritants.

Plans for opening day of the Congress include a nurses symposium and a series of 15 clinic visits to hospitals and teaching centers in London. In addition to the symposia there will be tutorial sessions, luncheon panels, scientific seminars and medical motion pictures.

For further information, please contact: Bradford W. Claxton, American College of Chest Physicians, 112 East Chestnut Street, Chicago, Illinois 60611.

FIFTH INTERNATIONAL CONGRESS OF RADIATION RESEARCH

The Congress will be held in Seattle, Washington, U.S.A., July 14–20, 1974.

The program of plenary sessions, symposia, and contributed papers encompasses original research with ionizing and nonionizing radiation in physics, chemistry, medicine, and biology, including biomedical and other applications, and environmental hazards of radiation.

The deadline for the receipt of registration forms, abstracts, housing reservations, etc., is February 1, 1974.

Reduced rate transportation to Seattle is being arranged from various points in the world. Partial travel support for some participants may be applied for. Further information is contained in the final brochure.

Anyone interested in attending the meeting who has not received the final brochure together with registration and other forms should write, at once, to: Dr. W. K. Sinclair, Secretary-General, Argonne National Laboratory, Argonne, Illinois 60439 U.S.A.

IAEA SYMPOSIUM ON DYNAMIC STUDIES WITH RADIOISOTOPES IN CLINICAL MEDICINE AND RESEARCH

This symposium, sponsored by the International Atomic Energy Agency (IAEA), will be held in Knoxville, Tennessee, U.S.A., July 15–19, 1974.

The Symposium, which is a sequel to the symposium on the same subject held by the International Atomic Energy Agency in Rotterdam in 1970, will be concerned with all those applications of radionuclides in clinical medicine and research that involve investigation of the temporal patterns of uptake, metabolism, clearance or excretion of administered radioactive materials.

Emphasis will be placed on the methods used and their potentialities. Topics covered will include techniques (radioactivity measurement, data analysis, etc.), applications in studies of the function of organs and systems of the body (specific organs, tumors, the circulatory system, etc.) and applications in studies of the metabolism of substances in the body (minerals, proteins, radiopharmaceuticals etc.). Studies based on scintigraphic techniques will be excluded except insofar as they are concerned with dynamic situations.

Further information and forms to accompany abstracts of papers intended for presentation at the Symposium may be obtained from national authorities for atomic energy matters. Abstracts must be submitted through these authorities so as to reach the International Atomic Energy Agency by February 8, 1974.

For further information, please write to: R. A. Dudley and E. H. Belcher, Scientific Secretaries, IAEA Symposium on Dynamic Studies with Radioisotopes in Clinical Medicine and Research, Kärntner Ring 11–13, P.O. Box 590, A-1011, Vienna, Austria.

BOOK REVIEWS

THE ROENTGENOGRAPHIC DIAGNOSIS OF RENAL MASS LESIONS. By Erich K. Lang, M.D., Professor and Chairman, Department of Radiology, Louisiana State University Medical Center, School of Medicine in Shreveport, Shreveport, La. Cloth. Pp. 182, with many illustrations. Price, $11.50. Warren H. Green, Inc., 10 South Brentwood Boulevard, St. Louis, Mo. 63105, 1971.

This monograph covers the technique modalities of retroperitoneal gas insufflation, aortography, selective arteriography of the kidney and adjacent arterial vessels (lumbar, adrenal, subcostal, phrenic, gonadal, hepatic and splenic) with and without vasoconstrictor enhancement and, lastly, special emphasis on percutaneous puncture, aspiration, and double contrast studies of renal cysts. Other longer chapters, besides the introduction, deal with renal tumors, renal cystic disease and the co-existence of renal cysts and carcinoma.

All of the illustrations come from the author's own material as do most of the tables (although a few of the latter are adapted from other contributors) and this is both a strength and limitation. It should be read critically and with an open mind by the resident or those who have limited daily exposure to the interpretation of roentgenologic studies of renal cysts and neoplasm, since the experienced worker will ignore such statements as " . . . invasion of the pyelocalyceal system and amputation of calyces may occasionally occur with cysts." Table IX is confusing to this reviewer, and there are variations between the text and the data presented in other tables. The bibliography is quite comprehensive (through 1969); however, there are many bothersome editing errors to these references in the text.

The author's attempt at staging of renal cell carcinoma on the basis of arteriographic and venographic criteria is commendable, but subject to considerable inaccuracy in addition to its limited clinical usefulness.

This monograph reflects an appropriate thrust of diagnostic radiology and reflects the author's long interest in the delineation of renal masses. It represents an effort to bring together scattered material in the literature and the author's own experience under a single cover. Although there is considerable repetition of some material throughout the text, the style of the book makes for easy reading, and the index is easy to use. It represents a comprehensive review and is a valuable reference work in its area.

RICHARD C. PFISTER, M.D.

TESTICULAR TUMORS. Edited by Douglas E. Johnson, M.D., Associate Professor (Urology) and Chief, Section of Urology, The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Texas. Cloth. Pp. 300, with 145 illustrations. Price, $20.00. Medical Examination Publishing Company, Inc., 65-36 Fresh Meadow Lane, Flushing, N. Y. 11365, 1972.

This review by 12 contributing authors from the University of Texas M. D. Anderson Hospital and Tumor Institute of Houston representing each of the specialties that deal with testicular neoplasms (pathologist, urologist, diagnostic radiologist, chemotherapist and radiation therapist), suffers from the uneven nature of works generated by several authors.

The chapter on pathology could have presented more discussion of the British Classification, but the presentation was provided for a clinically oriented overview, to paraphrase the author, and the problems of histogenesis were intentionally avoided.

The second chapter on etiology is well written, but extremely brief for such a complex and contradictory subject.

The third chapter is a complete and well written review of the epidemiologic data of the M. D. Anderson series of 469 testicular tumors.

The chapters on clinical diagnosis, laboratory and radiologic investigation are a concise discussion easily recommended to house staff, students and the general medical community.

The chapter on lymphangiography is a lengthy (one-third of the text pages), but needed review by the foremost student in the field. Particularly useful is Doctors Wallace and Jing's discussion of diagnostic criteria and accuracy.

Doctor Johnson's chapter on surgical management presents a clear, brief review of surgical techniques and, together with the subse-

quent chapter on radiation therapy, sets forth the sensibly integrated approach to malignant disease usual at M. D. Anderson. Results are presented in considerable detail and are not surprising.

The chapter on chemotherapy is a good review of drugs utilized in testicular malignancies with enthusiasm, occasionally bordering on the overly optimistic—yet thought provoking.

The book is completed by a chapter on testicular tumors in children and 3 chapters on non-germinal, lymphomatous and metastatic tumors.

The reference list is extensive, with nearly 550 references. Unfortunate is the technical quality of the book. The variable type quality could be forgiven in the interests of economy, the photographic quality, especially of key roentgenograms, cannot.

John D. Earle, M.D.

BOOKS RECEIVED

Analysis of Roentgen Signs in General Radiology. In Three Volumes. By Isadore Meschan, M.A., M.D., Professor and Director of the Department of Radiology, Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, N. C.; Consultant, Walter Reed Army Hospital; Member, Committee on Radiology, National Research Council, National Academy of Sciences; with the assistance of R. M. F. Farrer-Meschan, M.B., B.S. (Melbourne, Australia), M.D., Clinical Assistant Professor, Department of Obstetrics and Gynecology and Associate in Radiology, Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, N. C. Cloth. *Volume 1:* Introduction/Skeletal System (Including Joints, Skull and Spine). Pp. 697, with 402 illustrations. Price, $25.00. *Volume 2:* Respiratory System/Heart (Excluding Special Contrast Studies). Pp. 516, with 277 illustrations. Price, $20.00. *Volume 3:* Abdomen/Urogenital Tracts/Alimentary Tract. Pp. 702, with 412 illustrations. Price, $25.00. Price of set of three volumes, $70.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

Tumours of Childhood: a Clinical Treatise. By I. G. Williams, M.B., B.S.(Lond.), F.R.C.S.(Eng.), F.F.R., D.M.R.E., Director, Radiotherapy Department, St. Bartholomew's Hospital, London; Consultant Radiotherapist, The Hospital for Sick Children, Great Ormond Street, London, England. Cloth. Pp. 169, with some illustrations. Price, $15.00. Appleton-Century-Crofts, 440 Park Avenue South, New York, N. Y. 10016, 1973.

Cystourethrography: a Radiographic Atlas. By M. Hertz, Radiologist, Department of Diagnostic Radiology, The Tel-Aviv University Medical School, Shaim Sheba Medical Center, Tel-Hashomer, Israel. Cloth. Pp. 280, with many illustrations. Price, $41.00. American Elsevier Publishing Company, Inc., 52 Vanderbilt Avenue, New York, N. Y. 10017, 1973.

Mercer's Orthopaedic Surgery. Seventh edition. By Robert B. Duthie, M.A.(Oxon.), Ch.M.(Edin.), F.R.C.S.(Edin. and Eng.), F.A.C.S., Nuffield Professor of Orthopaedic Surgery, Oxford University; Honorary Surgeon to the Nuffield Orthopaedic Centre and to the Accident Department, Radcliffe Infirmary, Oxford; Consultant Adviser in Orthopaedic and Accident Surgery to the Department of Health and Social Security in England and Wales; Fellow of the British Orthopaedic Association; Member of the Société International d'Orthopédie et de Traumatologie; Member of the British Orthopaedic Research Society; Member of the British Society for Surgery of the Hand; Corresponding Member of the American Orthopaedic Association; Fellow of the Royal Society of Medicine; Late Professor and Chairman of Orthopaedic Surgery, University of Rochester School of Medicine and Dentistry and Orthopaedic Surgeon in Chief, Strong Memorial Hospital, Rochester, N. Y.; and Albert B. Ferguson, Jr., B.A., M.D., F.A.C.S., Silver Professor of Orthopaedic Surgery, and Chairman, Department of Orthopaedic Surgery, Chief of Orthopaedic Surgery, University of Pittsburgh Medical Center Hospitals; Chief Orthopaedic Consultant, Veterans Hospital, Oakland; Consultant to Mercy, St. Francis and St. Margaret's Hospitals; Member American Board of Orthopaedic Surgery, American Orthopaedic Association, American Academy of Orthopaedic Surgery and American College of Surgeons. Cloth. Pp. 1,236, with some figures. Price, $77.50. Williams & Wilkins Company, Baltimore, Md. 21202, 1973.

Conference on Particle Accelerators in Radiation Therapy, October 2–5, 1972. Held at the Los Alamos Scientific Laboratory of the University of California, Los Alamos, New Mexico under the auspices of The American College of Radiology and The Interagency Panel on Medical Physics. Paper. Pp. 158. Price, $3.00. National Technical Information Service, U. S. Department of Commerce, 5285 Port Royal Road, Springfield, Va. 22151, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Surfer Hotel, Maui, Hawaii, April 21–25, 1974.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 25–30, 1973.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Roosevelt Hotel, New Orleans, La., April 1–6, 1974.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Chicago, Ill., June 15–19, 1974.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel; New York, N. Y., June 10–14, 1974, New York Hilton Hotel; and Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers in June 1974.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1974 was September 30, 1973.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

The third examination is scheduled for Saturday, Sept. 21, 1974. Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017. The deadline for receipt of completed applications is June 1, 1974.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D. C. 20010. Annual Meeting: Jointly with RSNA, Palmer House, Chicago, Ill., Nov. 25–30, 1973.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting: Royal Sonesta, New Orleans, La., Oct. 24–28, 1973.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting: Ann Arbor, Mich., Oct. 14–18, 1973.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907. Meeting: Feb. 16–21, 1975.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Meeting in June, 1975 in Edinburgh, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Richard H. Greenspan, Dept. of Radiol., Yale University School of Medicine, 333 Cedar St., New Haven, Conn. 06510. Twenty-second Annual Meeting, New York, N. Y., May 8–11, 1974. Dr. Norman E. Chase is in charge of arrangements, New York Medical Center, New York, N. Y. 10016.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Charles J. Masters, Maj. MC, Department of the Air Force, United States Air Force Hospital, Wiesbaden (USAFE) APO New York 09220.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

***Secretaries of societies are requested to send timely information promptly to the Editor.**

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Giacomo Pantaleo, 69-13 Forest Ave., Brooklyn, N. Y. 11227. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Ru-Kan Lin, 191 North St., Buffalo, N. Y. 14201. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jasumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month Holiday Inn, Morgantown, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer, Dr. William H. Roush, Radiology Service, Veterans Administration Hosp., Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Lee Radford, 6011 Harry Hines, Suite 900, Dallas, Tex. 75235. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Menzie H. Brown, 155 S. 18th St., Kansas City, Kans. 66102.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Richard E. Litt, 6125 S. W. 31st St., Miami, Fla. 33155. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Shirley D. Vickers, Catholic Medical Center of Brooklyn and Queens, 88-25 153rd St., Jamaica, N. Y. 11432. Annual Meeting: Houston, Texas, June 16–20, 1974.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John R. Dehner, Reid Memorial Hosp., Richmond, Ind. 47374.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Eli A. Etscovitz, 10 Stonewood Dr., Newton Centre, Mass. 02159.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. A. G. Fleurquin, Pontiac General Hosp., Pontiac, Mich. 48053. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Howard L. Copas, 827 South 131st St., Omaha, Neb. 68154. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

New England Roentgen Ray Society
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting: May 10, 1974.

New England Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 25–27, 1974. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 S. University Dr., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 2100 Rockwood Dr.,

Sacramento, Calif. 95825. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 22–24, 1974.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

San Diego Radiological Society
President, Dr. Leland E. Kellerhouse, 2466 First Ave., San Diego, Calif. 92101. Meets the first Monday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Chairman, Dr. Henry W. Wiggins, Jr., Mercy Hosp., 2537 South Prairie Ave., Chicago, Ill. 60616. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M. September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. 21st Annual Meeting: Town and Country Hotel, San Diego, Calif., June 11–14, 1974.

South Bay Radiological Society
Secretary, Dr. Virgil H. Voss, 900 Kiely Blvd., Santa Clara, Calif. 95051. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane I. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 311 Medical Arts Bldg., Chattanooga, Tenn. 37402. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neil, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205. Annual Meeting: La Mansion Hotel, San Antonio, Tex., March 21–23, 1974.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P.Q., Canada.
First European Course in Chest Radiology at the Royal College of Surgeons, London, W.C.1., May 20–23, 1974.
Hon. Secretary, Dr. Ian H. Kerr, 156 Lambeth Rd., London, S.E.1.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Don H. Nichols, 5770 S. 300 East, Murray, Utah 84107. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 8952 Bellfonte Rd., Richmond, Va. 23229.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Isaiah J. Seligman, 16 Guion Pl., New Rochelle, N. Y. 10801. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Francisco Miranbell S., Apartado 3352, San José, Costa Rica. Thirteenth Central American Congress of Radiology, San José, Costa Rica, Dec. 4–8, 1973.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England. Annual General Meeting: May 15–18, 1974.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, 1555 Summerhill Ave., Montreal 109, Que., Canada. Thirty-seventh Annual Meeting, Regency Hyatt House, Vancouver, B. C., April 21–26, 1974.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, Lincolns Inn Fields, London WC2A 3PN, England. Annual General Meeting: May 15–18, 1974.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE. 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

Sociedad Argentina de Radiología
Secretary-General, Dr. Tomás J. Gutiérrez E, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 p.m.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

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ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

HEAD

FISH, MATHEWS B., BARNES, BARBARA, and POLLYCOVE, MYRON. Cranial scintiphotographic blood flow defects in arteriographically proven cerebral vascular disease. *J. Nuclear Med.*, Aug., 1973, *14*, 558–564. (Address: Dr. Mathews B. Fish, Clinical Laboratories Building 100, Room 163, San Francisco, Calif. 94110.)

Static radionuclide imaging has been used to study cerebral vascular disease (CVD) and the initial applications were concerned with the focal alterations of the blood brain barrier (BBB) due to cerebral infarction. The majority of patients with CVD are without acute infarction, but may have significant changes in cerebral circulation which are not manifested by conventional static scintigraphy.

Rapid sequential cranial scintigraphy after intravenous bolus injection of Tc^{99m} has permitted imaging of cranial vascular filling and regional perfusion.

The authors studied the accuracy and sensitivity of this dynamic approach by correlating the results with the clinical status and arteriographic determination of sites and degrees of stenosis and occlusion in 84 patients with angiographically proven CVD. The patients were divided into 3 groups: (1) asymptomatic and transient ischemic attacks; (2) acute and subacute strokes; and (3) chronic completed strokes. Abnormality of the scintiphotographic study was noted in 53 per cent of Group 1, 83 per cent of Group 2, and 75 per cent of Group 3 patients. The abnormalities were divided into extracranial vascular disease and intracranial vascular disease. Of those with isotopic abnormalities, all but 1 patient showed blood flow abnormality. Most patients in Group 1 were without BBB defects and only one-half of Group 2 and one-third of Group 3 patients had BBB defects.

Correlation of blood flow studies with specific arteriographic findings revealed isotopic abnormalities in 68 per cent of the patients with extracranial vascular disease and in 85 per cent of patients with intracranial disease. BBB defects occurred in 25 per cent of patients with extracranial involvement and in 44 per cent of patients with intracranial involvement.

The over-all diagnostic accuracy of 75 per cent includes over one-half of the patients who were not in the acute or subacute phase. The diagnostic accuracy was reduced from 75 to 32 per cent, if only the conventional BBB studies were used.—*Charles W. Cooley, M.D.*

NECK AND CHEST

BRIGGS, V. A., REILLY, B. J., and LOEWIG, K. The chest roentgenogram in babies of 1,000 grams or less. *J. Canad. A. Radiologists*, June, 1973, *24*, 119–125. (From: Department of Radiology, The Hospital for Sick Children, Toronto, Ontario, Canada.)

The authors examined the roentgenograms of 75 children who weighed under 1,000 grams in the premature age group in an effort to see if there were any differing patterns from those of more mature infants and if these patterns changed following treatment. Most of the chest roentgenograms were made within ½–6 hours after birth in the supine position with a portable unit at 40 inches. Autopsy findings were obtained on 32 of 54 of the infants who died, and were correlated with the roentgenographic appearance of the chest.

Twelve of the babies had normal chest roentgenograms. There were 4 main pathologic roentgenographic patterns: (1) 17 children had a cystic bubbly pattern and oxygen had been administered for a longer time period to the babies showing this pattern. Ten of these children died and autopsies revealed patchy focal atelectasis and some hyaline membrane formation with marked overdistention of the bronchioles; (2) chest roentgenograms that showed total opacity, all patients died and many had high concentration oxygen. All of the patients who were autopsied had respiratory distress syndrome with marked hyaline membrane involvement, and pulmonary hemorrhage and atelectasis were present in about half of all of these patients; (3) the basal opaque group comprised 17 patients of whom 3 survived, and all of the autopsied patients in this group had atelectasis, but no hyaline membranes were found; and (4) the fourth group comprised 6 patients, who had consolidation and 2 of these patients survived. The consolidation was patchy and was not confined to the bases. Pathologic findings involved infection only and 1 patient showed evidence suggesting oxygen toxicity.

The authors, in their discussion, point out that pathologic findings with the bubbly cystic pattern probably represented alveolar collapse with adjacent alveolar overdistention; and they further postulate that the overdistended alveoli may have had the effect of narrowing small bronchioles. They did not find evidence of oxygen toxicity which could be implicated in the delayed clinical and roentgenologic findings of the Wilson-Mikity syndrome, but no biopsies were carried out on those who survived with this pattern, and so they cannot confirm or disprove whether oxygen toxicity plays a part in the production of cystic bubbly lung. Those infants, who had the respiratory distress syndrome, had severe disease with the opaque pattern described on the chest roentgenogram and it was in this group that one could not distinguish pulmonary hemorrhage from severe hyaline membrane involvement. There was a high incidence of intracranial hemorrhage in the autopsied patients.—*Robert P. Andrews, M.D.*

LOPEZ-MAJANO, V. Diagnosis of pulmonary embolism. *Respiration*, 1973, *30*, 201–222. (Ad-

dress: Professor V. Lopez-Majano, Nuclear Medicine Service, Section of the Veterans Administration Hospital, Hines, Ill. 60141.)

Pulmonary embolism occurs frequently and can be successfully treated if diagnosed, and if not diagnosed and treated may have a mortality rate of 51 per cent. Ten per cent of the cases of pulmonary embolism develop infarction.

Pulmonary embolism may occur: (1) as a single embolus; (2) as multiple emboli; (3) massively in the acute, subacute and chronic stages; and (4) with infarction.

Pulmonary embolism can be diagnosed from the history, clinical findings, and by using a combination of the chest roentgenograms, scintigraphy or imaging, and pulmonary arteriography.

Clinical symptoms and signs vary and are suggestive but not entirely diagnostic. Sudden chest pain, cough, dyspnea, tachycardia and hemoptysis may be present.

The chest roentgenograms are not always diagnostic and may vary from a normal appearing chest to one with an enlarged heart with decompensation. Decreased vascularity on the involved side (Westermark's sign) with an increased vascularity on the opposite side may occur.

Lung imaging or scintigraphy with a gamma camera using technetium 99m albumin microspheres or I^{131} albumin macroaggregates as the agents for perfusion is very helpful in diagnosing pulmonary embolism. The radioactive particles cannot enter the embolized area producing an area of decreased activity. Xenon 133 can be used for both perfusion and ventilation studies. Lung imaging can be used to follow the course of pulmonary embolism and evaluate the treatment.

Pulmonary arteriography is an accurate method to diagnose pulmonary embolism as the emboli can be demonstrated; *i.e.*, if they are not microemboli.

A normal chest roentgenogram, normal ventilation, and an abnormal lung scintigram or arteriogram are diagnostic of pulmonary embolism.—*Charles W. Cooley, M.D.*

Freundlich, Irwin M., and Israel, Harold L. Pulmonary aspergillosis. *Clin. Radiol.*, April, 1973, *24*, 248–253. (From: Department of Radiology, Arizona Medical Center, Tucson, Ariz. 85724.)

The ubiquitous fungus *Aspergillus* is not pathogenic in healthy individuals except under rare circumstances, but is a common opportunistic invader of chronically diseased pulmonary tissues. Invasive aspergillosis and allergic aspergillosis are both relatively uncommon. The proposed classification is:

A. Noninvasive Pulmonary Aspergillosis
   1. Aspergilloma
   2. Allergic Bronchopulmonary Aspergillosis

B. Invasive Pulmonary Aspergillosis
   1. Associated with Altered Host Defenses Secondary to
      a. Malignant disease
      b. Severely debilitating nonmalignant disease
      c. Immunosuppressive therapy
   2. Overwhelming Inhalation of Aspergillus Spores.

The purpose of this communication is to call attention to superimposed aspergillosis as a cause of recurrent hemoptysis in patients with chronic cystic and cavitary lung disease. Twenty-six patients were included in this report, 18 of whom, approximately 70 per cent, presented with hemoptysis, 2 of whom died of exsanguination. The aspergillomata were secondary to sarcoidosis in 12 of these patients, but this is not a true reflection of the most common type of primary pulmonary disease, as it comes from a large clinic devoted to sarcoidosis. By far, the next most common underlying primary pulmonary disease process was tuberculosis. In 2 of the patients the secondary infection presented as fluid-filled cysts rather than as solid fungus balls.

Roentgenographically, a diagnosis can be established by the demonstration of a fungus ball, which, when it fills the entire cavity, will require tomography to demonstrate the crescent of air between the mycetoma and the wall of the cavity.

Sputum cultures and the serologic tests for the antibodies of *Aspergillus* can also establish the diagnosis. It should be noted that the antibodies for *Aspergillus* were detected only at the time of the active disease when the patients were culturally positive for the fungus or shortly after recovery. This is contrary to the findings in other fungi, such as histoplasmosis, in which the antibodies may persist in the sera for several years after recovery.

The authors conclude that when the patients with chronic cystic or cavitary lung disease return with recurrent symptomatology, including hemoptysis, one should not assume that the signs and symptoms are all secondary to the primary disease process. Patients with chronic lung disease and recurrent symptoms merit routine tomography as well as sputum cultures and blood serology to rule out the presence of an opportunistic *Aspergillus* infection.—*Donald M. Monson, M.D.*

Lesobre, R., Hadengue, A., and Legrand, M. Les manifestations pleurales de l'asbestose. (Pleural manifestations of asbestosis.) *Presse méd.*, June, 1973, *2*, 1491–1494. (Address: R. Lesobre, Centre de Pneumologie, Hôpital Beaujon, 100 boulevard du Général Leclerc, F. 92110 Clichy, France.)

Pulmonary asbestosis represents a well known entity. A predominantly pleural involvement, however, has been considered unusual in the past, although it actually occurs more frequently than the pulmonary

component. It develops mostly in the parietal pleura of the lower chest and the bases in the form of hyaline plaques. As it progresses in an upward direction, it tends to encase the lower lobes.

Characteristic calcifications following the exposure to the asbestos dust may also coat the domes of the diaphragm and the lower rib cage. These changes can develop even many years after exposure to the specific dust has been eliminated. A rare exudative pleurisy has also been encountered. This condition is apt to recur and eventually lead to a malignant mesothelioma.

The benign pleurisy, as observed in a personal case, is characterized by an insidious onset without causing pain or presenting generalized signs. The exudate usually is serofibrinous, but also can be hemorrhagic containing lymphocytes and much albumen. On thoracotomy, only pleural plaques are encountered.

The malignant mesothelioma also presents with an insidious onset, but there are progressive asthenia, anorexia, and slight fever. The exudate usually contains tumor cells, asbestos fibers, and hemosiderin. A history of specific exposure has been established in 6 of 12 such personal cases encountered over a period of 8 years.

Roentgenograms show rapidly growing tumor masses mostly in the lateral chest wall and bases with lobulation and scalloping. The lymph nodes are rarely involved, but there is metastatic seeding to the mediastinum, pericardium, contralateral pleura, and the peritoneal cavity.

The prognosis is poor, with a survival rate of only 1 to 3 years.—*Ernest Kraft, M.D.*

Abdomen

Reddy, Bandaru S., and Wynder, Ernest L. Large-bowel carcinogenesis: fecal constituents of populations with diverse incidence rates of colon cancer. *Cancer*, June, 1973, *50*, 1437–1442. (From: Division of Nutrition and Division of Epidemiology, Naylor Dana Institute for Disease Prevention, The American Health Foundation, New York, N. Y. 10021.)

The authors have studied 4 population groups with different dietary habits for fecal constituents. The groups included 17 people on a "Western" diet, 12 American vegetarians, 11 American Seventh Day Adventists, 21 Japanese and 11 Chinese.

Multiple biochemical tests were carried out on the stool specimens including beta glucuronidase, neutral sterols and bile acids.

The results showed that the group on a "Western" diet had a significant increase in fecal bile acids and neutral sterols. Fecal cholesterol and bile acids were degraded more actively in the "Western" group. The authors suggest that the findings probably relate to the associated increase in beta glucuronidase activity found in patients on a Western-American diet. The level of beta glucuronidase activity seems to depend on the number of anaerobic bacteria in the stool and the bacterial microflora is dependent on diet. While beta glucuronidase activity cannot be directly related to carcinogenesis, the fact that people eating a "Western" type diet have an increased enzymatic activity for degradation of byproducts may help to explain the reported 20 per cent decrease in the incidence of colon cancer in Seventh Day Adventists.

The authors point out that more study is needed to actually determine the incidence of colon cancer in the various diet groups before a definite correlation between diet and colon cancer is established.—*Daniel P. Link, M.D.*

Hickey, M. C., Ghosh, S. K., and Hugh, A. E. Radiology of the lesser sac. *Clin. Radiol.*, April, 1973, *24*, 162–165. (From: North Staffordshire Hospital Group, Stoke-on-Trent, England.)

Embryologically, following development of the peritoneal space, the midline ligament suspending the developing alimentary tract becomes folded, as the potential stomach and duodenum rotate to the right to form a sac within a sac—the lesser sac which lies behind the stomach and communicates through the epiploic foramen with the remainder of the peritoneal cavity, the "greater sac." Rotation of the distal alimentary tract—the colon—complicates but does not compound the extent of the lesser sac, which lies in front of the rotated transverse colon.

Posteriorly, the sac is bounded by the left adrenal gland, the upper pole of the left kidney, and most of the anterior surface of the pancreas. Below these, but still posteriorly, are the third part of the duodenum and transverse colon, with the latter being suspended in the transverse mesocolon. In front of the sac are the stomach and the caudate lobe of the liver. In addition there are extensions of the sac superiorly into the left subphrenic area, and inferiorly into the greater omentum.

In cadaver experiments, the best technique found to roentgenographically visualize the extent of the lesser sac was to fill it with a positive contrast medium (barium) introduced through a Foley catheter which had been inserted through the epiploic foramen, inflated, and drawn back against the foramen to seal it. Both humans and dogs were investigated, and the relationships of various organs to the lesser sac were studied. The well-defined impressions of the left adrenal gland and the pancreas posteriorly were noted.

Consideration was given to clinical applications of this technique for these hard to visualize and evaluate areas.

The authors suggest that it might be possible to use a modified peritoneoscope to introduce a Foley type of balloon catheter through the epiploic fora-

men without a large abdominal incision, and then inject a water-soluble positive contrast medium. Further evaluation of this technique is recommended.

Reproductions of roentgenograms, and 4 diagrams are included for illustration.—*Donald M. Monson, M.D.*

Berkman, M., Seban, C., Favre, M., and Chiche, P. Nécrose hémorragique du tractus digestif et cardiopathie: à propos de cinq observations. (Hemorrhagic necrosis of the digestive tract and cardiopathy: 5 observations.) *Presse méd.*, May, 1973, *2*, 1417–1422. (Address: P. Chiche, Service de Cardiologie, Hôpital Tenon, 4 rue de la Chine, F 75020 Paris, France.)

Hemorrhagic necrosis of the digestive tract is caused by a lack of blood supply to the intestine. It differs from mesenteric infarction by an absence of vascular occlusion. The localized anemia originates from an underlying ischemic cardiopathy.

The disease occurs in the elderly with chronic heart failure, arrhythmia, and ectopic tachycardia. A sudden fall in cardiac output can bring about this failure. At this stage, myocardial and cerebral circulation remains intact, but an ischemia of the intestinal mucosa gives rise to the hemorrhagic necrosis.

Clinical manifestations comprise violent abdominal pain, melena, and a general deterioration. In addition to the acute cardiac failure, atrial tachycardia and ventricular extrasystoles may be encountered.

Five pertinent cases have been observed in the aged. The clinical condition proved so severe that only 1 patient survived. Histologically, typical lesions of hemorrhagic necrosis were found. The mesenteric blood vessels were patent and contained no blood. In 1 case only, the vasculature was filled with cholesterol crystals. The patency of the vessels in the other cases was illustrated by the angiogram of a necropsy specimen.

The necrosis can be either disseminated or segmental. There is absence of inflammatory reactions except for shallow ulcerations. The condition can be provoked and aggravated by medication with digitalis, adrenaline, and noradrenaline. An operation also proves disastrous and may result in irreversible shock and sudden death. On the other hand, medical management with isoproterenol and antibiotics can be helpful. When the hemorrhagic necrosis remains segmental instead of being widespread, a regeneration of the mucosa with eventual recovery remains a possibility.—*Ernest Kraft, M.D.*

Rösch, Josef, Lakin, Paul C., Antonovic, Ruza, and Dotter, Charles T. Transjugular approach to liver biopsy and transhepatic cholangiography. *New England J. Med.*, Aug., 1973, *289*, 227–231. (Address: Dr. Josef Rösch, Department of Diagnostic Radiology, University of Oregon Medical School, 3181 S.W. Sam Jackson Park Road, Portland, Oregon 97201.)

A transjugular approach to liver biopsy and cholangiography is described. Under local anesthesia and television control a catheter is placed in the hepatic vein. A long needle is then advanced through the catheter into the liver parenchyma. For biopsy a saline aspiration is done through the needle and for cholangiography the needle is placed in a bile duct and its position is checked by test injection under fluoroscopy. For enlarged duct systems 50–100 cc. of contrast material may be necessary for a proper opacification.

The authors show that utilizing this technique successful biopsies and cholangiographies can be obtained in a high percentage of patients (89 per cent for biopsies, and 93 per cent for cholangiography in patients with dilated duct systems).

In this technique the risk of bile peritonitis and hemoperitoneum is minimal, even in patients with coagulopathies. The potential risk of bile-venous fistula was not a problem in this study or in the quoted study by Hanafee.

The authors suggest that the main disadvantage to this technique is that it requires specialized equipment and personnel (a radiographic room with a fluoroscopic television monitor and an experienced angiographer). Attempts may be unsuccessful in patients with venous abnormalities or anomalies. This approach is contraindicated in patients with acute cholangitis because of the risk of sepsis. The transjugular technique is proposed as a useful complement to the transperitoneal approach especially in high risk patients.—*Daniel P. Link, M.D.*

Blood and Lymph System

Doremieux, D. J., Carrier-Clerambault, R., Heim, B., and Masson, J.-Cl. Quatorze cas de chylurie filarienne observés à l'Île de la Réunion. (Fourteen cases of filarial chyluria observed at the Île de la Réunion.) *J. d'urol. et de néphrol.*, March, 1973, *79*, 277–290. (Address: D. J. Doremieux, Pavillon chirurgical A., Service de Chirurgie Urologique, Hospices Civils, F-67 Strasbourg, France.)

This is a report on 14 cases of filarial chyluria observed during a 21 month stay at the Île de la Réunion.

The authors review several reports which appeared in the French literature since 1812 on endemic chyluria and hematuria on islands in the Indian Ocean and, more recently, French Polynesia.

In the Île de la Réunion chyluria occurs at all ages, twice as often in the male as in the female;

may involve any of the inhabitants with no distinction as to racial background.

The manifestations of the disease may be insidious, the course irregular and unpredictable.

In about 50 per cent of the cases, hematuria, fever, lumbar pain, and the passage of fibrinous clots may be the only manifestations. Chyluria may appear soon after or at a later date. When chyluria develops, the urine is white and whitish gelatinous clots may develop.

There is weight loss and progressive weakness. The patient seems to lose "all benefit from his nourishment."

Radiologic studies include routine excretion urography, retrograde cysto-urography and lymphangiography. The latter is more accurate in an attempt to determine the site and number of fistulas. These invariably communicate with the upper urinary tract to the exclusion of the lower urinary tract.

The renal and perirenal lymphatics are irregularly dilated, the thoracic duct is obstructed.

Reproductions of 3 roentgenograms and 7 tables accompany this article.—*William H. Shehadi, M.D.*

Serres, J. J., Lesquerre, Cl., Lamouche, P., Bouvry, M., and Pene, P. Étude lymphographique de la chylurie filarienne: revue de la littérature à propos de 8 cas. (Lymphographic study of filarial chyluria: revue of the literature in reporting 8 cases.) *J. de radiol., d'électrol. et de méd. nucléaire,* March, 1973, *54*, 223–229. (Address: Pr agr. J. J. Serres, CUSS, B. P. 1364, Yaoundé, Cameroun.)

At the *International Symposium on Lymphography* held in Zürich in 1966, it was estimated that only 30 lymphographic studies in chyluria had been performed in the entire world. By 1971, over 100 such examinations had been reported to which the authors add 8 of their own.

The advent of lymphography has given added dimension to the radiologic study of patients with chyluria, to a great extent superseding intravenous and retrograde urography.

By means of lymphography, the flow of contrast material into all components of the renal lymphatics is clearly visualized; likewise, fistulous communications between the lymphatic and the urinary tract.

The authors follow the technique of Kinmouth. Since the lymphatic channels are dilated, instead of increasing the volume of ethiodol (lipiodol ultrafluid), the progression of the contrast material is assured by the additional injection of normal saline. By injecting each side separately, at a few weeks interval, better visualization of the lymphatics occurs. For the most part, the rate of flow of contrast material remains within normal limits. The lumbar lymphatic channels are usually increased, are tortuous, present saccular dilatation and numerous anastomoses.

The lymph nodes present irregular variations in pattern. The thoracic duct was dilated in 4 of the 8 cases examined, elongated and tortuous in 2, and normal in 2.

A direct lympho-urinary fistulous communication was demonstrated in 4 out of the 8 cases examined.

Reproductions of 14 roentgenograms accompany this article.—*William H. Shehadi, M.D.*

Skeletal System

Columbaro, Guido, and Rusconi, Umberto. Le osteonecrosi asettiche della testa del femore. (Aseptic osteonecrosis of the head of the femur.) *Radiol. med.*, Nov., 1972, *58*, 886–922. (From: Istituto di Scienze Radiologiche dell'Università degli Studi di Milano e Reparto di Radiologia dell'Ospedale di Circolo di Merate, Como, Italy.)

This review is limited to the aseptic necrosis which affects the femoral head of patients in the pediatric age and adults, in whom the pathogenetic mechanism of the lesion is an altered vascularization.

The authors suggest the following classification which is based on the type of pathologic changes involving the vascular bed of the femoral head:

Type I aseptic necrosis includes the lesions which are secondary to a traumatic laceration of one or more nutritional arteries of the head of the femur; in Type II the ischemia is produced by alterations in the arterioles and in the intraosseous capillaries; under Type III are grouped those cases in which the aseptic necrosis is secondary to a venous "tamponage" at the level of the articulation; and in Type IV the ischemia is secondary to an obstruction in the lumen of the arteries and capillaries.

The roentgen findings have been divided into 3 fundamental groups which are characterized by the pathologic changes demonstrable in the femoral heads of: (1) children, such as seen in Perthes' disease; (2) adults, such as seen in caisson disease, in patients treated by cortisone or following severe trauma to the hip; and (3) adolescents. In this latter group the extent of the lesions is limited, and the evolution of the process can result either in a complete return to normalcy, or in sequestration. In this group is included osteochondritis dissecans of the adolescent.

The authors then review the normal and abnormal pattern of vascularization of the head of the femur, and reference is made to the well-known studies by Trueta.

Finally, analyzed and discussed in detail are the pathologic and roentgenologic findings of aseptic necrosis in accordance with the group and type of their classification.

Of interest is the authors' finding of an idiopathic necrosis of the femoral head in a few alcoholic pa-

tients in whom the disease appeared slowly and "silently" and with a roentgen appearance characterized by a uniform zone of osteoporosis of the head, limited by a thin layer of sclerosis. The authors believe that this finding should be further investigated because it could lead to a type of aseptic necrosis of the head of the femur secondary to an "alcoholic vasculopathy."

Numerous demonstrative illustrations accompany the article and the bibliography is accurate and up-to-date.—*A. F. Govoni, M.D.*

Miller, Ray, Kettelkamp, Donald B., Laubenthal, Keyron N., Karagiorgos, Athanase, and Smidt, Gary L. Quantitative correlations in degenerative arthritis of the knee. *J. Bone & Joint Surg.*, July, 1973, *55-A*, 956–962. (Address: Dr. Ray Miller, Department of Orthopedic Surgery, University of Iowa, Iowa City, Iowa 52240.)

The authors report the progression of roentgenographic changes, the relationships of clinical to roentgenographic findings, and the relationship between clinical and roentgenographic findings with quadriceps strength in a group of patients with degenerative arthritis of the knee.

Progression of roentgenographic changes over 5 to 11 years and the relationship of roentgenographic changes to symptoms, function and quadriceps strength were evaluated in 50 knees (35 patients). Patellofemoral arthritis progressed more than that of the weight-bearing compartments. Increased degeneration of the medial compartment occurred in less than 50 per cent of the varus knees. Severity of roentgenographic degeneration was not related to body weight.

Women had more severe patellofemoral arthritis and greater limitation of activity than men.

Increased patellofemoral arthritis was associated with increased pain, increased functional impairment, and decreased quadriceps strength.—*John H. Harris, Sr., M.D.*

Bowerman, Jack W., and Muhletaler, Carlos. Arthrography of rheumatoid synovial cysts of the knee and wrist. *J. Canad. A. Radiologists*, March, 1973, *24*, 24–32. (Address: Dr. J. W. Bowerman, Henry Dale Laboratories, Institute of Orthopaedics, Stanmore, Middlesex, HA7 4LP, England.)

The authors review the plain film roentgenographic and arthrographic findings in 6 patients with rheumatoid arthritis. Three presented with calf pain and were initially diagnosed as having thrombophlebitis. One patient, treated with heparinization, developed pelvic hematoma. Subsequently, each patient underwent knee arthrography and in each case a large popliteal and calf cyst was demonstrated. In 2 of these patients, review of the plain film roentgenograms revealed soft tissue swelling distal to the knee joint and medial to the tibia. In 2 additional patients with joint pains, but without specific symptoms referrable to the calf area, arthrography also demonstrated synovial cysts about the knee. In the sixth case there was soft tissue swelling about the wrist and distal forearm, particularly on the volar aspect of the wrist; wrist arthrography and direct injection of a volar cyst demonstrated synovial hypertrophy and a large bilobed flexor tendon sheath with synovial hypertrophy creating filling defects.

The technique for arthrography described is similar to that previously published. The authors emphasize the need for exercise and ace bandage wrapping to fill the cystic outpouchings. The arthrographic abnormalities in these patients are similar to those previously described in cases of rheumatoid arthritis and include popliteal, calf and wrist synovial cysts, villous hypertrophy of the synovium, cysts of palmar flexor tendon sheaths, rupture of calf or popliteal cysts and associated tears of the collateral ligaments of the minisci at the knee. The largest calf cyst described in the present series extended downward to the musculotendinous junction of the Achilles' tendon and contained several filling defects reflecting synovial hypertrophy. Plain film roentgenograms often showed minimal bone changes of rheumatoid arthritis.

Idiopathic noncommunicating popliteal cysts previously have been described in children. Posttraumatic cysts usually are associated with a meniscal tear. Cysts have also been reported in many diseases, which are associated with joint effusion including osteoarthritis, tuberculosis, brucellosis, Sjögren's syndrome, Reiter's syndrome, pigmented villonodular synovitis, Charcot joint, psoriatic arthritis, osteochondromatosis and rheumatoid arthritis. Widespread development of synovial cysts and hypertrophy in various portions of the body are well known in rheumatoid arthritis. At the knee cysts are seen superior to the suprapatellar bursa, anterior to the proximal tibia, and in the popliteal space and calf area.

Rupture of popliteal and calf cysts, as evidenced by "smudgy" appearance of the opaque medium, has been reported before. It may be associated with acute pain or may occur asymptomatically.—*John T. Underberg, M.D.*

Schlenker, James D., Clark, David Dick, and Weckesser, Elden C. Calcinosis circumscripta of the hand in scleroderma. *J. Bone & Joint Surg.*, July, 1973, *55-A*, 1051–1056. (Address: Dr. James D. Schlenker, 3147 East Derbyshire, Cleveland Heights, Ohio 44118.)

The authors' interest in this disease started with a

patient who appeared with localized calcification in a finger.

Ten additional cases of calcinosis circumscripta in the hand were found by reviewing the charts of 65 patients discharged from University Hospitals of Cleveland during the past 10 years with the diagnosis of scleroderma.

In a series of 65 patients with scleroderma, 11 had calcinosis circumscripta. Four other patients in the series had calcifications in the soft tissues elsewhere than in the hand. Nine of the 11 patients were women.

The diagnosis of calcinosis in the hands was made on the average of 17 years after the onset of Raynaud's phenomenon.

In only 2 of the patients was surgical excision of the calcifications performed.—*John H. Harris, Sr., M.D.*

GLYNN, JAMES J., and LICHTENSTEIN, LOUIS. Osteoid-osteoma with multicentric nidus: a report of two cases. *J. Bone & Joint Surg.*, June, 1973, *55-A*, 855–858. (From: Orthopaedic Department, Presbyterian Hospital, and Department of Pathology, University of California School of Medicine, San Francisco, Calif.)

Two case reports describe the presence of more than one nidus in an osteoid-osteoma.

1. A 12 year old boy with pain for 7 years had cortical thickening of the femur with one nidus 2 cm. in length and another 1 cm. Surgery confirmed the roentgenographic changes.
2. A 17 year old boy had previous surgery for osteoid-osteoma with curettage only. Pain recurred shortly and roentgenograms 2 years later showed localized cortical thickening with 3 separate radiolucent foci. This area was removed by *en bloc* resection and the photomicrographs demonstrated the 2 separate osteoid-osteoma foci.

The authors emphasize that roentgenograms should be obtained in surgery of the excised specimen as well as the operative site to confirm that no portion of the lesion persists.—*Martha Mottram, M.D.*

KILLEBREW, KATE, GOLD, RICHARD H., and SHOLKOFF, STEPHEN D. Psoriatic spondylitis. *Radiology*, July, 1973, *108*, 9–16. (From: Department of Radiology, University of California School of Medicine, San Francisco, Calif.)

Twenty-one patients with psoriatic arthritis were evaluated for spondylitis. Twenty patients were men. All 21 of these patients showed evidence of sacroiliac disease with subchondral erosion, sclerosis or fusion. In the majority (62 per cent), the erosions were symmetric. In 2 of 5 patients with asymmetric erosion, progression to bilaterally symmetric erosion was noted.

Syndesmophytes were visualized in the cervical spine in 60 per cent of the patients; in the thoracic spine in 66 per cent of the patients; and in the lumbar spine in 71 per cent of the patients. The syndesmophytes are characteristically large, coarse, asymmetric, and tend to skip vertebrae. Apophyseal fusion was observed in only 4 patients in the cervical spine, 1 patient in the thoracic spine, and 3 in the lumbar spine.

Squaring off of the vertebral bodies was visualized in 4, 7, and 19 patients, respectively, in the cervical, thoracic, and lumbar areas. Paravertebral ossification was not seen in the cervical spine and was seen in 5 patients in the thoracic spine and in 1 patient in the lumbar spine. Additional discussion of atlanto-axial subluxation is noteworthy. The authors believe that this subluxation is a characteristic feature of psoriatic spondylitis.

In conclusion, the authors indicate that sacroiliitis is the most common manifestation of psoriatic spondylitis. Syndesmophytes are the next most common finding. Atlanto-axial subluxation was found in 45 per cent of the patients and according to the authors is a characteristic feature of psoriatic spondylitis.—*J. P. Eberts, M.D.*

MURPHY, MICHAEL C., SHINE, IAN B., and STEVENS, DAVID B. Multiple epiphyseal dysplasia: report of a pedigree. *J. Bone & Joint Surg.*, June, 1973, *55-A*, 814–820. (From: Department of Community Medicine, University of Kentucky Medical Center, Lexington, Ky.)

One hundred and eleven persons in 6 generations of one family were studied and 24 subjects had multiple epiphyseal dysplasia.

There was an autosomal dominant inheritance with complete penetration and some variation in expression. Most of the patients noted the first symptoms between 5 and 10 years of age with waddling gait, small hands, short stature, or pain, swelling and stiffness of the large joints. Joint stiffness was the most disabling symptom. Six patients had intermittent paresthesias or temporary paralysis without anatomic explanation.

Investigation of patients with joint dysplasia should include roentgenograms of all major bones and joints.

Roentgenograms in this series showed the typical features of epiphyseal dysplasia involving the shoulder and hip joints, short metacarpals, and abnormal epiphyseal centers. Ten out of 12 patients had a slanting ankle joint mortise. The most obvious physical findings were short puffy digits and hands and short stature. Unusual dermatoglyphic patterns

were obtained. Three patients had osteochondritis dissecans and 1 had Blount-Barber disease.

Since the arthritic symptoms progress with age, the authors urge all family members with this condition to moderate their activity.—*Martha Mottram, M.D.*

Field, Joseph H., and Krag, Daniel O. Congenital constricting bands and congenital amputation of the fingers: placental studies. *J. Bone & Joint Surg.*, July, 1973, *55-A*, 1035–1041. (Address: Dr. Joseph H. Field, 259 Meridian Avenue, San Jose, Calif. 95126.)

The purpose of this paper is to present studies of 2 human placentas from patients who had the constricting bands popularly called Streeter's dysplasia and to support Torpin's concept of their pathogenesis.

It was found that in 2 infants with multiple involvement with congenital bands and partial amputation of the fingers, gross and microscopic examination of the placenta served to substantiate the concept of amnion rupture during development as the cause of the abnormalities.—*John H. Harris, Sr., M.D.*

Casey, Bernard M., Eaton, S. Boyd, Jr., Du Bois, James J., Tremaine, Myron D., and Mani, Carl J. Thoracolumbar neural arch fractures: evaluation by hypocycloidal tomography. *J.A.M.A.*, May, 1973, *224*, 1263–1265. (Address: James J. Du Bois, M.D., Department of Radiology, Letterman General Hospital, The Presidio of San Francisco, San Francisco, Calif. 94129.)

Twenty-eight patients who had sustained varying degrees of spinal trauma were examined by means of conventional roentgenography and hypocycloidal tomography. Conventional studies included anteroposterior, lateral and (in most instances) oblique roentgenograms. Hypocycloidal tomography was performed in the anteroposterior position only. Neural arch fractures were considered present, if the pedicles, laminae or articular processes (or all 3) could be shown to be disrupted. By these criteria, neural arch fractures were shown by tomography in 12 (43 per cent) of the 28 patients. Of these 12 neural arch fractures, only 2 (17 per cent) were detected at the initial interpretation of the conventional roentgenograms. Because of this discrepancy the conventional roentgenograms were then reviewed with specific attention paid to the neural arch integrity. Under these conditions, only 4 (33 per cent) of the 12 neural arch fractures went undetected, but there were 5 (41 per cent) false-positive diagnoses.

As treatment for thoracolumbar spinal fractures depends largely on whether or not the injury is stable or unstable, accurate diagnosis is important. Stable fractures are those in which the neural arches and posterior ligaments complex remain intact; if these are disrupted, the fracture is considered unstable. Unfortunately, neural arch fractures are difficult to detect on conventional roentgenograms, especially if the patient is maintained in a supine position throughout the examination. Tomographic study, however, is ideally suited for neural arch evaluation.

It is concluded that hypocycloidal tomography should occupy a position of importance for the assessment of thoracolumbar spinal injuries.—*David Morse, M.D.*

Snarr, Jack W., Abell, Murray R., and Martel, William. Lymphofollicular synovitis with osteoid osteoma. *Radiology*, March, 1973, *106*, 557–560. (Address: Jack W. Snarr, M.D., Department of Radiology, University of Michigan Medical Center, Ann Arbor, Mich. 48104.)

Lack of familiarity with the radiologic and histologic features of intraarticular osteoid osteoma often causes diagnostic errors.

With this in mind the authors report 5 cases of intraarticular osteoid osteoma occurring in the hip and elbow. These were surgically proven cases from the radiologic files at the University of Michigan, Medical Center. Seven cases of intraarticular mass lesions, other than osteoid osteoma, which were associated with bone destruction were also reviewed.

Certain features not generally associated with these lesions are emphasized. These include: uniform narrowing of the interosseous space; subperiosteal bone apposition involving the affected bone as well as the adjacent bone; and lymphofollicular inflammation in the adjacent soft tissue. This latter feature was felt to represent a consistent finding in intraarticular osteoid osteoma and not associated with other types of intraarticular mass lesion. It was noted further that such inflammation may involve the periosteal soft tissue at a distance from the joint. The similarity of this inflammatory response to rheumatoid arthritis is very interesting.

It has been postulated that a specific activator substance is responsible for the inflammation of synovial tissues in rheumatoid arthritis. One may speculate that perhaps such an activator substance is also elaborated in the presence of osteoid osteoma in the vicinity of synovial tissue.—*W. O. Havens, M.D.*

General

Pearson, Kent D., Wells, Samuel A., and Keiser, Harry R. Familial medullary carcinoma of the thyroid, adrenal pheochromocytoma and parathyroid hyperplasia: a syn-

drome of multiple endocrine neoplasia. *Radiology*, May, 1973, *107*, 249–256. (Address: Harry R. Keiser, M.D., Experimental Therapeutics Branch, National Heart and Lung Institute, National Institute of Health, Bethesda, Md. 20014.)

The clinical, pathologic, and radiographic features of a syndrome of familial medullary carcinoma of the thyroid, adrenal pheochromocytomas and parathyroid hyperplasia are presented. The authors assessed 21 members of a family with this familial multiple endocrine neoplasia.

Medullary carcinoma comprises 3.5–10 per cent of malignant thyroid tumors. It occurs both in sporadic cases and families. Medullary carcinoma tends to appear as multiple foci within both lobes and arises from the perifollicular cells of the thyroid gland. Both pheochromocytomas and medullary carcinoma of the thyroid are of neuroectodermal origin.

Pheochromocytomas associated with medullary carcinomas are bilateral in 70–80 per cent of the cases, whereas sporadic pheochromocytomas are bilateral about 9 per cent of the time. Associated parathyroid disease may be either parathyroid adenomas or parathyroid hyperplasia. Patients with medullary carcinoma of the thyroid have a persistently elevated calcitonin and the application of a radioimmunoassay for calcitonin has lead to early detection of medullary carcinoma of the thyroid in high risk families.

Dense calcifications were present in tumor deposits of medullary carcinoma of the thyroid in the neck in 7 of 19 patients examined. The calcifications tend to be large and discrete. Similar calcifications were also present in metastatic deposits in cervical lymph nodes and the liver. Seven patients had cervical lymph node metastases at the time of surgery, and 6 had metastases to the liver. Widespread metastases to the liver, lung, bone, and other organ systems were present in 2 patients. The pulmonary lesions in these 2 patients were widespread and of the interstitial nodular type and were most prominent in the mid lung fields. Pulmonary hyperaeration developed in both patients and there was extensive mediastinal and hilar lymph node involvement.

Calcification of articular cartilage may be present, particularly involving the knee and wrist. Aspiration of the knee joint in 1 patient documented the presence of calcium pyrophosphate crystals. None of the patients reviewed had subperiosteal resorption of bone, clavicular erosions, or cyst-like changes frequently associated with hyperparathyroidism.

Arteriographic abnormalities in 8 patients studied varied considerably from an extremely vascular tumor mass to filling defects of varying size and number. Widening and displacement of the loops of the inferior thyroid artery may be present. Assay of calcitonin and parathormone from samples obtained by selective catheterization of the thyroid venous bed is a useful diagnostic procedure.

I^{131} and Tc^{99m} scans were normal in those patients with multiple foci of medullary carcinoma in both glands and have shown areas of decreased uptake in the large tumors.—*J. L. Montgomery, M.D.*

Vermess, Michael, Waldmann, Thomas A., and Pearson, Kent D. Radiographic manifestations of primary acquired hypogammaglobulinemia. *Radiology*, April, 1973, *107*, 63–69. (Address: Michael Vermess, M.D., Department of Diagnostic Radiology, The Clinical Center, Building 10, Room 6S-211, National Institutes of Health, Bethesda, Md. 20014.)

Primary acquired hypogammaglobulinemia is an immunodeficiency syndrome or group of syndromes defined by the fact that immunologic deficiency and increased incidence of infection occurs spontaneously following a period of apparently normal immunologic function. Immunologically these patients lack plasma cells and have markedly decreased serum immunoglobulin levels due to decreased synthesis of these proteins and this leads to minimal or no antibody response to antigenic challenges. Clinically, these patients will show an increased susceptibility to pyogenic infections leading to episodes of sinus and pulmonary disease. Many of these patients also have recurrent otitis media, conjunctivitis, skin infections; and frequently a sprue-like syndrome with diarrhea, steatorrhea, occasionally with protein losing enteropathy, will be observed.

The authors report on their study of a group of 24 patients with primary acquired hypogammaglobulinemia seen at the National Institutes of Health. All of these patients showed markedly reduced concentrations of the 3 major classes of immunoglobulins in their serums. The 11 men and 13 women in this study ranged in age from 11 to 68 years. All of the available radiographic examinations on these patients were evaluated.

All the patients in the series had chest examinations and 22 of these had one or more abnormal chest roentgenograms. The most frequently found changes were those of repeated acute inflammatory processes consisting of variable patterns of bronchopneumonia, lobar pneumonia and atelectasis frequently leading to the development of bronchiectasis, fibrosis, and chronic obstructive lung disease. Of the 21 patients, who had roentgenograms of their sinuses, 20 showed abnormal findings consisting most frequently of maxillary sinus infections, often with associated involvement of the other sinuses. Eight patients had mastoid studies and all showed evidence of acute or chronic inflammatory disease on one or both sides and all of these had associated disease of the maxillary sinuses.

Roentgenograms showing the left upper quadrant

of the abdomen were available in 22 patients and 15 of these were felt to have some degree of splenomegaly, and hepatomegaly was noted in 7.

Of 15 patients, in whom gastrointestinal tract contrast examinations were available 10 were found to be abnormal. The most common change, found in 5 patients, was thickening of the valvulae conniventes. Two of these had additional changes such as dilatation, hypersecretion, and segmentation demonstrating a sprue-like syndrome. Four patients had mucosal nodularity consistent with lymphonodular hyperplasia with the size of these nodules varying from 2 to 4 mm. and involving the small bowel either diffusely or at a localized segment. One patient showed both thickening of the mucosal folds and nodularity of the small bowel. All of these 10 patients had chronic diarrhea and 8 of them had steatorrhea and malabsorption. Six of these 10 showed infestation by *Giardia lamblia*. The stomach was considered normal in all of these patients.

The differential diagnoses which should be considered in patients with all of these findings are Wegener's granulomatosis, Kartagener's triad, and cystic fibrosis of the pancreas. Since lung disease, sinusitis, and splenomegaly have been found in the majority of patients in this and other series, it is the authors' feeling that this combination of findings is an occasionally useful triad in the diagnosis of primary acquired hypogammaglobulinemia. It is also felt that since the roentgenologist is among the first of the physicians to encounter patients with immunodeficiency disease, he is in a good position to contribute to an early diagnosis of this condition and possibly help prevent the devastating and crippling consequences of this disease.—*Donald N. Dysart, M.D.*

RADIATION THERAPY

BIRKS, DAVID M., CRAWFORD, G. M., ELLISON, L. G., and JOHNSTONE, F. R. C. Carcinoma of the breast in women 30 years of age or less. *Surg., Gynec. & Obst.*, July, 1973, *137*, 21–25. (From: British Columbia Cancer Institute and the Department of Surgery, Faculty of Medicine, University of British Columbia, and Vancouver General Hospital, Vancouver, British Columbia, Canada.)

During a 25 year period up to 1966, 5,038 patients with carcinoma of the breast were seen in the British Columbia Cancer Institute, and 58 of these were 30 years of age or less. This young group was investigated to determine whether or not cancer of the breast in women under the age of 30 years was more lethal than that found in older women.

Of the group of 58, only 5 were less than 25 years of age. The presenting complaint in 43 was a solitary mass in the breast; 39 had symptoms for less than 6 months; and 19 were seen within 1 month of the first complaint. Only 3 had mothers who had had carcinoma of the breast, and all 3 of the young patients died of the disease within less than 5 years.

In 49 patients staging of the disease was possible on the basis of histologic material.

The primary treatment of these women was the same as in older women. Thirty-one were subjected to the standard Halstead radical mastectomy and postoperative radiotherapy, and another 5 had, in addition, surgical castration. Seven received radical irradiation alone, and another 7, radical irradiation and castration. The radiation treatment varied according to the pathologic findings.

The crude survival rates for these young women are the same as those for older women seen during the same 25 year period in this institution. In both groups the best survival rate, of course, was in the women with no axillary lymph node involvement.

In the total group of 58, there were 10 who were considered inoperable when first seen, and 3 of these survived 5 years or more even though no potentially curative surgical procedure was attempted. No difference was shown between the younger and older groups when compared as to duration of symptoms.

In 16 of the 58 patients there were 23 subsequent pregnancies, and 15 occurred within 5 years of diagnosis and treatment. Three of the 16 patients subsequently died of carcinoma of the breast, but all 3 had evidence of axillary lymph node involvement at the time of the initial operation. Of 2 patients who presented for treatment during pregnancy, 1 subsequently died of the disease, and the other is alive 8 years later. These figures suggest that subsequent pregnancies affect the prognosis adversely.

The risk of bilateral carcinoma of the breast in these women cannot be assessed because of the short observation period. The incidence of a second primary carcinoma might be increased because of the greater life span in the younger survivor.—*E. Frederick Lang, M.D.*

RADIOISOTOPES

HAYS, MARGUERITE T. ^{99m}Tc-pertechnetate transport in man: absorption after subcutaneous and oral administration; secretion into saliva and gastric juice. *J. Nuclear Med.*, June, 1973, *14*, 331–335. (Address: M. T. Hays, Nuclear Medicine Service, VA Hospital, 3495 Bailey Avenue, Buffalo, N. Y. 14215.)

Fifteen normal subjects were studied in multiple sessions, after administration of $Tc^{99m}O_4$, by the oral, subcutaneous, and intravenous routes.

Using plasma radioactivity after intravenous administration as the norm for 100 per cent absorption, subcutaneous $Tc^{99m}O_4$ was absorbed promptly

with 95 per cent or more absorption occurring 45.8 ±22.7 (S.D.) minutes after injection.

Orally administered $Tc^{99m}O_4$ was erratically absorbed with marked variability in the timing and extent of absorption. This variability occurred between subjects and between sessions in a single subject. The addition of human serum albumin to bind the oral $Tc^{99m}O_4$ did not significantly affect absorption.

Six patients with gastrointestinal disease were studied for absorption of oral $Tc^{99m}O_4$ and for quantification of $Tc^{99m}O_4$ and I^{131} transfer into the saliva and gastric juice after intravenous administration. Gastric juice pH did not affect absorption of oral $Tc^{99m}O_4$. Gastric juice/plasma (G/P) and saliva/plasma (S/P) ratios were greater for I^{131} than for $Tc^{99m}O_4$ in all instances when measured simultaneously.—*William T. McLaughlin, M.D.*

Piepsz, A., Decostre, P., and Baran, D. Scintigraphic study of pulmonary blood flow distribution in cystic fibrosis. *J. Nuclear Med.*, June, 1973, *14*, 326–330. (Address: A. Piepsz, Department of Pediatrics, Hôpital Saint-Pierre, 322 rue Haute, 1000, Brussels, Belgium.)

Considerable alterations of lung perfusion are evident in cystic fibrosis, although no primary vascular involvement has been shown.

Seventy-three children from the age of 6 weeks to 19 years have been investigated by means of perfusion scintigraphy with a scintillation camera.

The examinations were indexed as follows: Stage 0. No detectable lesions; Stage 1. Minimal localized involvement of apex, hilum, or bases; Stage 2. Advanced but localized lesions of apex, hilum, or bases; Stage 3. Generalized diffuse involvement of 1 or 2 lungs.

Special attention was paid to the actual clinical status (remission or acute phase) at the time of examination. In the remission phase, correlation was found between the perfusion abnormalities and the severity of the disease. In the acute phase, striking transient alterations developed; their prognostic value should be determined by further investigation.—*William T. McLaughlin, M.D.*

Mitchell, Marvin L. Normalized resin uptake of displaced ^{125}I-T_4 using denatured serum. *J. Nuclear Med.*, June, 1973, *14*, 336–340. (Address: Marvin L. Mitchell, State Laboratory Institute, Biologic Laboratories, 375 South Street, Boston, Mass. 02130.)

A procedure has been devised that exploits the ability of heat-denatured serum to displace I^{125}-T_4 from a source of labeled thyroxine-binding protein onto a resin sponge.

Heat-treated sera from 20 pregnant women and women on oral contraceptives resulted in resin uptake values of displaced I^{125}-T_4 that were the same as those in 57 normal controls. However, resin uptake values in 10 hypothyroid patients were significantly lower, and in 22 hyperthyroid patients were significantly higher than in the normal or pregnant groups.

This procedure has "normalized" the behavior of sera with elevated thyroxine-binding capacities, thereby eliminating a major source of confusion seen with measurements of total thyroxine and the resin T_3 uptake.—*William T. McLaughlin, M.D.*

MISCELLANEOUS

May, R., and Nissl, R. Primäre essentielle Phlebosklerose, perivenöse Kalzinose, manschettenförmige venöse Stauungsossifikation: ein Versuch einer neuen Klassifizierung der Phlebosklerose. (Primary essential phlebosclerosis, perivenous calcinosis, sleevelike ossification in venous insufficiency: an attempt of a new classification of phlebosclerosis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, June, 1972, *116*, 789–794. (Address: Dr. R. Nissl, Neuhäuserstrasse 2, 6020 Innsbruck, Austria.)

Based on the radiographic and histologic differences of venous calcifications a new classification of phlebosclerosis is suggested: (1) primary essential phlebosclerosis, in which delicate amorphous calcifications in the walls of varicose veins are found; (2) perivenous calcinosis, in which heavy calcific plaques are seen along the large veins. These calcific deposits are in the perivenous fat and connective tissues; this type is very rare, only 4 cases were seen by the authors within a period of 25 years; and (3) sleevelike ossification in venous insufficiency, which consists of web-like plaques of subcutaneous ossifications without calcareous deposits in the walls of the veins. This type of phlebosclerosis is found only in the lower legs.

The pathogenesis of the different venous calcifications is discussed.—*Peter F. Winter, M.D.*

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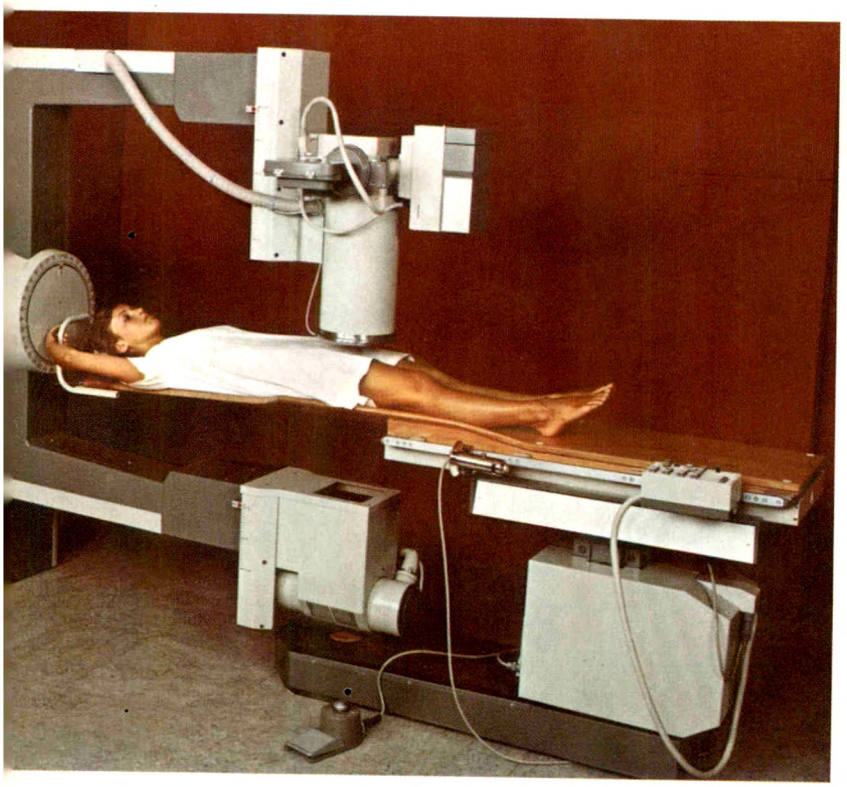
Zanon, Bert, Jr., *335
Zaret, Barry L., 675

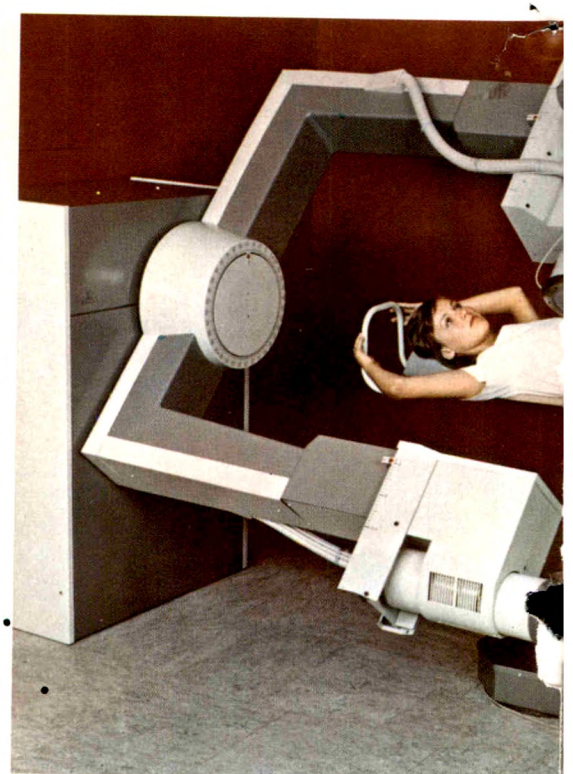
SIEMENS

Everything revolves around the heart.



Cardoskop U. For the kind of cardio-centric examinations you may have thought impossible.



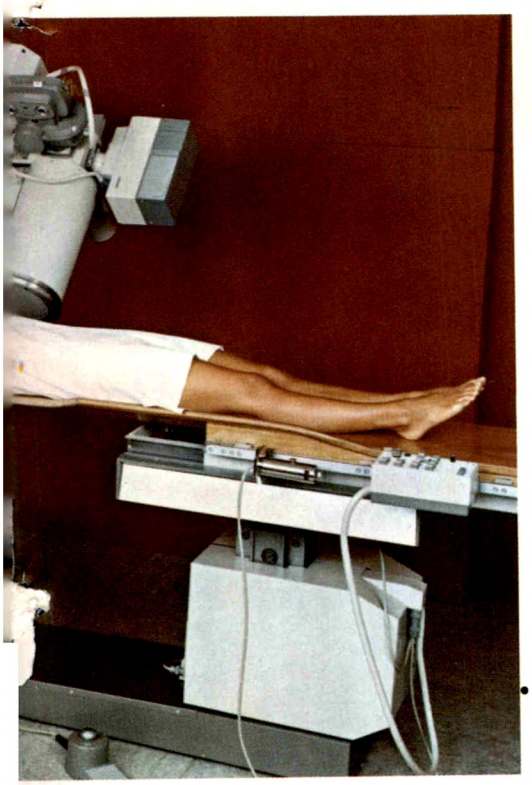


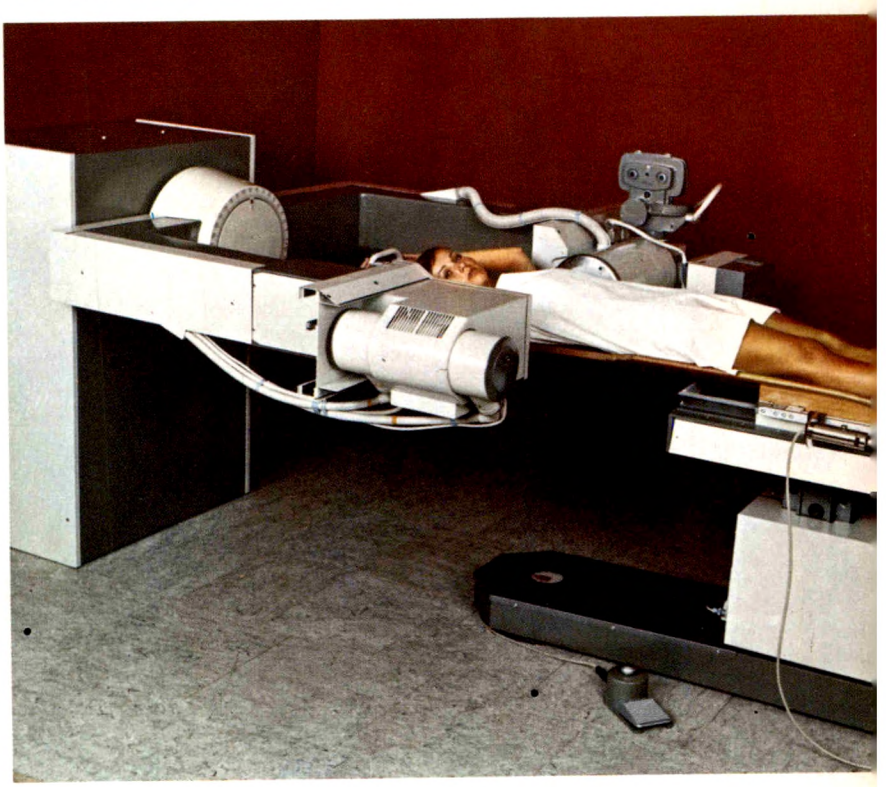
Special radiographic procedures for angiocardiography and coronary angiography have typically required that the patient be placed in a cradle and then rotated to the proper angle for x-ray projections.

This is no longer necessary with Siemens Cardoskop U, because the U-arm rotates and the patient remains stationary. You can now perform examinations that used to be extremely difficult, if not impossible.

Quick longitudinal, transverse and vertical adjustments under fluoroscopic control are readily achieved with Siemens floating table top. The U-arm swivels ±110° for all radial oblique or lateral projections. By turning the table up to 100°, you even get cranial or caudal oblique projections. And the patient's heart remains in the center.

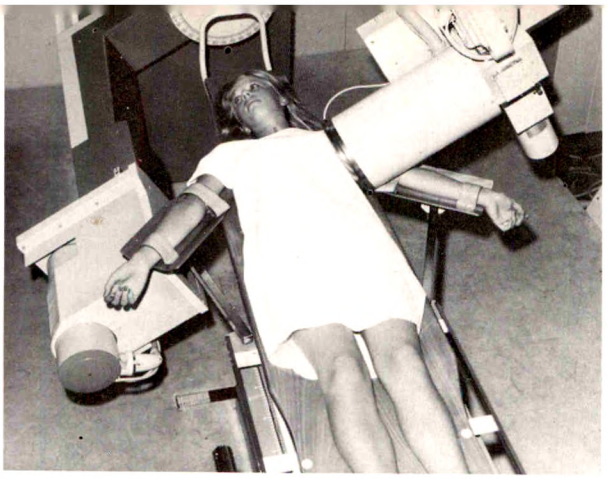
Cardoskop U comes with a high-resolution 7-inch image intensifier, or a dual-mode 10/6-inch image intensifier. The two- or three-channel light distributor to which the TV camera is attached can be fitted with a 70mm rollfilm camera, 100mm cutfilm, 105mm rollfilm, or 35mm cine camera.



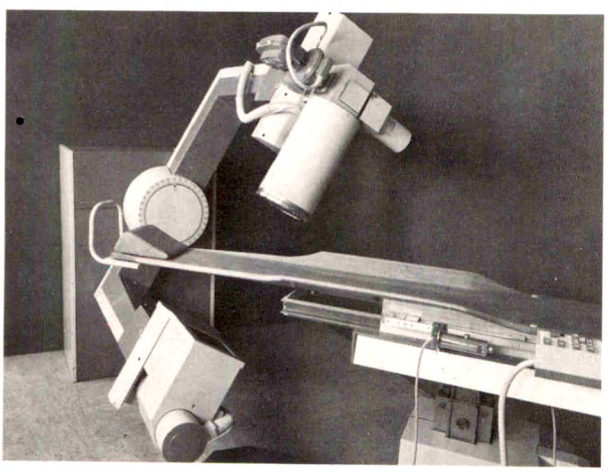


Cardoskop U. A state of the art system for cardiocentric angiocardiography and coronary angiography. As with all Siemens x-ray systems, Cardoskop U has been designed in response to professional requirements.

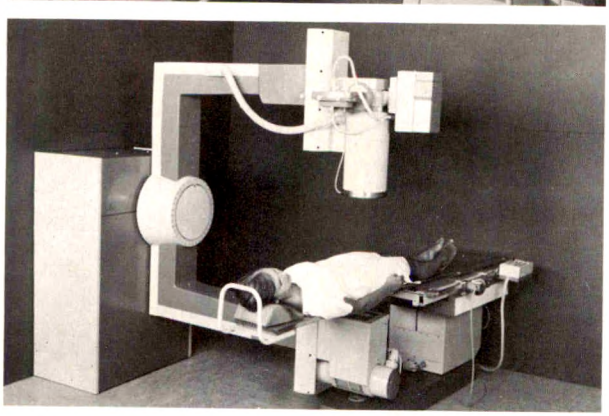
For complete information, get in touch with your local Siemens office, or the Medical Systems Division in Iselin, New Jersey.



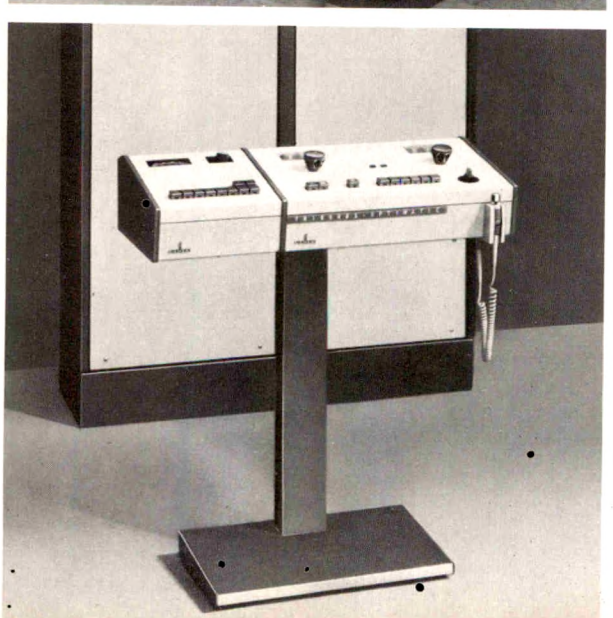
Special armrests built into the Cardoskop U examination table make catheterization techniques easier.



Only Siemens Cardoskop U provides "mixed" oblique projections, often used to set certain valve planes on head-wall areas in the projection, or perpendicular to it.



Emergency cardiac treatment, thoracic examinations and oblique projections are performed easily by swiveling the table top 90°.



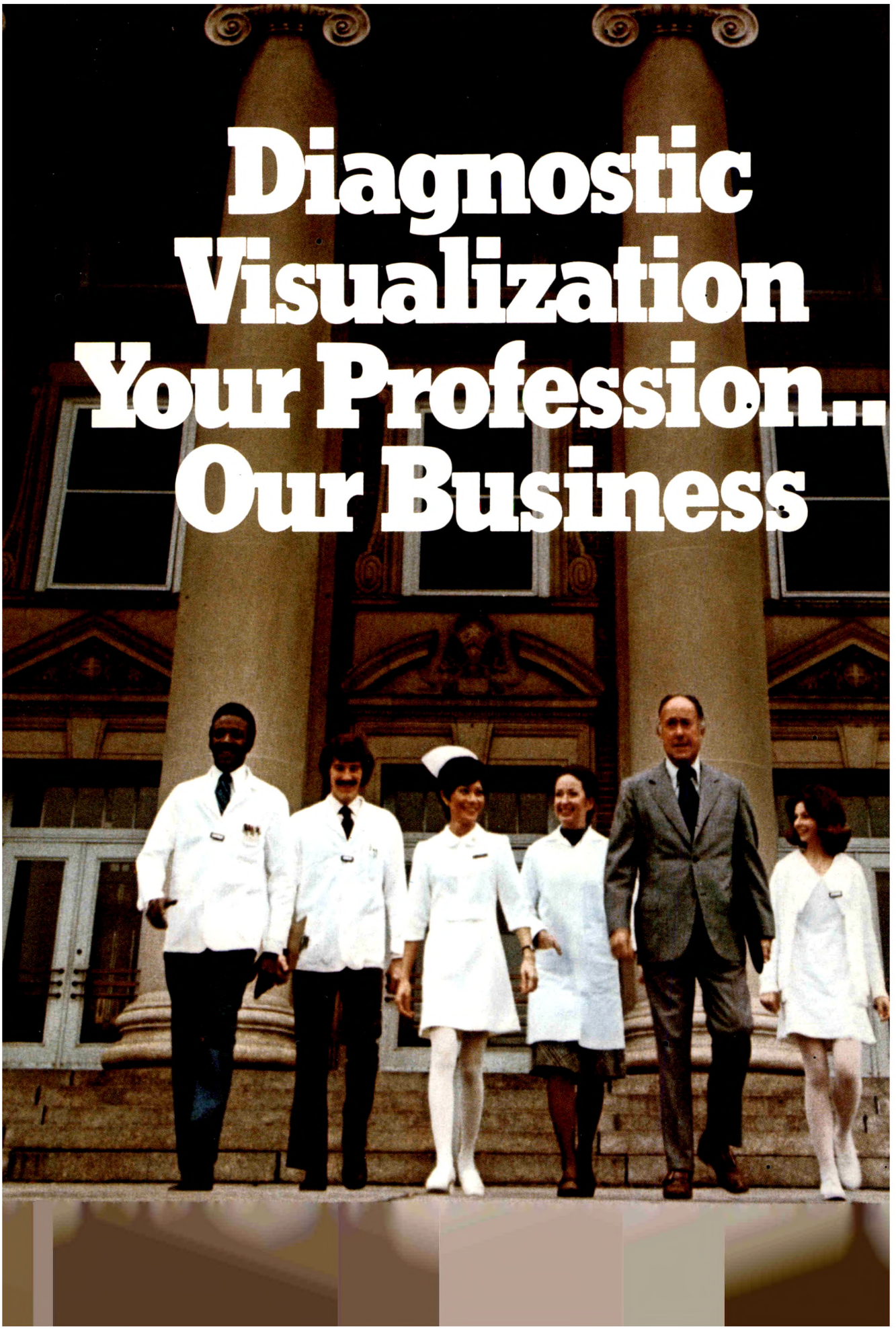
The New Optimatic Generator system perfectly matches the Cardoskop U. **Benefits include:**

- Time-savings, through simple operating techniques.
- Organ-related preselection of programmed radiographic data.
- Independent working at each examination unit, through storage of the selected data.
- Individual setting of radiological data at a master console.

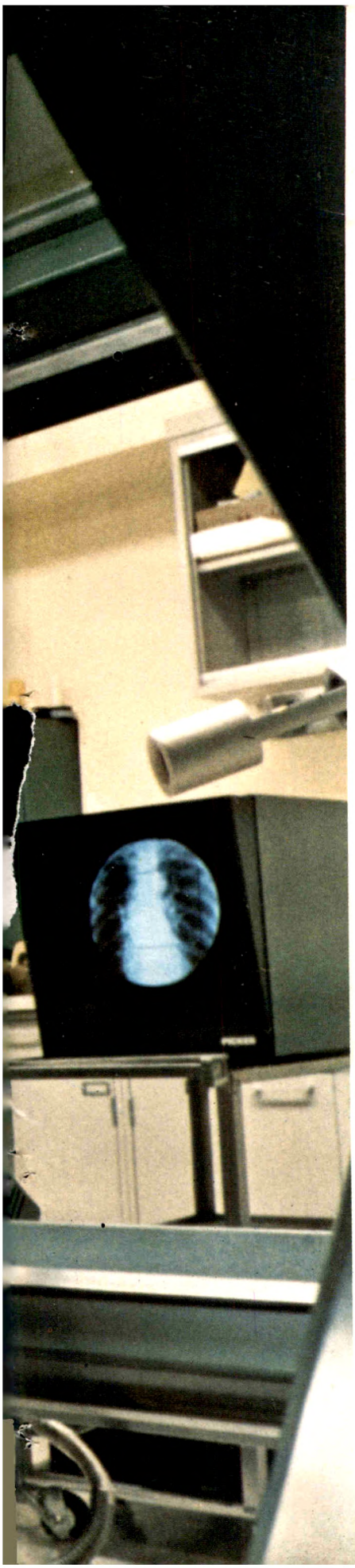
Tridoros Optimatic: 50 kw.
Gigantos Optimatic: 100 kw.
Pandoros Optimatic: 150 kw.

Siemens Corporation
Medical Systems Division
186 Wood Avenue South
Iselin, New Jersey 08830

In Canada: Siemens Canada Limited
P.O. Box 7300, Pointe Claire 730-P.Q.


Diagnostic
Visualization
Your Profession..
Our Business

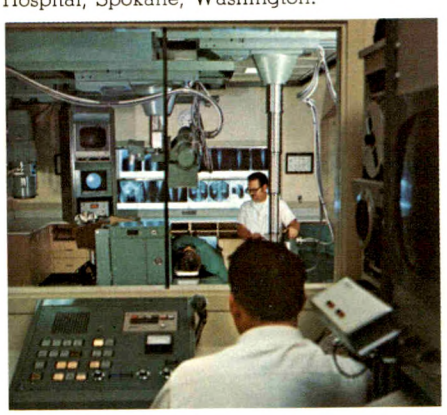

Picker's business philosophy is simple: Total commitment to better diagnostic visualization and radiation therapy. Picker is a pioneer, an innovator and a major producer of state-of-the-art systems used in diagnostic x-ray, therapy, nuclear medicine, and ultrasound. Picker also provides most of the supplies and accessories needed to operate these departments. And Picker backs its efforts with the largest medical technical-service organization in the field. Picker marches to a different beat because serving your profession is our only business.



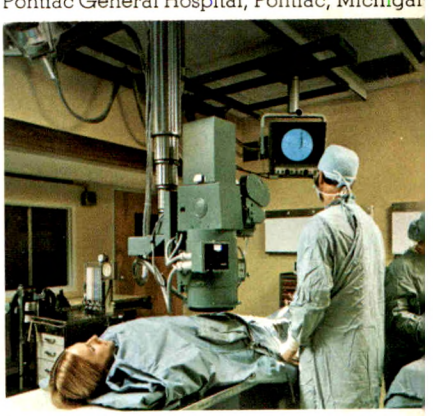
Special-Purpose Rooms

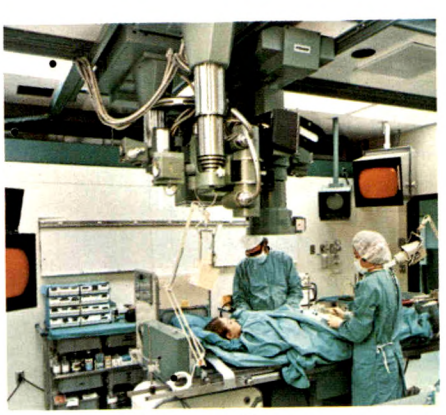
Vast experience in virtually all phases of the x-ray discipline has given Picker a synergistic know-how that enables us to serve the most demanding requirements in diagnostic special procedures. Picker's sophisticated designs result in greatest flexibility in adapting to caseload profiles . . . maximum efficienc for patient throughput . . . boost utilization of both space and facilities.

Special-Purpose Room. Photo courtesy Radiology Department, Holy Family Hospital, Spokane, Washington.

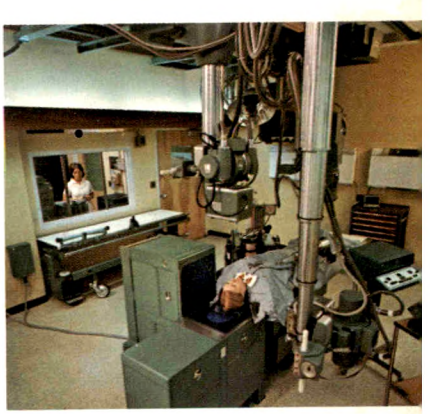


Special-Purpose Room. Photo courtesy Pontiac General Hospital, Pontiac, Michigar





Cardiology Laboratory. Photo courtesy Radiology Department, University Hospital of Jacksonville, Jacksonville, Florida.



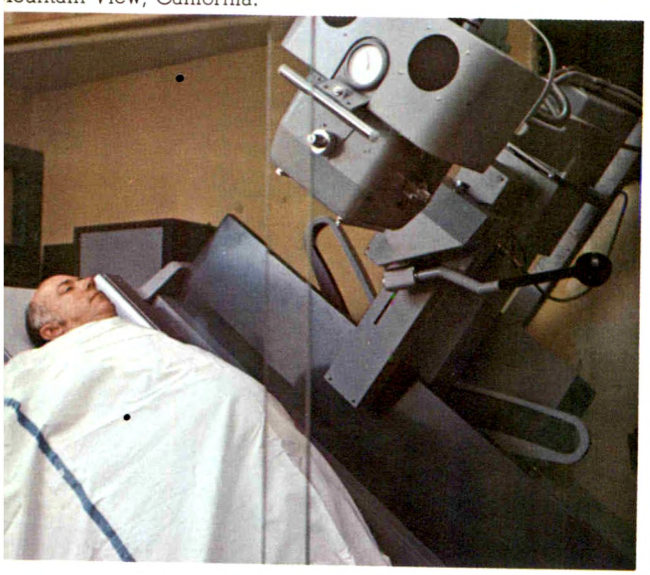
Special-Purpose Room. Photo courtesy Pontiac General Hospital, Pontiac, Michiga

Photo courtesy Rainbow Babies and Childrens Hospital of University Hospitals, Cleveland, Ohio

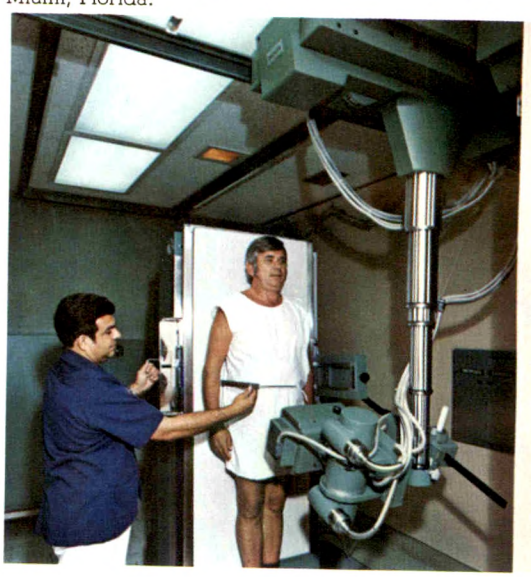
Radiographic & Fluoroscopic Rooms

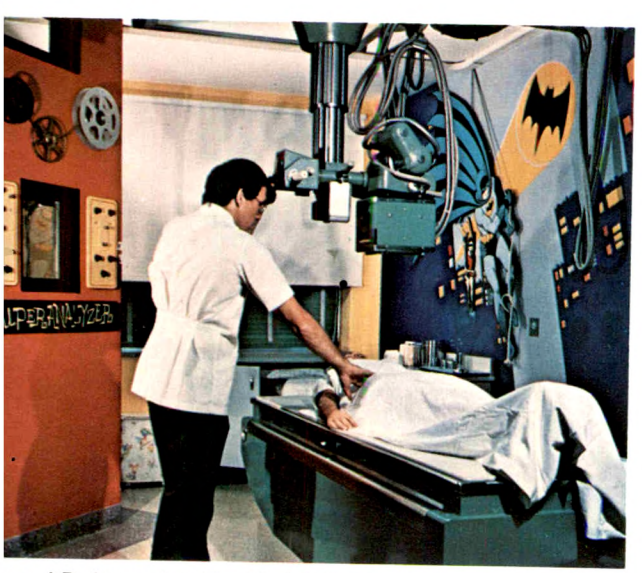
Picker carries the philosophy of the fully integrated diagnostic system almost to its ultimate degree in Radiographic & Fluoroscopic Rooms. In TV fluoroscopy, the entire process—including patient positioning, programming, phototiming, etc.—is precisely controlled and monitored. This enables the radiologist to obtain desired diagnostic data on film the first time so as to minimize unnecessary retakes.

Remotely-Controlled R&F Room contains Picker Epic remotely operated diagnostic x-ray system. Photo courtesy Radiology Dept., El Camino Hospital, Mountain View, California.

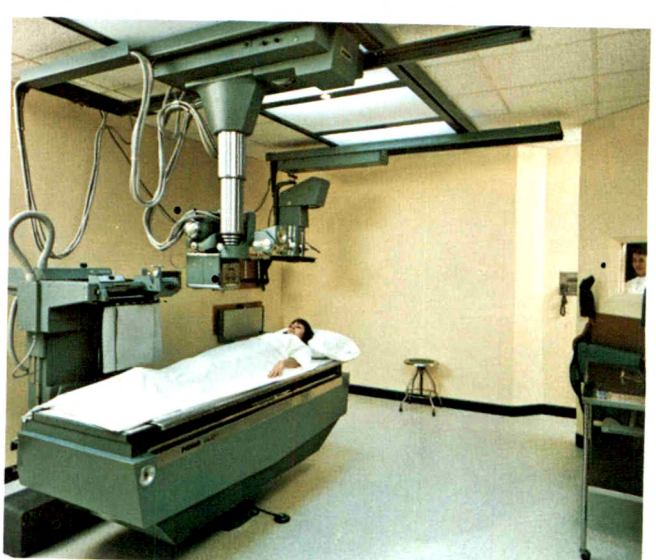


General Radiographic Room contains Galaxy table, Horizontal/Vertical Tomographic attachment, 800 mA generator, ceiling tube mount, Collimatic collimator. Photo courtesy Jackson Memorial Hospital, Miami, Florida.





neral Radiographic (Bat Man) Room contains ng radiographic table with manually adjustable limator, illuminated ceiling tube mount, 300 mA nerator. Photo courtesy Cardinal Glennon morial Hospital for Children, St. Louis, Missouri.



Radiographic/Fluoroscopic Room with Galaxy x-ray table, spot film device, image intensifier, ceiling tube mount, 500 mA generator. Photo courtesy Port Huron Hospital, General Hospital, Port Huron, Michigan.

Mammography

The Picker Mammorex® is a completely coordinated breast radiographic system – the first specialized mammography unit that consistently shows the entire breast, from nipple back to the rib cage. Because Mammorex shows the complete breast in every desired position, it significantly increases the possibility of seeing even the smallest microcalcifications. Superb image quality of Mammorex is a function of the latest state-of-the-art x-ray tube, especially designed for mammography, combined with the unique Mammorex breast compressor. Since Mammorex is an isocentric system that completely moves around the patient, examination time is significantly decreased.

Unretouched radiographs courtesy of Royal Victoria Hospital, Montreal.



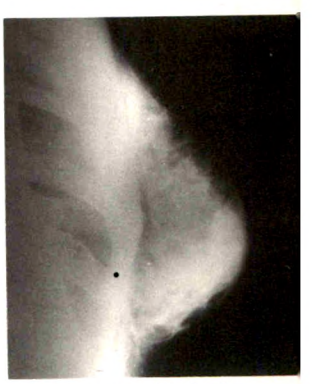
Mammorex is the first such specialized system that shows the entire breast all the way back to the rib cage.



Only when the chest wall is visible can one be sure that all of the breast is on the radiograph.



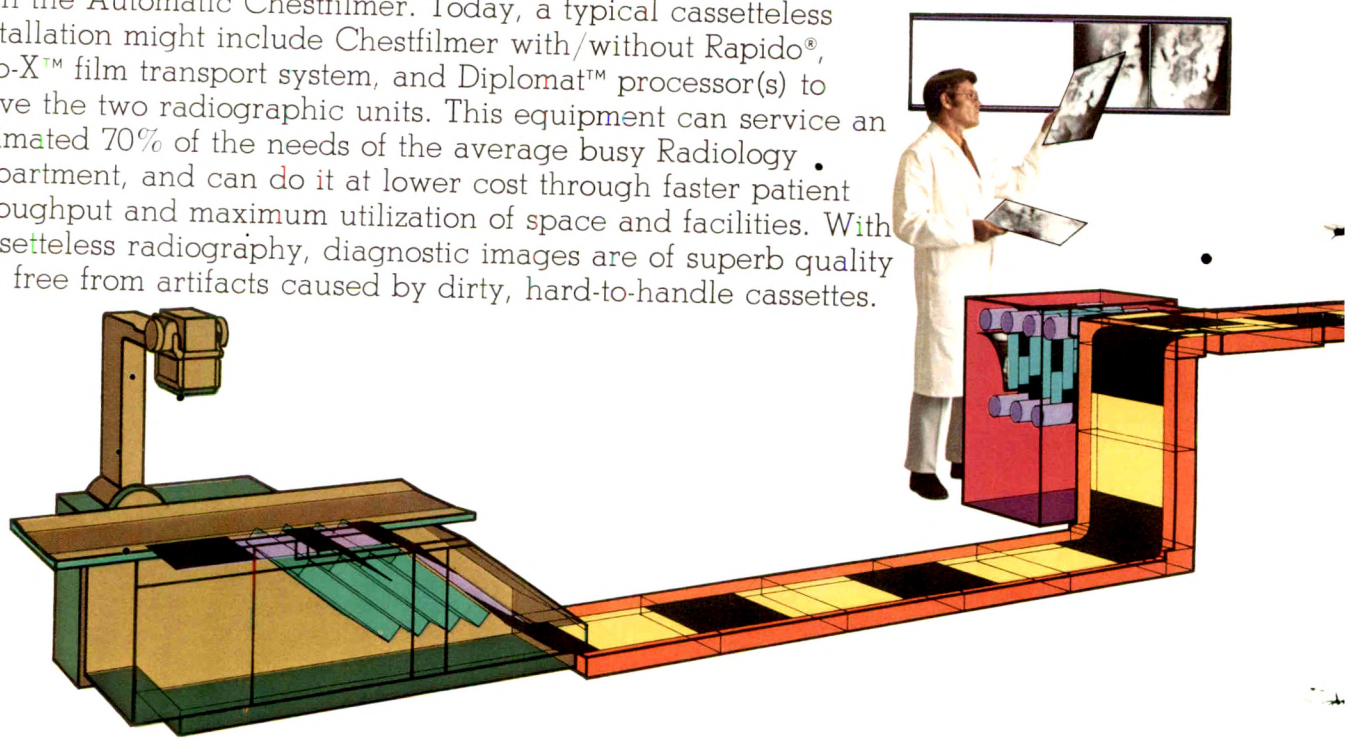
Malignancy shows no geographic preference; lesions proximal to the chest wall are visible with Mammorex.



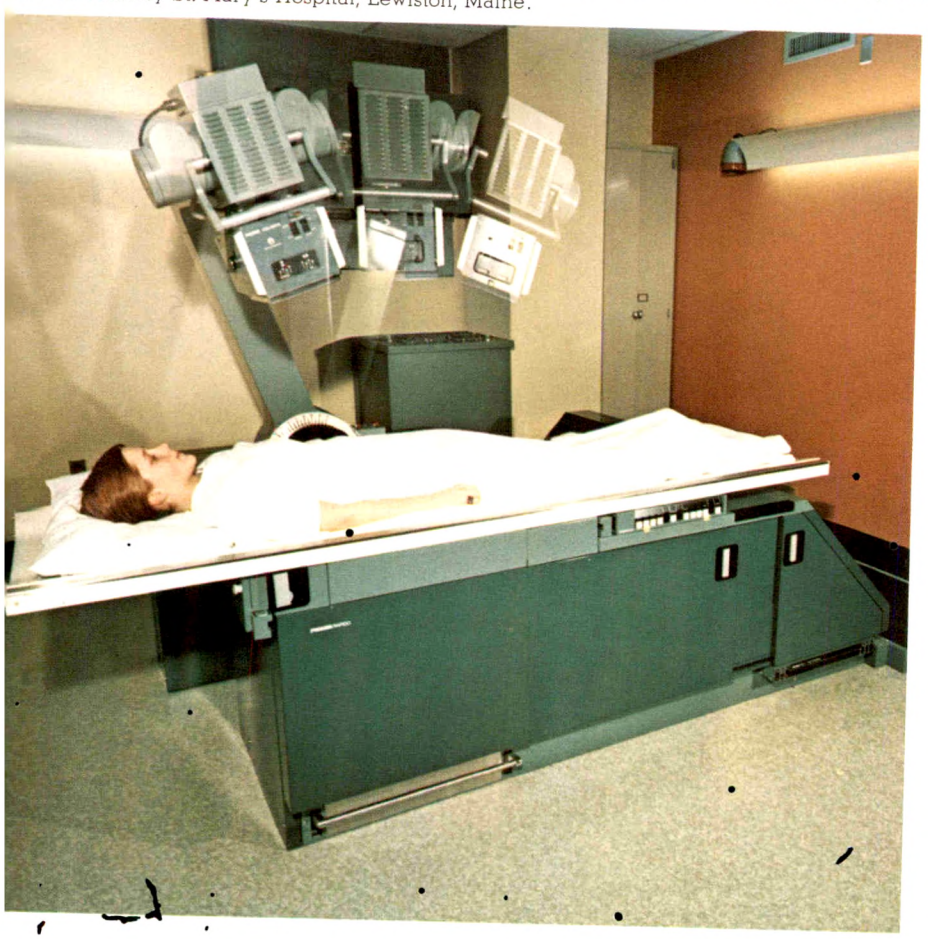
Mammorex yields high contrast even in the case of the dense young breast.

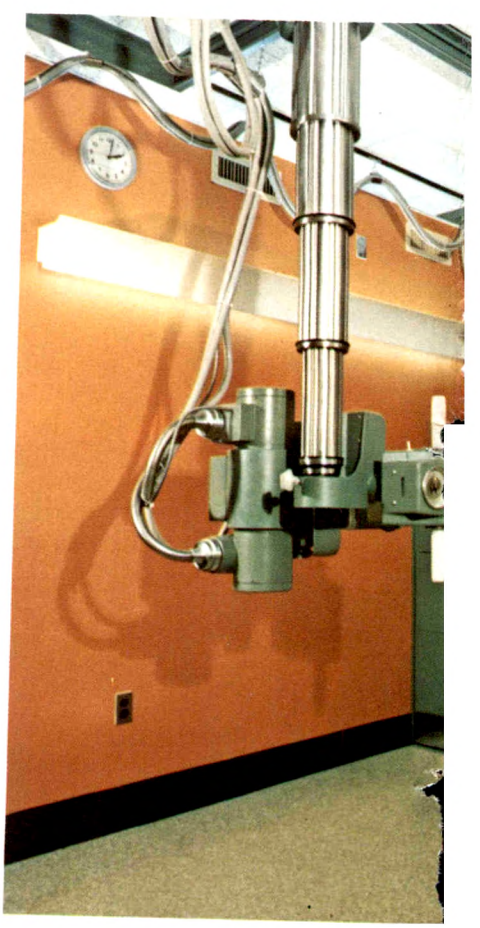
Cassetteless Radiography

Picker is the unquestioned leader in cassetteless radiography, having introduced the cassetteless concept several years ago with the Automatic Chestfilmer. Today, a typical cassetteless installation might include Chestfilmer with/without Rapido®, Rap-X™ film transport system, and Diplomat™ processor(s) to serve the two radiographic units. This equipment can service an estimated 70% of the needs of the average busy Radiology Department, and can do it at lower cost through faster patient throughput and maximum utilization of space and facilities. With cassetteless radiography, diagnostic images are of superb quality . . . free from artifacts caused by dirty, hard-to-handle cassettes.



Complete Cassetteless Radiography room contains automatic Chestfilmer and Rapido, both integrated with and feeding into automatic processor via Rap-X film transport system. A single generator with phototimer provides power to both rooms. Photos courtesy St. Mary's Hospital, Lewiston, Maine.

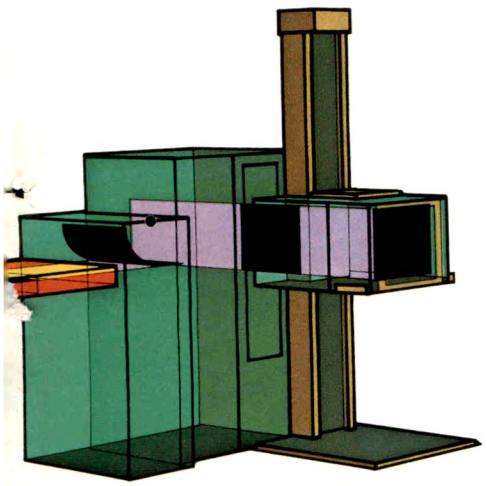




Ultrasound

Ultrasound approaches the ideal as a diagnostic tool in that it is noninvasive, nontraumatic, and nondestructive at normal diagnostic levels. Significant among its advantages is the abi of ultrasound to permit visualization of soft-tissue structures that cannot be seen well, or even at all, with radiography. Improved diagnostic procedures have been developed utilizi ultrasound in conjunction with x-ray and nuclear studies.

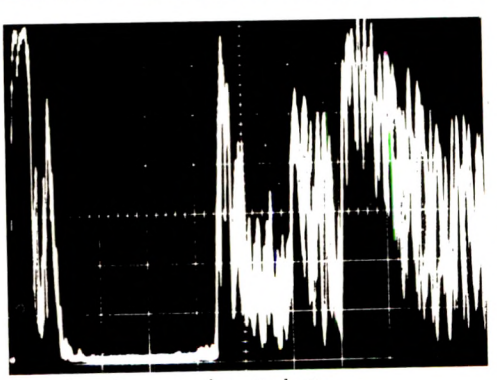
Ultrasound is finding widespread uses in cardiology, in hepatic and renal disease diagnoses, and in cerebral studies of various types. Perhaps the most significant use of ultrasound is in gynecology and obstetrics since it avoids ionizing radiation of the pregnant uterus. Picker Echoview™ systems, including the new Echoview X cardiac unit, are the world's best known and most universally used apparatus for imaging with ultrasound.





Echoview VI Laminograph being used in OB/GYN application.

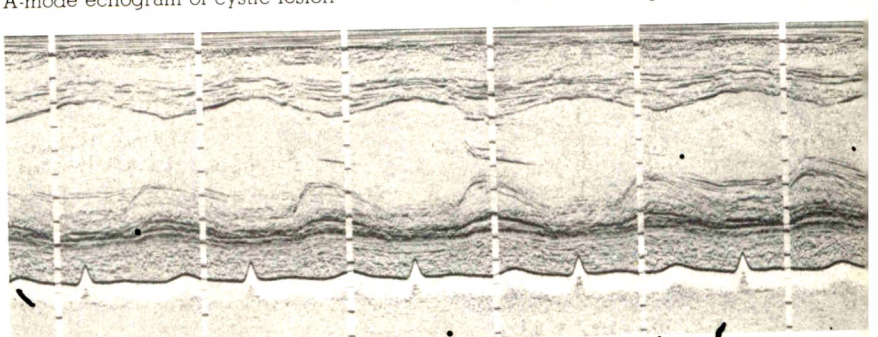




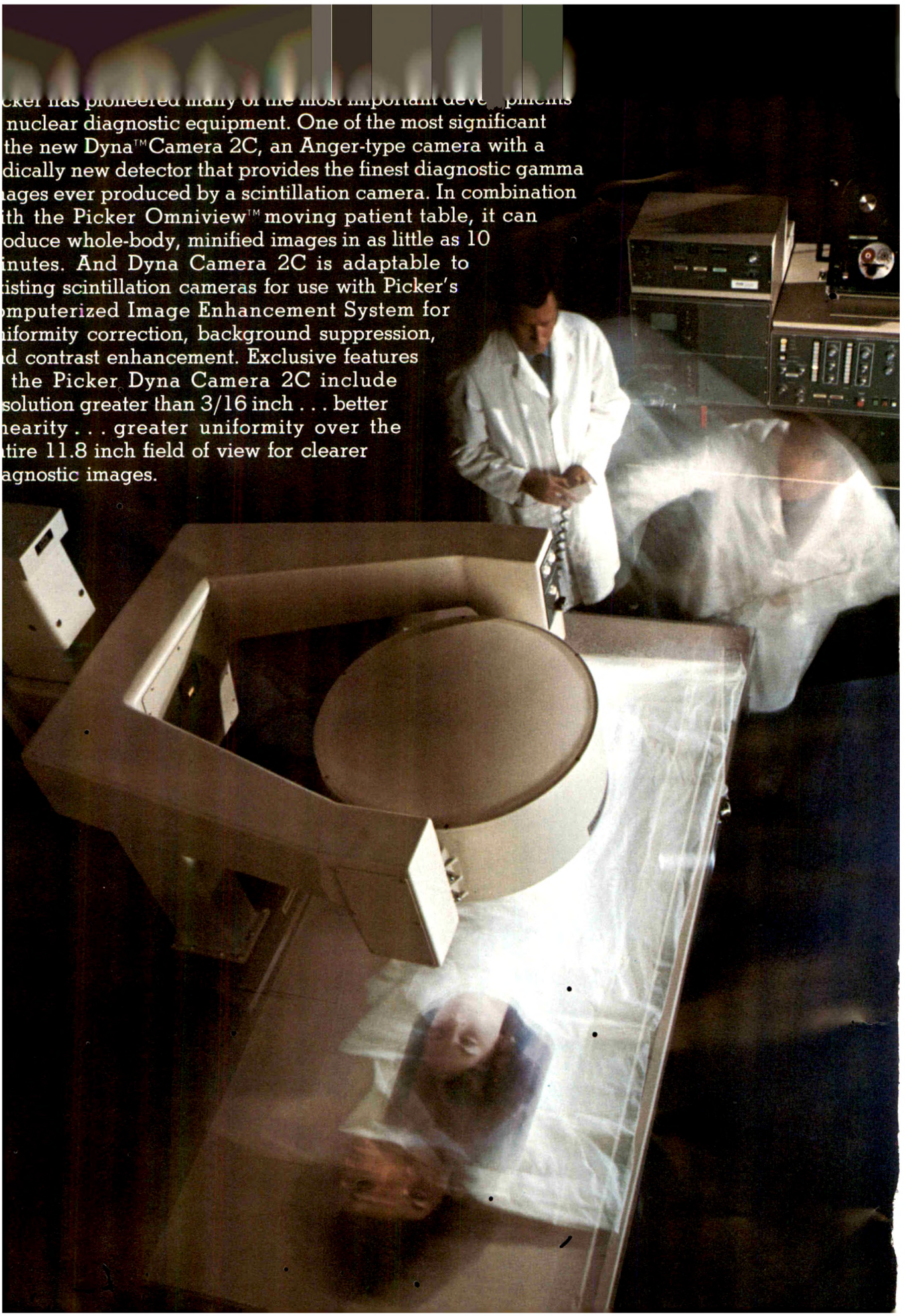
A-mode echogram of cystic lesion



B-scan laminogram of fetus

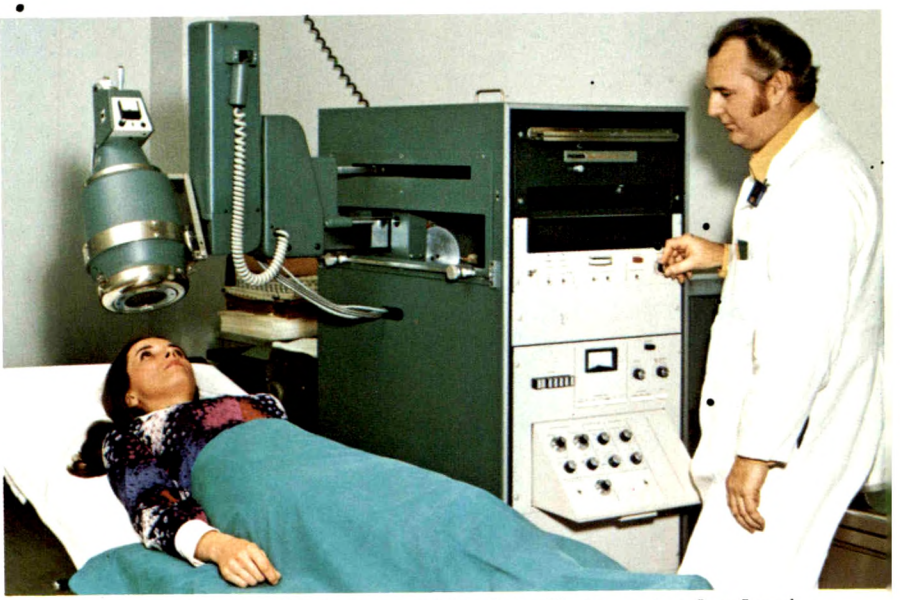


Ultrasound scan of left ventricle posterior wall taken on Echoview X cardiac unit.

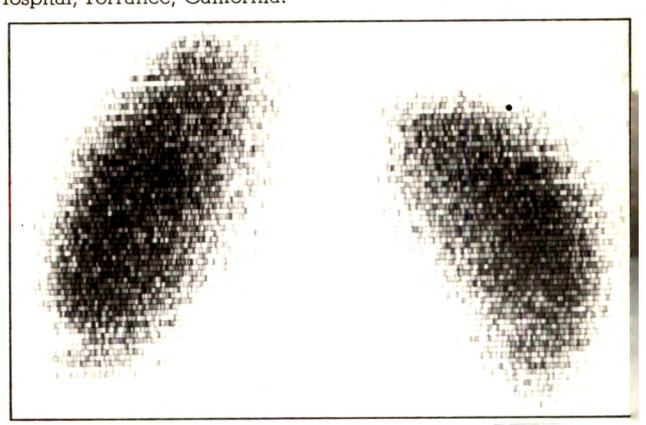


cker has pioneered many of the most important developments
nuclear diagnostic equipment. One of the most significant
the new Dyna™ Camera 2C, an Anger-type camera with a
dically new detector that provides the finest diagnostic gamma
ages ever produced by a scintillation camera. In combination
ith the Picker Omniview™ moving patient table, it can
oduce whole-body, minified images in as little as 10
inutes. And Dyna Camera 2C is adaptable to
isting scintillation cameras for use with Picker's
mputerized Image Enhancement System for
iformity correction, background suppression,
d contrast enhancement. Exclusive features
the Picker Dyna Camera 2C include
solution greater than 3/16 inch . . . better
nearity . . . greater uniformity over the
tire 11.8 inch field of view for clearer
agnostic images.

The Picker Magna® Scanners in use today throughout the world probably outnumber all other gamma-imaging devices combined. The new Magna Scanner, and dual Magna Scanner provide virtually automated scan setups, including highest quality color scans. Productivity is high because the instrument gives consistent scans with minimal retakes.

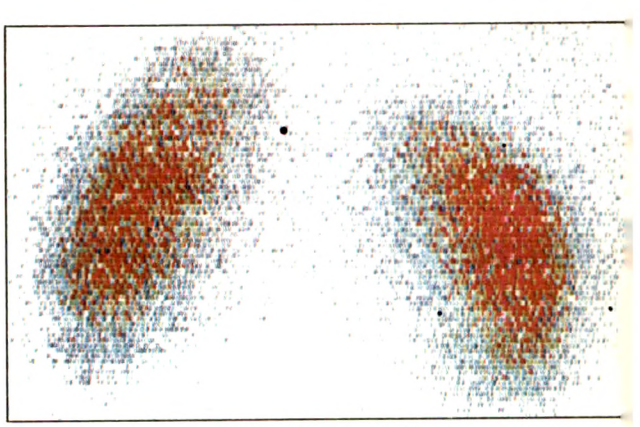


Magna Scanner 500D using 10-degree caudal technic. Photo courtesy Los Angeles County Harbor General Hospital, Torrance, California.





Technetium 99^{m} Polyphosphate Whole-Body Image



Posterior kidney scan: Dose 300 μCi 197 Hg; line spacing 2 mm; scanning speed 250 cm/min. Impression — Possible cysts in both kidneys.



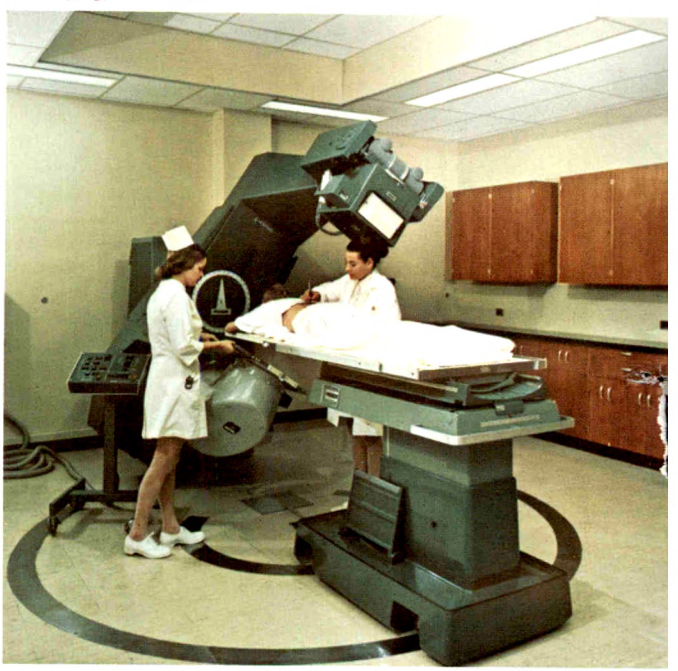
Left Lateral Brain Image from Dyna Camera 2C (50% reduction)

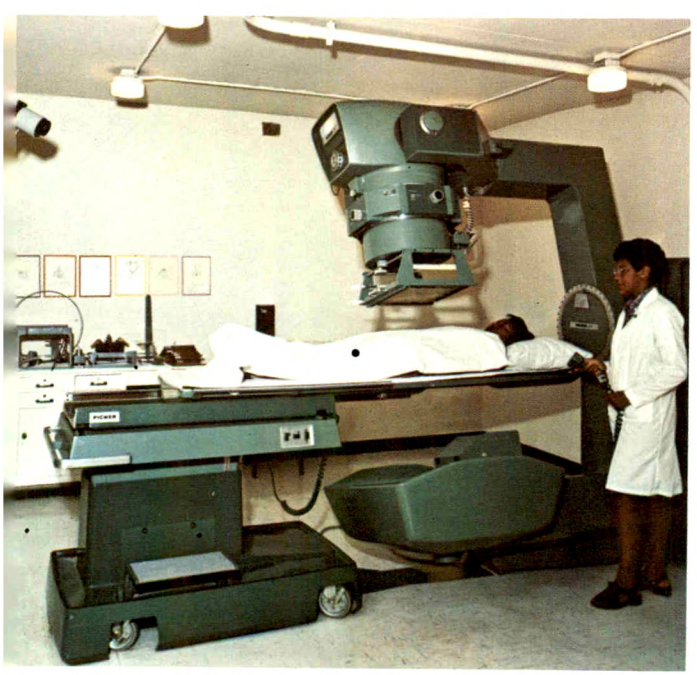
Radiation Therapy

Using diagnostic x-rays, Picker's TherX™ simulator pinpoints tumors, confirms entry and exit ports, and verifies beam coverage at every phase of the treatment to maintain the vital radiation beam/patient relationship. TherX is also invaluable as a teaching tool, or in aiding the radiotherapist in developing radiation treatment parameters.

Picker's credentials in radiation therapy extend from Cobalt-60 capsule manufacture to designing and producing the new C9 and V9 therapy units. These are a fourth-generation technological advance in cobalt radiation-therapy equipment. The C9 unit, featuring wide-ranging capability in moving-beam techniques, also has unprecedented isocentric accuracy, and enormous flexibility for setting up accurate, multiple-fixed-beam therapy. The Picker V9 vertical unit offers a full complement of technical advantages identical to the C9 rotational unit. Safety is paramount in all Picker designs and both units incorporate triple-safeguard systems for source security.

Therapy Simulator Room contains TherX, 300 mA generator, Decalix TV imaging system with isocentric diagnostic stretcher. Photo courtesy Manitoba Cancer Treatment and Research Foundation, Winnipeg, Manitoba.





herapy Room with Picker C9 Cobalt-60 unit. Photo ourtesy Radiology Department, George W. Hubbard Hospital f Meharry Medical College, Nashville, Tennessee.

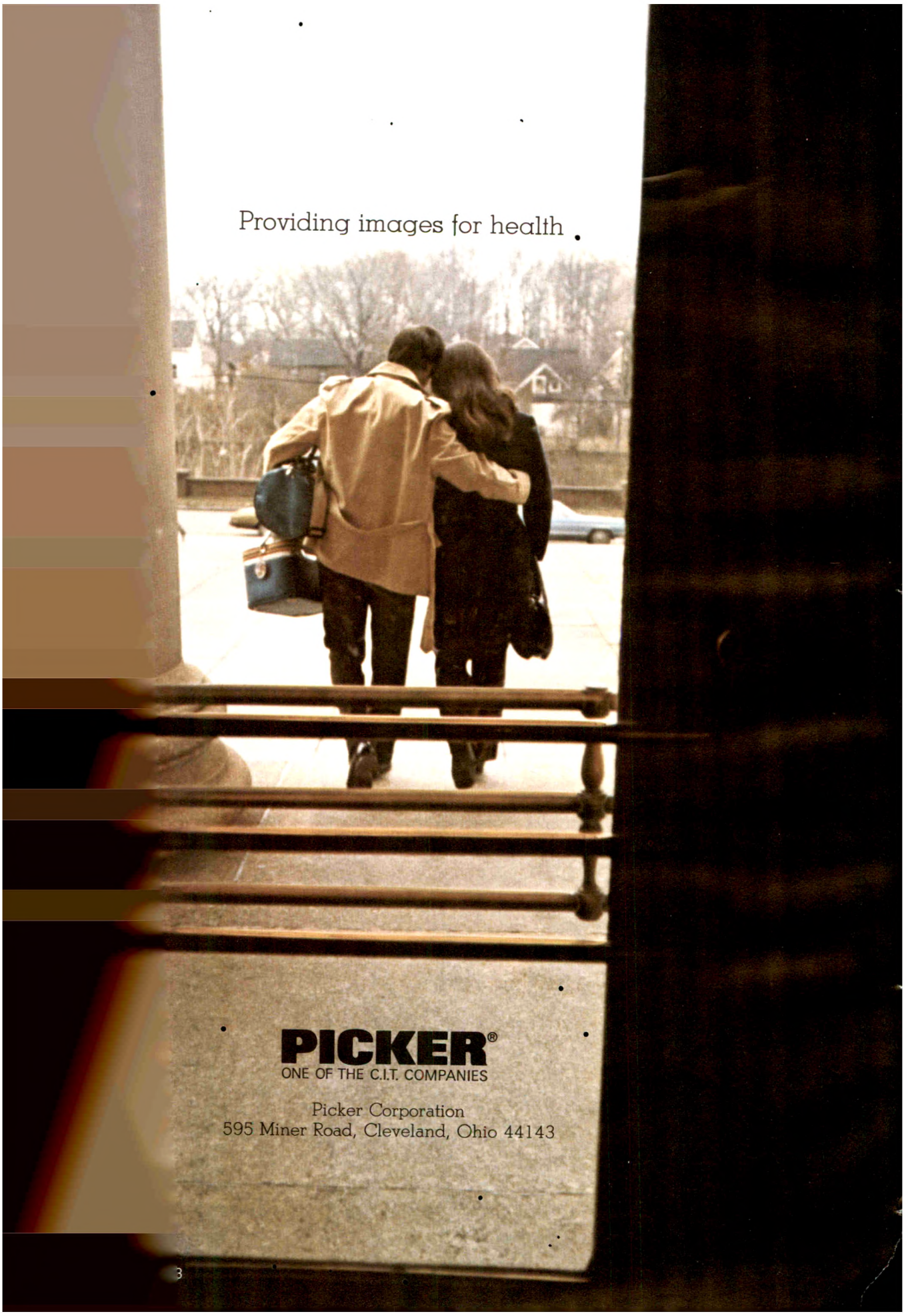


Therapy Simulator Room as seen from control booth. Photo courtesy Manitoba Cancer Treatment and Research Foundation, Winnipeg, Manitoba.

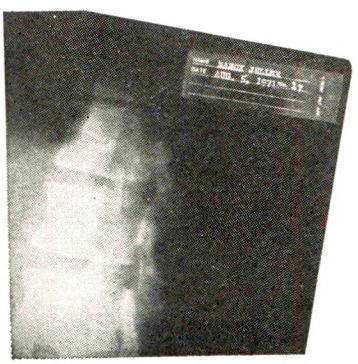
Other reasons to buy from Picker...

Now that you've seen some of Picker's diagnostic visualization and radiation therapy equipment in actual hospital applications, look at other aspects of Picker's working corporate philosophy. For example, our Film Systems and Supplies organization stocks over 3000 individual items to keep your Radiology Department operating smoothly, efficiently and economically. (A complimentary copy of the Picker catalog is available to you on request to your Picker representative.) And to close the circle of total commitment to the medical profession, Picker maintains the largest technical sales/service organization in the industry numbering over 1000 strong.



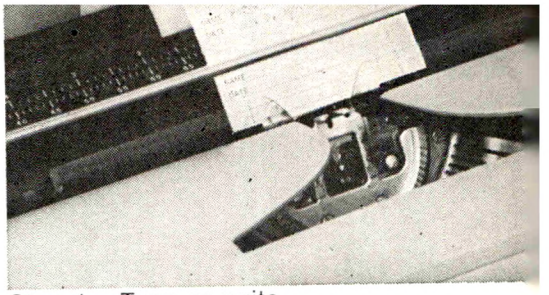

Providing images for health
PICKER®
ONE OF THE C.I.T. COMPANIES
Picker Corporation
595 Miner Road, Cleveland, Ohio 44143

When it comes to patient-film identification, you can't beat the system.

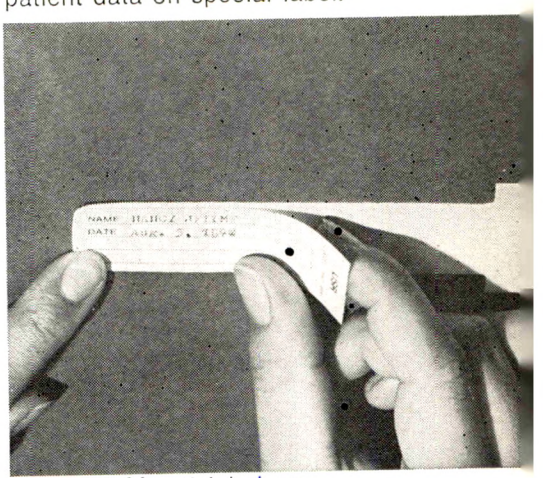


Halsey Chron-I-System. It lets you see the problem in a new light.

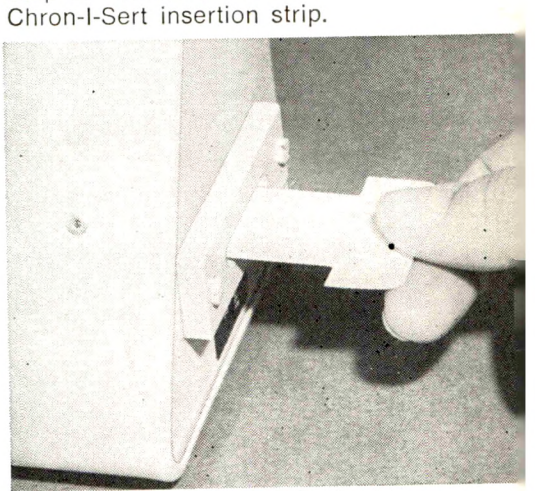
New Halsey Chron-I-System provides positive patient-film identification in ordinary daylight — without opening the cassette or entering the darkroom. Chron-I-System is a simple 4-step technique that takes only seconds. The system consists of special labels, insertion strips, an Exciter and Halsey Chron-I-Settes. It's extremely flexible — can also be used with patient "Charge Plate" imprinters — and permits positive film identification before or after X-ray examination. Chron-I-System requires no X-radiation, thus no "burn-out" or "coning cut off". It eliminates identification printers and minimizes the possibility of film misidentification. Halsey Chron-I-System. You can't beat it for speed or efficiency. Call your dealer for a demonstration today.



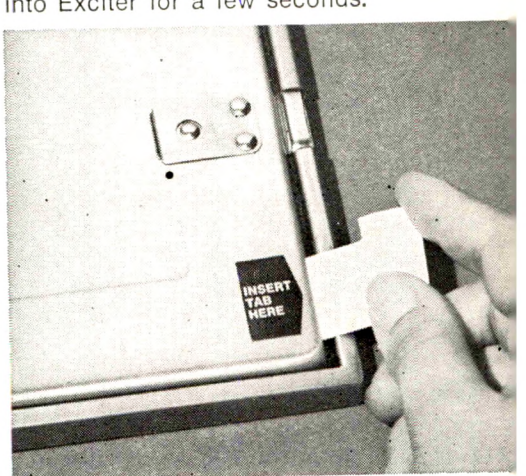
Step 1. Type or write patient data on special label.



Step 2. Mount label on Chron-I-Sert insertion strip.



Step 3. Insert Chron-I-Sert into Exciter for a few seconds.

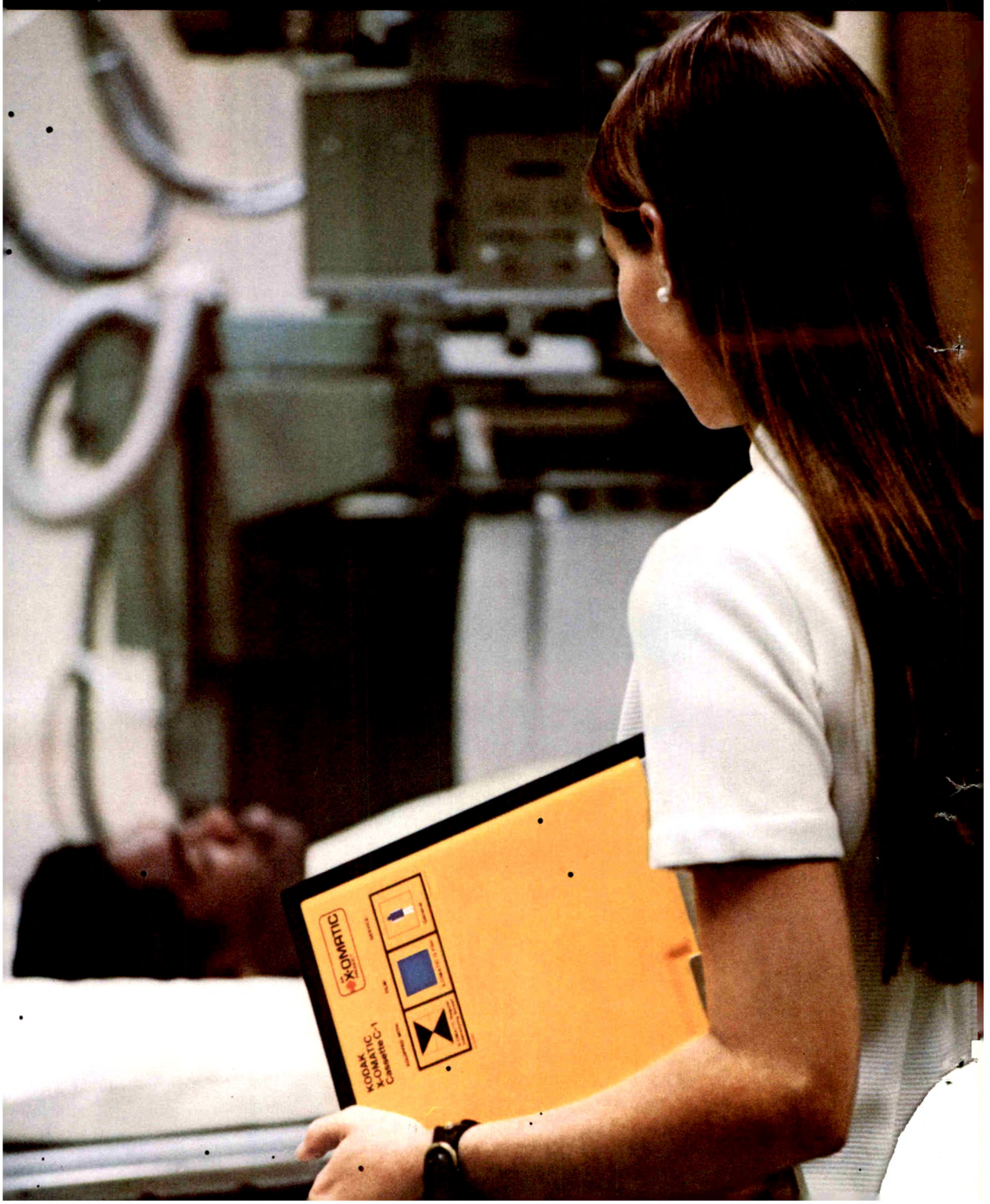


Step 4. Place Chron-I-Sert in Chron-I-Sette slot. Remove in 3 seconds.

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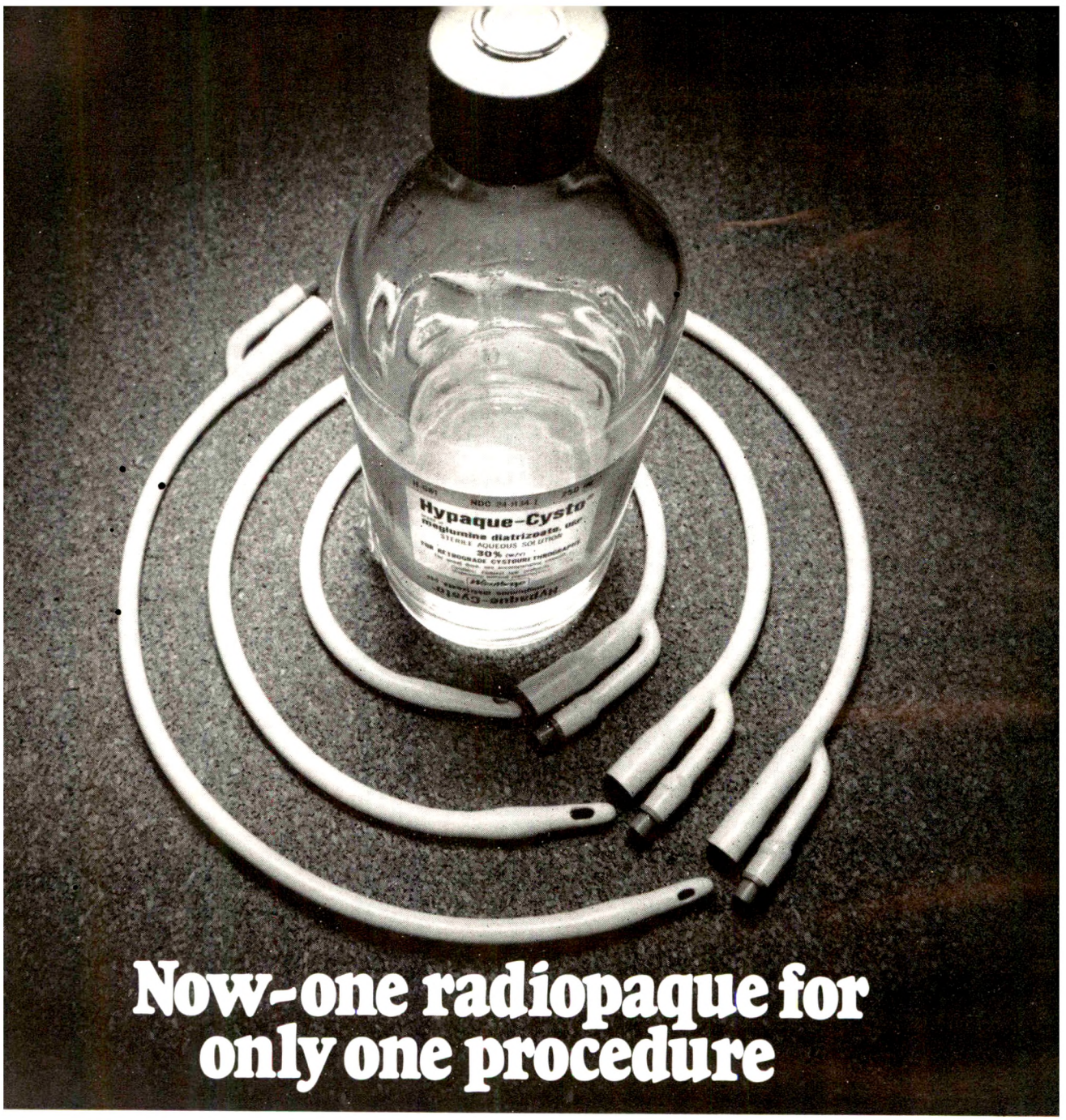
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Indication: Hypaque-Cysto is indicated for retrograde cystourethrography.

Adverse Reactions: No irritation or other undesirable effects have been reported following the administration of Hypaque-Cysto in clinical investigations.

Dosage and Administration: After the bladder is emptied, Hypaque-Cysto is gently instilled without force, often beyond the first desire to micturate, but not beyond the point of urgency or mild discomfort. The volume required to fill the bladder to slightly less than capacity may vary from patient to patient.

Bladder capacity in normal adults is generally 200 to 300 ml., and rarely, up to 600 ml. Capacity at birth is 20 to 50 ml., and increases about 400 per cent in the first year. In children 3 to 5 years old, bladder capacity is 150 to 180 ml. In children older than 8 years, it is in the low adult range.

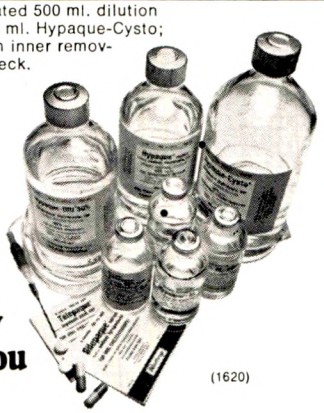
In disease, bladder capacity in adults may vary from 50 ml. in neurogenic "reflux" bladder to over 1000 ml. in autonomous bladder or chronic lower urinary tract obstruction.

Repeat examination may be required to detect reflux, or in function studies.

The concentration varies with technique and equipment used. Hypaque-Cysto may be diluted with sterile water or 5 per cent dextrose solution, as indicated in the following table.

To Make		Add	Final Solution Contains
Final conc.	Final volume	Sterile water or 5% dextrose solution	Iodine
30 %	250 ml.	—	141 mg./ml.
25 %	300 ml.	50 ml.	118 mg./ml.
21.4%	350 ml.	100 ml.	101 mg./ml.
20 %	375 ml.	125 ml.	94 mg./ml.
18.8%	400 ml.	150 ml.	88 mg./ml.
16.7%	450 ml.	200 ml.	78 mg./ml.
15 %	500 ml.	250 ml.	71 mg./ml.

How Supplied: Calibrated 500 ml. dilution bottles containing 250 ml. Hypaque-Cysto; rubber stoppered, with inner removable seal and screw neck.



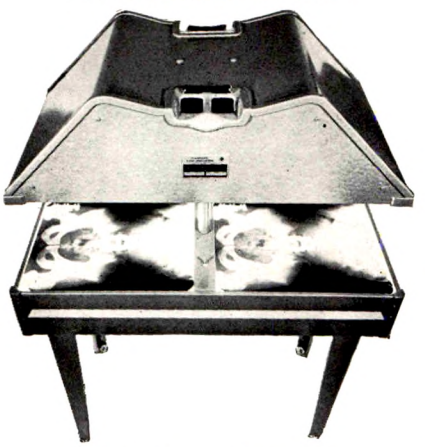
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This book contains the lectures presented at the Annual Nuclear Medicine Seminar and also presents illustrated materials from this lecture. It includes several sections explaining the basic science of nuclear medicine, radiopharmaceuticals and radiation therapy. Some of the topics discussed are interaction of gamma rays with matter, reactor and accelerator production of radionuclides, application of the dilution principle, basic mathematics of nuclear medicine, electrolytes and body composition, essentials of rectilinear scanning, evaluation of renal function and anatomy, principles of radiation therapy with radionuclides, radionuclide cisternography, particle and photon radiations, biological effects of radiation, radiation detection, hematologic application of radionuclides, blood flow measurement with radionuclides and intracavitary radiocolloid therapy.

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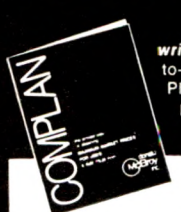
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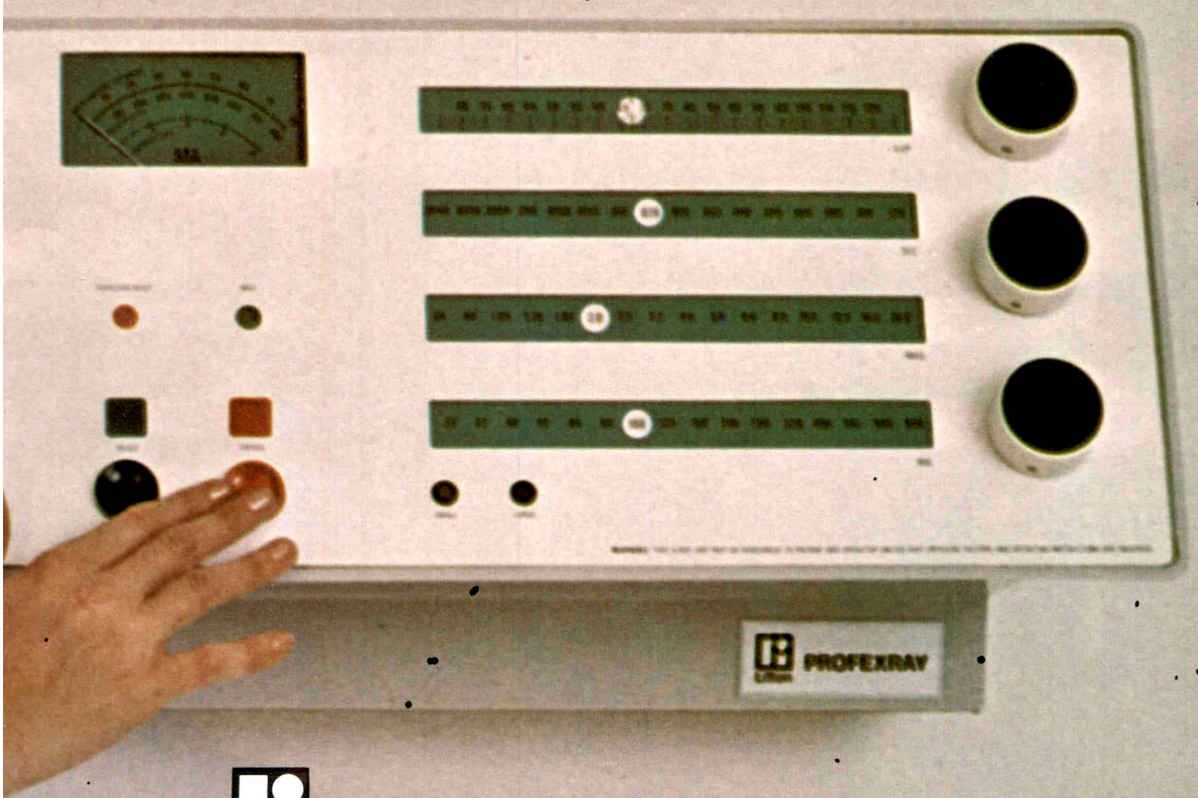
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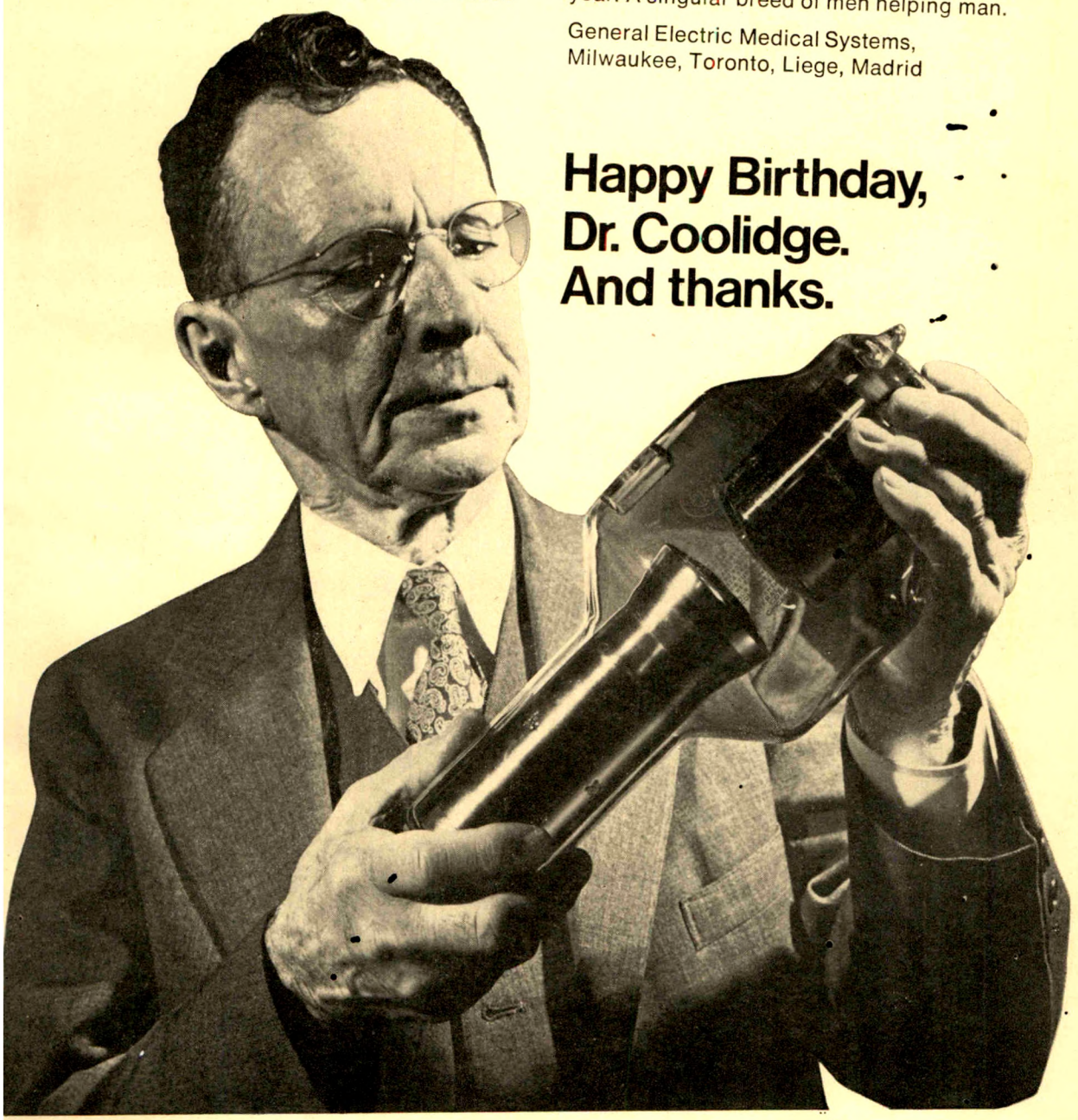
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Intrinsic Diseases of Bones

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